ORIGINAL RESEARCH PAPER

General Medicine

KEY WORDS: Takotsubo Cardiomyopathy, Acute Ischemic Stroke, Stress-Induced Cardiomyopathy, Concurrent Presentation

WHEN THE BRAIN BREAKS THE HEART : STROKE AND TAKOTSUBO CARDIOMYOPATHY

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ABSTRACT

Background: Takotsubo cardiomyopathy is a reversible stress-induced cardiomyopathy that rarely presents concurrently with acute ischemic stroke. The co-occurrence of these conditions poses significant diagnostic and therapeutic challenges in medicine. **Case Presentation:** We report a 45-year-old female who presented to the emergency department with a 7-day history of giddiness, left-sided weakness, slurred speech, and facial deviation. Initial examination revealed hypotension (90/60 mmHg), bradycardia (50/min), and left upper motor neuron facial palsy. ECG showed changes like QT prolongation and troponin I levels were elevated(77.3) suggesting some cardiac defect. Echocardiography revealed severely reduced ejection fraction (25-29%) with apical akinesia and apical ballooning, suggesting Takotsubo cardiomyopathy with severe tricuspid regurgitation, moderate mitral regurgitation and severe pulmonary hypertension. Coronary angiography showed normal coronaries, confirming stress-induced cardiomyopathy. MRI brain revealed acute non- hemorrhagic infarct in the right parietal lobe. The patient was started on dual antiplatelet therapy, anticoagulation, diuretics and ionotropic support. Serial echocardiography showed improvement in ejection fraction to approximately 40% within 4 days and then to 59% in next 3 days. **Conclusion:** This case highlights the rare but clinically significant concurrent presentation of acute ischemic stroke and Takotsubo cardiomyopathy, emphasizing the importance of comprehensive cardiac evaluation in stroke patients presenting with hemodynamic instability.

INTRODUCTION

Takotsubo cardiomyopathy, also known as stress-induced cardiomyopathy or broken heart syndrome, is a transient left ventricular dysfunction characterized by regional wall motion abnormalities that extend beyond a single epicardial coronary distribution[1]. First described in Japan in 1990, this condition typically affects postmenopausal women and is often precipitated by intense emotional or physical stress[2]. The pathophysiology involves excessivecatecholamine release leading to myocardial stunning, with characteristic apical ballooning and basal hypercontractility[3].

Acute ischemic stroke represents a major cause of morbidity and mortality worldwide, affecting approximately 795,000 people annually in the United States[4]. While cardiac complications following stroke are well-recognized, the concurrent presentation of acute ischemic stroke with Takotsubo cardiomyopathy is exceptionally rare and poses unique diagnostic and therapeutic challenges[5].

The relationship between stroke and Takotsubo cardiomyopathy is complex and bidirectional. Stroke can trigger stress-induced cardiomyopathy through excessive sympathetic stimulation, while cardiac dysfunction can predispose to embolic stroke[6]. The diagnosis requires a high index of suspicion, especially in patients presenting with stroke and concurrent cardiac symptoms or hemodynamic instability[7].

Case Presentation

A 45-year-old female presented to the emergency department with a 7-day history of progressive giddiness, left-sided weakness, slurred speech, involuntary movements and deviation of the mouth to the left side. She denied fever, headache or seizures. The patient had no significant past medical history or known cardiovascular risk factors.

On initial examination, the patient was conscious and oriented with a Glasgow Coma Scale of 15/15. Vital signs revealed hypotension (90/60 mmHg), bradycardia (50 beats per minute) and normal temperature. Neurological examination demonstrated left upper motor neuron facial palsy with sided hemiparesis (power 3/5), while right-sided power was normal (5/5). Cardiovascular examination revealed pan systolic murmur at MA, AA. Respiratory system examination revealed bilateral basal crepitations.

Initial investigations included a non-contrast CT brain performed on the day of admission (25/08/2025), which revealed hypodensity (Hounsfield units 22-27) in the right parietal region, punctate gliosis in the right midbrain, and multiple ill-defined hypodensities in the bilateral centrum semiovale consistent with small vessel ischemic changes.

Laboratory investigations showed mild anemia (hemoglobin 10.3-11.3 g/dL), leukocytosis (13,400/μL), normal electrolytes (sodium 138 mEq/L, potassium 3.5 mEq/L), normal renalfunction (creatinine 0.7 mg/dL) and significantly elevated troponin I (77.3 ng/mL). Random blood sugar was 128 mg/dL with HbA1c of 5.7%. Lipid profile showed total cholesterol 146 mg/dL, LDL 109 mg/dL, and HDL 43 mg/dL. Thyroid function tests were within normal limits. Urine analysis revealed proteinuria (albumin ++) but was otherwise unremarkable.

Electrocardiography demonstrated sinus bradycardia (heart rate 50 bpm), T-wave inversions in leads V2-V6, QT prolongation (QTc up to 525 ms), left ventricular hypertrophy pattern, and occasional premature ventricular contractions.

MRI brain with angiography performed on 26/08/2025 confirmed an acute non-hemorrhagic infarct in the right parietal lobe with small vessel ischemic changes (Fazekas Grade I). The MR angiogram showed normal caliber and flow-related enhancement in intracranial internal carotid, vertebral, and basilar arteries with no significant stenosis, aneurysm, dissection, or vascular malformation.

Echocardiography revealed severely reduced ejection fraction (25-29%), dilated cardiac chambers (right atrium, left atrium, right ventricle, left ventricle), apical ballooning with apical akinesia, severe tricuspid regurgitation, moderate

mitral regurgitation, severe pulmonary hypertension (right ventricular systolic pressure ~73 mmHg), and dilated non-collapsing inferior vena cava (23mm). No left ventricular clot or pericardial effusion was observed. These findings were highly suggestive of Takotsubo cardiomyopathy.

To exclude obstructive coronary artery disease, coronary angiography was performed on 01/09/2025 using a 5F radial approach with 15 mL contrast. This revealed normal left main coronary artery, left anterior descending artery, left circumflex artery, and right coronary artery with no evidence of obstructive disease, confirming the diagnosis of stress-induced cardiomyopathy.

Arterial blood gas analysis showed pH 7.38, pCO_2 38.4 mmHg, HCO_3 24.3 mmol/L, lactate 1.72 mmol/L, and oxygen saturation 96.3%, indicating normal acid-base status with borderline elevated lactate.

Table 1: Summary of Clinical Parameters and Key Investigations

Parameter	Day 1 (25/08/2025)	Day 2 (26/08/2025)	Day 4 (28/08/2025)	Day 7 (31/08/2025)	Reference Range
Vital Signs					
Blood Pressure (mmHg)	90/60	90/60	120/70	120/70	120/80
Heart Rate (bpm)	50	50-70	74	70	60-100
Glasgow Coma Scale	15/15	15/15	15/15	15/15	15/15
Neurological					
Left-sided power	3/5	Improving	4/5	4/5	5/5
Right-sided power	5/5	5/5	5/5	5/5	5/5
Laboratory Values					
Hemoglobin (g/dL)	10.3	11.3	10.2	10.8	12-15
Total Leukocyte Count (/μL)	13,400	-	9000	9900	4,000-11,000
Troponin I (ng/mL)	77.3	-	-	-	<0.04
Creatinine (mg/dL)	0.7	-	0.8	0.9	0.6-1.2
Cardiac Assessment					
Ejection Fraction (%)	25-29	25-29	~40	~59	>55
RVSP (mmHg)	~73	~73	~45	~45	<30
Tricuspid Regurgitation	Severe	Severe	Moderate	Moderate	None/Trivial
Mitral Regurgitation	Moderate	Moderate	Mild	Mild	None/Trivial
ECG Findings					
Rhythm	Sinus bradycardia	Sinus bradycardia	Sinus rhythm	Sinus rhythm	Normal sinus
QTc (ms)	525	-	-	-	<450
T-wave changes	V2-V6 inversion	V2-V6 inversion	V2-V4 inversion	V2-V4 inversion	Normal

The patient was treated with dual antiplatelet therapy (ecosprin and clopidogrel), anticoagulation with heparin (3500 IU iv thrice daily), diuretics (furosemide 20 mg every 12 hours) and ionotropic support with noradrenaline (16 mg in 50 mL normal saline at 2 mL/hour). Supportive care included intravenous fluids, oxygen therapy and continuous cardiac monitoring.

Serial echocardiography showed progressive improvement in left ventricular function with ejection fraction improving to approximately 40% by 29/08/2025 and 59% by 31/08/2025. Pulmonary hypertension improved with right ventricular systolic pressure decreasing to ~45 mmHg, and the inferior vena cava became collapsible, indicating improved hemodynamic status. The patient's neurological status gradually improved, and hemodynamic parameters stabilized. Her ionotropic support was successfully weaned off and she was transferred from the ICU to the general ward.

DISCUSSION

This case represents a rare concurrent presentation of acute ischemic stroke and Takotsubo cardiomyopathy in the medicine department. The co-occurrence of these conditions is exceptionally uncommon, with limited case reports in the literature describing this dual pathology[8].

Takotsubo cardiomyopathy is characterized by transient left ventricular dysfunction in the absence of obstructive coronary artery disease, typically triggered by intense emotional or physical stress[2]. The condition predominantly affects postmenopausal women, as seen in our patient. The pathophysiology involves excessive catecholamine release leading to myocardial stunning, with characteristic apical ballooning pattern observed on echocardiography[3].

The relationship between stroke and Takotsubo cardiomyopathy is complex and bidirectional. Acute stroke can trigger stress-induced cardiomyopathy through activation of the hypothalamic-pituitary-adrenal axis and excessive sympathetic stimulation[9]. Conversely, the cardiac dysfunction associated with Takotsubo cardiomyopathy can predispose patients to embolic stroke through formation of intracardiac thrombi or hemodynamic compromise[10].

In our case, the temporal relationship suggests that the stroke likely preceded and potentially triggered the development of Takotsubo cardiomyopathy. The patient's initial presentation withneurological symptoms followed by the development of cardiac dysfunction supports this sequence. The elevated troponin I levels (77.3 ng/mL) and characteristic ECG changes including T-wave inversions and QT prolongation are consistent with stress-induced cardiomyopathy[11].

The diagnostic challenges in this case were significant. The concurrent presentation of stroke and cardiac dysfunction required differentiation from acute coronary syndrome, which was accomplished through coronary angiography demonstrating normal coronaries. The characteristic echocardiographic findings of apical akinesia with apical ballooning, along with the clinical context, supported the diagnosis of Takotsubo cardiomyopathy[12].

Management of concurrent stroke and Takotsubo cardiomyopathy requires a multidisciplinary approach. Antiplatelet therapy for stroke prevention must be balanced against the risk of bleeding in patients with cardiac dysfunction. The use of anticoagulation in our patient was guided by the stroke etiology and cardiac function. Hemodynamic support with inotropes was necessary due to the severely reduced ejection fraction and hypotension[13].

The prognosis for Takotsubo cardiomyopathy is generally favorable, with most patients showing complete recovery of cardiac function within weeks to months[14]. Our patient demonstrated typical improvement with ejection fraction recovering from 25-29% to approximately 59% within 7 days, supporting the reversible nature of this condition. Long- term follow-up is essential to monitor for complete cardiac recovery and prevent recurrence[15].

This case highlights the importance of comprehensive cardiac evaluation in stroke patients, particularly those

presenting with hemodynamic instability or elevated cardiac biomarkers. Physicians should maintain a high index of suspicion for concurrent cardiac pathology in stroke patients, as early recognition and appropriate management can significantly impact outcomes.

CONCLUSION

This case demonstrates the rare but clinically significant concurrent presentation of acute ischemic stroke and Takotsubo cardiomyopathy. The bidirectional relationship between these conditions necessitates a high index of suspicion and comprehensive evaluation when patients present with stroke and concurrent cardiac symptoms or hemodynamic instability.

The successful management of this patient emphasizes the importance of a multidisciplinary approach involving the departments of medicine, neurology and cardiology. Early recognition of stress-induced cardiomyopathy through appropriate cardiac evaluation, including echocardiography and coronary angiography, is crucial for optimal patient outcomes. The generally favorable prognosis of Takotsubo cardiomyopathy, as demonstrated by the improvement in cardiac function in our patient, underscores the importance of supportive care and monitoring during the acute phase.

Declarations

Patient Consent: Written informed consent was obtained from the patient for publication of this case report.
Conflict of Interest: None declared.
Ethical Approval: Not applicable for this case report.
Funding: None declared.
Author Contributions: All authors contributed to the clinical management of the patient and preparation of this manuscript.

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