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Research Article

Clinical Characteristics of Takotsubo Cardiomyopathy in Neurological Emergencies: A Systematic Review of Brain-Heart Interaction

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Abstract

Background: Takotsubo cardiomyopathy (TCM), also known as stress-induced cardiomyopathy or transient left ventricular apical ballooning syndrome, is a reversible form of acute myocardial dysfunction characterized by transient ventricular systolic impairment in the absence of obstructive coronary artery disease. Although emotional stress was initially believed to be the primary trigger, increasing evidence demonstrates that acute neurological disorders may precipitate this condition through complex neuro-cardiac interactions.

Objective: This systematic review aims to analyze the clinical characteristics, underlying mechanisms, diagnostic features, and clinical outcomes of Takotsubo cardiomyopathy occurring in the setting of neurological emergencies.

Methods: A systematic literature review was conducted following preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines. Electronic databases including PubMed, Scopus, and Web of Science were searched for relevant studies published between 1990 and 2024. Studies describing Takotsubo cardiomyopathy associated with neurological conditions were included.

Results: Forty-seven relevant studies were identified and analyzed. Neurological emergencies most commonly associated with Takotsubo cardiomyopathy included subarachnoid hemorrhage, ischemic stroke, intracerebral hemorrhage, traumatic brain injury, and seizure disorders. Clinical manifestations often mimicked acute coronary syndrome and included electrocardiographic abnormalities, modest elevations of cardiac biomarkers, and reversible ventricular wall-motion abnormalities on echocardiography.

Conclusion: Takotsubo cardiomyopathy represents a significant manifestation of the brain-heart axis in patients experiencing acute neurological injury. Early recognition and interdisciplinary management are essential to reduce morbidity and improve outcomes.

Keywords: *Takotsubo Cardiomyopathy; Neurogenic Myocardial Injury; Neurological Emergencies; Brain-Heart Axis; Stress Cardiomyopathy*

Introduction

Takotsubo cardiomyopathy is an increasingly recognized cardiovascular syndrome characterized by transient systolic dysfunction of the left ventricle in the absence of significant coronary artery obstruction. The condition was first described in Japan and derives its name from the distinctive ventricular configuration observed during systole, which resembles a traditional Japanese octopus trapping pot [1]. Since its initial recognition, Takotsubo cardiomyopathy has emerged as an important clinical entity that often mimics acute coronary syndrome.

While early reports primarily associated Takotsubo cardiomyopathy with severe emotional stress, subsequent investigations have demonstrated that physical stressors may play an equally important role in triggering the syndrome [19]. Among these triggers, acute neurological events have gained increasing attention due to their strong association with transient myocardial dysfunction.

Neurological emergencies such as aneurysmal subarachnoid hemorrhage, ischemic stroke, intracerebral hemorrhage, traumatic brain injury, and status epilepticus have all been implicated as precipitating factors for Takotsubo cardiomyopathy [4]. These conditions can provoke profound activation of the sympathetic nervous system, resulting in excessive catecholamine release that exerts toxic effects on myocardial tissue.

The interaction between the brain and the cardiovascular system is commonly described as the brain-heart axis, a complex network of neural and hormonal pathways responsible for maintaining cardiovascular homeostasis. Disruption of this axis following acute brain injury can result in various cardiac complications including arrhythmias, myocardial injury, and transient ventricular dysfunction [16].

Among neurological disorders, subarachnoid hemorrhage demonstrates one of the strongest associations with stress cardiomyopathy. Studies have shown that cardiac abnormalities occur in a substantial proportion of patients with subarachnoid hemorrhage, highlighting the importance of neuro-cardiac interactions in critical illness [9].

Despite increasing recognition of this phenomenon, Takotsubo cardiomyopathy remains underdiagnosed in patients with neurological emergencies because cardiac manifestations may be overshadowed by severe neurological symptoms. A comprehensive understanding of the clinical characteristics and pathophysiological mechanisms of this condition is therefore essential.

Aim of the Study

This systematic review aims to synthesize existing evidence regarding the clinical characteristics, diagnostic features, and outcomes of Takotsubo cardiomyopathy occurring in neurological emergencies, with particular emphasis on the underlying brain-heart interaction.

Methods

Study design

This systematic review was conducted in accordance with the preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines.

Search strategy

A comprehensive literature search was performed using the following electronic databases:

- PubMed
- Scopus
- Web of Science
- Google Scholar.

The search included studies published between 1990 and 2024 using combinations of the following keywords:

- Takotsubo cardiomyopathy
- Stress cardiomyopathy
- Neurogenic myocardial injury
- Neurological emergencies
- Subarachnoid hemorrhage
- Stroke
- Brain-heart interaction.

Inclusion criteria

Studies were included if they:

1. Reported Takotsubo cardiomyopathy associated with neurological disorders.
2. Included adult patient populations.
3. Described clinical presentation, diagnostic findings, or outcomes.
4. Were published in peer-reviewed journals in English.

Exclusion criteria

Studies were excluded if they:

- Focused solely on emotional stress triggers.
- Included pediatric populations.
- Lacked sufficient clinical data.

Data extraction

Data extracted from eligible studies included:

- Demographic characteristics.
- Neurological diagnosis.
- Electrocardiographic findings.
- Echocardiographic features.
- Cardiac biomarker levels.
- Clinical outcomes.

Results

Neurological triggers

Analysis of included studies demonstrated that several neurological emergencies were associated with Takotsubo cardiomyopathy.

The most frequently reported triggers included:

- Subarachnoid hemorrhage.
- Ischemic stroke.
- Intracerebral hemorrhage.
- Traumatic brain injury.
- Seizures and status epilepticus.

Subarachnoid hemorrhage represented the most commonly reported neurological trigger, likely due to the abrupt sympathetic surge that occurs following aneurysm rupture [25].

Clinical presentation

Patients with Takotsubo cardiomyopathy in neurological emergencies often present with clinical features resembling acute coronary syndrome. Common symptoms include chest pain, dyspnea, hypotension, and signs of acute heart failure. However, in neurocritical care settings, these cardiac manifestations may be difficult to recognize due to the severity of neurological symptoms.

Electrocardiographic abnormalities are frequently observed and may include ST-segment elevation, T-wave inversion, QT interval prolongation, and ventricular arrhythmias [14].

Cardiac biomarkers such as troponin and B-type natriuretic peptide are typically elevated, although the magnitude of elevation is generally lower than that observed in myocardial infarction [2].

Echocardiographic findings

Echocardiography plays a central role in diagnosing Takotsubo cardiomyopathy. Characteristic findings include transient left ventricular systolic dysfunction with regional wall-motion abnormalities that extend beyond the distribution of a single coronary artery.

The most commonly observed pattern is apical ballooning, although several variants have been described including mid-ventricular and basal patterns [3].

Discussion

The findings of this systematic review highlight the strong association between neurological emergencies and Takotsubo cardiomyopathy. Acute neurological injury can disrupt autonomic regulation and trigger profound sympathetic activation, resulting in excessive catecholamine release and myocardial dysfunction [21].

Catecholamine toxicity has been widely proposed as the primary mechanism underlying Takotsubo cardiomyopathy. Elevated catecholamine levels can induce myocardial stunning through calcium overload, oxidative stress, and microvascular dysfunction [12].

In addition to catecholamine toxicity, several other mechanisms have been proposed including coronary microvascular spasm, autonomic imbalance, and direct neurogenic myocardial injury [8].

Subarachnoid hemorrhage appears to represent the most important neurological trigger of Takotsubo cardiomyopathy. The sudden increase in intracranial pressure and sympathetic activation following aneurysm rupture can produce profound cardiovascular disturbances [25].

Although Takotsubo cardiomyopathy is typically reversible, its occurrence in neurological emergencies may significantly influence clinical outcomes. Cardiac complications such as arrhythmias, pulmonary edema, and cardiogenic shock can contribute to increased morbidity and prolonged intensive care unit stays [20].

Conclusion

Takotsubo cardiomyopathy represents an important manifestation of the brain-heart interaction in patients with neurological emergencies. Acute neurological injury can trigger excessive sympathetic activation and catecholamine release, resulting in transient myocardial dysfunction.

Recognition of this condition is essential because cardiac complications may significantly affect patient outcomes in neurocritical care settings. Early diagnostic evaluation and multidisciplinary management involving neurologists, cardiologists, and intensive care specialists are crucial for improving prognosis.

Future research should focus on prospective studies aimed at identifying risk factors, clarifying pathophysiological mechanisms, and developing targeted therapeutic strategies for patients with neurogenic stress cardiomyopathy.

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