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TAKOTSUBO CARDIOMYOPATHY: PATHOPHYSIOLOGY, CLINICAL FEATURES, AND CURRENT PERSPECTIVES

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Abstract: Takotsubo cardiomyopathy (TTC), also known as stress-induced cardiomyopathy or "broken heart syndrome," is a transient left ventricular dysfunction that mimics acute coronary syndrome (ACS) but occurs without obstructive coronary artery disease. This article explores the pathophysiology, clinical features, diagnostic principles, and neurohormonal mechanisms underlying TTC, emphasizing the roles of catecholamine toxicity, microvascular dysfunction, estrogen deficiency, and the neuro–cardiac axis.

Keywords: Takotsubo cardiomyopathy; stress-induced cardiomyopathy; catecholamines; microvascular dysfunction; apical ballooning; neuro–cardiac axis.

Introduction

Takotsubo cardiomyopathy was first described in Japan in 1990 and is named after an octopus pot due to the characteristic apical ballooning. TTC accounts for 1–2% of suspected ACS cases and predominantly affects postmenopausal women. Emotional or physical stress frequently triggers TTC, although one-third of cases occur without a clear trigger. Despite being reversible, TTC may cause acute heart failure, arrhythmias, or cardiogenic shock.

Pathophysiological Mechanisms

1. Catecholamine Surge:

Excessive sympathetic activation leads to extremely high circulating catecholamines—levels 2–3 times higher than those seen in myocardial infarction. Catecholamines cause myocardial stunning through calcium overload, oxidative stress, mitochondrial dysfunction, and contraction-band necrosis.

2. Coronary Microvascular Dysfunction:

TTC is strongly associated with transient coronary microvascular spasm and endothelial dysfunction. These mechanisms impair coronary perfusion despite angiographically normal coronary arteries.

3. β-Adrenergic Receptor Distribution:

The apex of the left ventricle has a higher density of β-adrenergic receptors, making it susceptible to catecholamine-induced negative inotropy. This receptor distribution pattern explains the apical ballooning phenotype.

4. Estrogen Deficiency:

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The predominance of TTC in postmenopausal women suggests estrogen loss plays a major role. Estrogen supports endothelial nitric oxide production and modulates sympathetic activity, and its deficiency reduces myocardial resilience to stress.

5. Neuro–Cardiac Axis:

Neuroimaging shows altered activation in the amygdala, insular cortex, and limbic system—regions regulating emotional stress. These abnormalities promote autonomic imbalance and excessive sympathetic discharge, linking stress processing to cardiac dysfunction.

Clinical Presentation
Patients typically present with acute chest pain, dyspnea, palpitations, or syncope, closely mimicking ACS. ECG changes include ST-segment elevation, T-wave inversion, or QT prolongation. Troponin elevations are modest compared to the extent of LV dysfunction. Echocardiography shows apical ballooning or, in variants, midventricular or basal dysfunction.

Diagnostic Criteria
Diagnostic evaluation uses the modified Mayo Clinic criteria:

1. Transient left ventricular systolic dysfunction beyond a single coronary territory.

2. Absence of obstructive coronary artery disease.

3. New ECG changes or mild troponin elevation.

4. Exclusion of myocarditis and pheochromocytoma.

Cardiac MRI helps differentiate TTC from myocarditis or other cardiomyopathies.

Clinical Significance
Although reversible, TTC can cause serious complications, including acute heart failure, left ventricular outflow tract obstruction, ventricular arrhythmias, thromboembolism, and cardiogenic shock. In-hospital mortality is 3–5%. Recurrence occurs in 5–10%, highlighting the need for long-term follow-up.

Conclusion
Takotsubo cardiomyopathy represents a complex interplay between emotional stress, sympathetic overstimulation, microvascular dysfunction, estrogen deficiency, and neuro–cardiac signaling. Although prognosis is generally favorable, TTC may present with life-threatening complications. Further research is needed to clarify chronic mechanisms and develop targeted therapies.

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