



A Brief Note on Transient Cardiomyopathy

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EDITORIAL

In a significant minority of severe coronavirus disease-19 (COVID-19) patients, acute cardiac dysfunction with new-onset cardiomyopathy showing clinically as heart failure with or without accompanying hemodynamic instability has been documented. Cardiomyopathy, defined as an echocardiographic evidence of a globally decreased left ventricular ejection fraction (LVEF) along with clinical features of cardiogenic shock, an elevation in cardiac biomarkers, or a decrease in central venous oxygen saturation, was present in a third of patients in a case series study of 21 critically ill COVID-19 patients admitted to the intensive care unit (33 percent). The major proposed mechanisms for cardiac dysfunction in COVID-19 patients include myocardial suppression due to a heightened systemic inflammatory response, a myocarditis-like syndrome due to direct viral invasion of cardiac myocytes, and a myocarditis-like syndrome due to direct viral invasion of cardiac myocytes. Stress-induced cardiomyopathy and myocytes. We provide a case of a COVID-19 patient who developed transient reversible cardiomyopathy, demonstrating the link between acute cardiac failure and significant systemic inflammation in COVID-19 patients. The heart's and brain's interaction is intricate and crucial to maintaining appropriate circulatory function. Certain pathological diseases can affect cardiovascular function by interfering with the normal brain-heart regulating processes. Samuels recently reviewed the methods through which the central and autonomic nervous systems govern the heart, as well as how their dysfunction negatively affects cardiovascular function. The goal of this paper is to give an up-to-date review of the clinical manifestations of stress-related cardiomyopathy syndromes, examine potential causative pathways, and draw parallels [1].

Despite the fact that there is no conventional treatment for takotsubo cardiomyopathy, most people recover completely within a month. Angiotensin-converting enzyme (ACE) inhibitors and angiotensin-receptor blockers (ARB), two types of medications used to treat high blood pressure, have been demonstrated in studies to improve survival after the condition has cleared. Beta-blockers, a type of drug that reduces the activating effects of catecholamine's on the heart, are prescribed to a large number of patients. However, there is no empirical evidence that taking beta-blockers helps people with takotsubo cardiomyopathy live longer.

Excessive endogenous or exogenous catecholamine stimulation of the myocardium can cause stress-related cardiomyopathies, which appear to arise during times of increased sympathetic tone. Although there is a lot of clinical commonality among people who have stress-related cardiomyopathy, it's uncertain whether cardiac adrenergic hyper stimulation is the main cause [2].

In most individuals, the symptoms of TC are similar to those of an ACS, with symptoms largely consisting of ischemia-like chest pain and ischemia-like ECG abnormalities. As a result, TC is now listed as an important differential diagnosis of ACS in both US and international standards. In 27 percent of recorded cases, acute emotional stress preceded presentation with TC, according to reports. Death of a family member or pet, public speaking, heated arguments, financial loss, surprise parties, traffic accidents, and natural calamities such as earthquakes have all been reported as emotional precipitants. After a large earthquake in Japan in 2004, over half of the apparent ACS was discovered to be TC. Approximately 38% of TC cases occur in the United States [3].

The prevalence of stress cardiomyopathy in those who have been subjected to physical or emotional stress is unknown. In a prospective analysis of 92 patients admitted to a medical critical care unit with a non-cardiac diagnosis and no prior history of cardiac disease, 26 individuals (28%) demonstrated left ventricular (LV) apical ballooning, which is consistent with stress cardiomyopathy. In 20 of these individuals, LV function returned to normal after an average of seven days. Sepsis was the only predictor of LV apical ballooning in multivariable analysis. The high rate of transitory LV apical ballooning in this series needs to be confirmed in bigger studies; however it appears that this is a typical occurrence in a medical intensive care unit [4].

Transient Stress Cardiomyopathy (TSC) is a rare cause of reversible Left Ventricular (LV) failure with symptoms that are similar to those of acute coronary syndromes (ACS), especially ST-segment elevation myocardial infarction (STEMI) (1). A postmenopausal woman with abrupt chest discomfort, dyspnea, or syncope and an identified "trigger" is the typical patient (i.e., an acute emotional or physiologic stressor). Due to the unique form of the LV near the end of systole, takotsubo cardiomyopathy, the Japanese word for octopus trap, was first documented by Japanese investigators. "Broken heart syndrome," "stress cardiomyopathy," and "apical

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ballooning syndrome" have all been used to characterise it in the literature. The following are some of the clinical features that are now present: Chest pain/pressure, dyspnea, and/or syncope are all symptoms of a heart attack [5].

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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