Broken Heart Syndrome

■ Lois S. Marshall, PhD, RN

ABSTRACT: **Individuals can die from a broken heart. Although the concept of actually dying from a broken heart used to be thought of as an old wives' tale, there is now evidence that one's heart can suffer a severe and sometimes fatal physiologic consequence after a severe emotional stressor. The body's response to stress, both positive and negative stress, is the release of stress hormones or catecholamines that can have varied and extreme effects on the body. Stress cardiomyopathy, otherwise known in lay terms as broken heart syndrome, occurs when the cascading effects of catecholamines lead to changes in the cardiac system, particularly the coronary arterial system and/or the cardiac cells, which can lead to cardiac dysfunction. With prompt diagnosis, including an accurate history of a recent emotional stressor, this condition can be treated and is reversible in most cases. But in the small percentage of cases that may be fatal, those individuals do indeed die from a broken heart. (J Radiol Nurs 2016;35:133-137.)**

KEYWORDS: **Stress; Heart disease; Takotsubo cardiomyopathy; Women; Radiology nursing.**

INTRODUCTION

She lost her only son from a massive cardiac arrest when he was only 47 years old. It was unexpected and sudden; and he was so young and always healthy. He died in New York. She lived in Miami where he was now to be buried. It took days to bury him as a hurricane was potentially going to strike in Miami. Flights were delayed in and out. Her anxiety deepened. Her tears continued to flow. Finally, it was time. Against her religious beliefs, she had the cemetery open the coffin to be sure it was her son, to say her final goodbye.

For the next 4 months, she never quite recovered. She cried in disbelief. She questioned why over and over again. She started to pull away from her husband, remaining adult children, and friends. She spent extra time with her adult granddaughter, her best friend. It was as if she knew … her heart was breaking.

She had just been to the doctor for a checkup. Everything was ok, she was told. But it was not, and she was not. And 4 months to almost the day her only son died, she collapsed and died too. No matter what the death certificate indicated as the cause of death, we knew, it was a broken heart that took her ….

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BACKGROUND/EPIDEMIOLOGY/ETIOLOGY

Stress or stress-induced cardiomyopathy (SICM) is also known as Takotsubo cardiomyopathy and broken heart syndrome. No matter the terminology, this transient left ventricular apical ballooning syndrome (Golabchi & Sarranfzadegan, 2011) occurs secondary to intense emotional or physical stressor(s). It is also often referred to as a similar event to acute heart failure (2011). Broken heart syndrome was first described by the Japanese in the early 1990s. The midventricle and apex of the heart, when viewed on echocardiography or cardiac catheterization, appeared as a spherical bottle with a narrow neck or pot-like shape, in time of heart systole (2011), which resembled the old Japanese octopus trap called, Takotsubo.

SICM is primarily found in postmenopausal women, although males can develop this condition as well. The predominant age range for development of SICM is 60 years and older. This condition has been found to occur in individuals even who are considered healthy.

The incidence of broken heart syndrome is not really known as of this time. Because it is often misdiagnosed as a myocardial infarction, statistics on the incidence of stress cardiomyopathy are not readily available or accurate up to this time. Deshmukh et al. (2012) did find that Takotsubo cardiomyopathy was diagnosed in approximately 0.02% of all hospitalizations in the United States using data from the *International Classification of Diseases, Ninth Revision* (2008). In addition, it should be noted that since 1990, when stress cardiomyopathy was first described in Japan, it has become increasingly recognized around the world, thus one would expect more accurate incidence statistics to become more readily available.

Precipitating factors that may lead to the development of SICM include a diverse group of severe emotional and/or physiologic stressors. The term broken heart syndrome was given for this disorder as often the emotional stressor was determined to be grief and loss of a loved one. In addition, other stressors that have been identified include fear, extreme anger, surprise (good and bad), financial difficulties, losing one's job, and a frightening medical diagnosis. Physiologic stressors include diverse chronic diseases, congestive heart failure, transplantation, seizures, diabetes, hypothyroidism, pheochromocytoma, and other severe medical illnesses. This may be related to changes in hormonal levels in these patients especially as the body responds with an influx of stress hormones.

As stress increases, the body responds with a cascading hormonal response, predominately catecholamines. The catecholamines produce a fight or flight physiologic response, which overwhelms the cardiac muscle as there is a large influx of adrenaline that may lead to narrowing of the arteries supplying the cardiac muscle with blood flow and thus oxygen. Thus, the presenting symptoms occur, similar to a myocardial infarction, which will be elaborated on further down in this article. Alternatively, the adrenaline may bind to the cardiac cells directly, which causes large amounts of calcium to enter the cells, thus leading to temporary cardiac dysfunction. In either case, it is postulated that the cardiac cells are stunned by adrenaline/stress hormones, but the cells are not killed. This in essence produces a condition, which is generally temporary and reversible.

In a review of studies related to SICM, Golabchi and Sarranfzadegan (2011) described a more detailed explanation of the catecholamine response within the heart. Catecholamine-induced myocardial stunning, as evidenced by elevated serum catecholamine, has been found in more than 70% of affected patients (Pilgrim & Wyss, 2008). Myocardial scintillography (diagnostic technique in which a two-dimensional picture of internal body tissue is produced through the detection of radiation emitted by a radioactive substance administered into the body [Merriam-Webster, 2016]) with I-metaiodobenzylguanidine in these patients cleared a decreased uptake of radiotracer in several segments of the left ventricle, emphasizing a severe adrenalin secretion produced by stress (Prasad, Lerman, & Rihal, 2008; Sharkey et al., 2005; Soares-Filho et al., 2010; Tsuchihashi et al., 2001). In patients with SICM, the large interdivisional differences in I-metaiodobenzylguanidine may reflect varied responses to adrenergic stimulation. Other studies reflect that there may be a genetic inheritance at adrenal synthesis, functions, storage, and elimination that may play a role in SICM in some patients (Sharkey et al., 2010).

In relation to the greatest incidence of SICM in postmenopausal women, Prasad et al. (2008) hypothesized that reduced estrogens and the effect on the microvascular system after menopause may be the main cause of SICM in this population. It has also been hypothesized that sex hormones may influence the sympathetic nervous system, and in turn, this may affect coronary vasoreactivity (Gianni et al., 2006). In other words, the activation of the sympathetic nervous system may cause an increase in a tendency to spasm in the cardiovascular system. In addition, Ueyama et al. (2003) found in animal studies that estrogen attenuates immobility effects of stressors on the myocardium (Golabchi & Sarranfzadegan, 2011).

Kleinfeldt et al. (2009) described a genetic link to SICM. In their study, they described the detection of a mutation in *FMR1* gene (alleles with sizes between 40 and 55 triplet permutations) in patients with first-time SICM. Other research by Kumar, Holmes, and Prasad (2010) is examining case studies related to familial apical ballooning.

Johns Hopkins University has also identified characteristics associated with stress cardiomyopathy (Acute Stress Cardiomyopathy, 2016). They found in their studies (2005) of postmenopausal women, with a median age of 63 years, that circulating plasma catecholamines were found to be 30 times higher than two-thirds of the participants.

The American Heart Association (2016) has reported that there are established ties between the development of SICM and some mental health disorders, particularly depression. This clearly makes sense, even to the novice, as changes in mental status can alter hormonal levels, particularly stress hormones. This can lead to the cascading effects of catecholamines as described previously.

Last, some of the pharmacologic agents used to manage the aforementioned physiologic disruptions to a patient may also be related to the development of SICM. These medications include epinephrine, duloxetine (Cymbalta), venlafaxine (Effexor XR), and synthroid. The impact of these medications may be related to hormonal changes that take place, which in turn can affect the cardiac muscle, similar to the one described previously.

CLINICAL PRESENTATION

The presenting clinical symptoms of SICM would be similar to those reported with a myocardial infarction. The most commonly reported symptoms are chest pain/angina pain and dyspnea. The patients may also complain of feeling their heart skip a beat, flutter, or other changes in cardiac rhythm. There may also be