

Hospital Peer Review

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Hospital Peer Review is a monthly newsletter sponsored by the Rural Healthcare Quality Network to alert Critical Access Hospitals regarding findings from the Peer Review Program. Summarized are a few of the key findings and best practices that would be helpful for other critical access hospitals to be knowledgeable about. This newsletter is edited by Myron Bloom, Medical Director and he can be reached at drmbloom@msn.com.

Beware of the Angry or Broken Heart

Can surges of emotion or unrelenting sadness lead to arrhythmia, myocardial infarction, or heart failure? The answer is yes. So we must not be cavalier, writing off symptoms as simply stress, when a despondent, elderly lady or angry older man present with atypical chest pain or shortness of breath. A recent study observed that in the year following a loved one's death, women were more than twice as likely to die than normal and men more than six times as likely.¹

A study of AICD shock-treated ventricular arrhythmias found an association in those patients who demonstrated significantly higher scores on measures of anger or anxiety. Studies have demonstrated that these emotions can precipitate arrhythmia, likely due to associated increases in sympathetic arousal. Alternatively, it may be that chronic, trait-based psychological factors such as anger and anxiety heighten the risk of arrhythmia due to chronic influences on sympathetic-parasympathetic balance.²

Duke University investigators evaluated emotional state and risk for ischemia in patients with coronary artery disease who recorded their emotions, physical activity, and cardiac symptoms in a diary, roughly every 20 minutes, during the waking hours of a 48-hour study while an ambulatory electrocardiographic monitor recorded ischemic events. Patients abstained from taking anti-ischemic medications during the study. The 58 patients underwent 2,760 hours of monitoring, during which 388 episodes of ischemia were observed. After adjusting for time of day and physical activity level, psychological stress was associated with a 2.2-fold higher likelihood of ischemia similar to moderate and light physical activity (2.1-fold higher risk), while heavy physical activity was associated with a 13.2-fold higher risk of ischemia. Interestingly, positive emotions, such as happiness and feeling "in control," were correlated with a small decrease in ischemic events.³

The number "4" and the word for death are pronounced similarly in Mandarin, Cantonese, and Japanese; and some Asian hospitals do not have fourth floors for that reason. Investigators hypothesized that Chinese and Japanese Americans would have higher mortality rates on the fourth day of the month and that white Americans (controls) would not. In an analysis of national mortality records for Chinese, Japanese, and white Americans from 1973 to 1998 (with control matching starting in 1989), they found heart disease deaths were more common on the fourth day of the month than during the rest of the month among Chinese and Japanese

Americans (RR, 1.13; 95% CI, 1.06-1.21) but not among white Americans (RR, 1.02; 95% CI, 0.99-1.05). The authors could find no explanation for this effect, which was not observed for other causes of death.⁴

Several years ago, investigators evaluated 19 previously healthy adults (18 female) who presented with chest pain or shortness of breath after an emotionally stressful event. Five had abnormal ECGs: 2 with ST-segment elevation and three with T-wave inversions. A prolonged QT interval was evident at presentation in five patients and within 48 hours of symptom onset in all 19. Only mildly elevated peak troponin and creatine-kinase levels were found. Echocardiography revealed a severely depressed LV systolic function (median LV ejection fraction of 20%), rebounding to 45% after about 4 hospital days and back to normal during outpatient follow-up (median 21 days after presentation). Eighteen of the 19 patients had normal coronary arteries. The 19 subjects were found to have median plasma catecholamine levels two to three times higher than control MI patient levels.⁵

The syndrome of stress-related reversible cardiomyopathy, commonly referred to as the "Broken Heart Syndrome," mimics myocardial infarction and is found generally in patients without historical cardiovascular risk factors or coronary vascular disease. The syndrome is more commonly seen in post-menopausal women (average age mid 60s), but may occur in childhood or young adults. The onset of signs and symptoms is usually sudden and usually occurs after a severe emotional (death of a loved one) or physical stressor (car wreck, severe asthma attack, etc.). Initial signs and symptoms resemble those of acute coronary syndrome (chest pain and dyspnea) and ECG changes and elevated cardiac enzymes are common. Because of acute left ventricular dysfunction, many patients develop pulmonary edema, congestive heart failure, or even cardiogenic shock. The syndrome is also called "takotsubo cardiomyopathy" because ventricular imaging shows a rounded, hypokinetic apex with a narrow, hypercontracted base during systole; with an echocardiographic appearance that resembles a pot historically used in Japan to catch octopus (*tako* in Japanese means octopus; *tsubo* means pot). The syndrome is thought to cause between 1.5% and 2.2% of Q-wave and ST-segment acute coronary syndrome cases with estimates of mortality rates ranging 1-3.2%.^{6,7,8}

Common ECG findings in takotsubo cardiomyopathy are ST-segment elevation (67-75%), typically in the anterior precordial leads, and T-wave abnormalities (61%), but ECGs can have normal findings (15%).^{8,9} Evolutionary changes with ST-segment elevation normalization and T-wave inversion are likely to occur in the first few days.

Because there is no way to immediately distinguish the signs and symptoms of takotsubo cardiomyopathy from those of myocardial infarction caused by acute coronary thrombosis, an urgent coronary catheterization is indicated. A diagnosis of takotsubo cardiomyopathy is suspected when a culprit vessel is not present to explain the patient's degree of left ventricular dysfunction and the diagnosis is confirmed by observation of the typical octopus pot morphology of the left ventricle on echo. A triggering emotional or physical stressor is not necessary to make the diagnosis. However, once takotsubo cardiomyopathy is diagnosed, treatment is primarily supportive.

Because takotsubo cardiomyopathy is initially indistinguishable from acute coronary syndrome, immediate treatment should include the usual management of coronary ischemia and pulmonary edema: telemetry, aspirin, antiplatelet and/or glycoprotein IIb/IIIa agents, anticoagulants, nitrates, β -blockers, and diuretics. Acute complications occur in approximately 20% of patients,

including pulmonary edema, heart failure, cardiogenic shock, dysrhythmias, left ventricular thrombus formation, and death. Late complications are rare because the syndrome is reversible.

Aspirin and antiplatelets can be discontinued unless there is concomitant vascular disease. β -blockers may be continued to protect against catecholamine sensitivity though, predisposing to this syndrome. Heparin and coumadin should be used if apical thrombus is present or likely because of a severe contractile defect. A repeat echocardiogram should be obtained to confirm that contractile abnormalities have resolved.

The dominant hypothesis for takotsubo cardiomyopathy (TCM) is stress-induced catecholamine toxicity and subsequent stunning of the myocardium. Endomyocardial biopsy of patients with takotsubo cardiomyopathy demonstrates reversible focal myocytolysis, mononuclear infiltrates, and contraction band necrosis. Also, cases of takotsubo cardiomyopathy have been reported in the literature following cocaine, methamphetamine, and excessive phenylephrine use.^{10, 11}

Another theory for takotsubo cardiomyopathy is multivessel coronary spasm. A systematic review of seven studies identified three studies in which provokable multivessel spasm occurred in 13 of 73 patients (18%) with the syndrome.⁶ In other cases, intravascular ultrasound has been used to image atypical LADs that were found to bend around the apex and extended along the diaphragmatic left ventricle in five patients with takotsubo cardiomyopathy, finding ulcerated atherosclerotic lesions in each patient's LAD. It was postulated that in these patients, the wide-spread apical akinesis was caused by transient occlusion and an aborted myocardial infarction.¹² The diagnosis requires management as if an acute myocardial infarction, echocardiography, and a trip to the cath lab - but once made, is generally reassuring and reversible.

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1. Spreeuw J and Wang X. Modeling the Short-Term Dependence Between Two Remaining Lifetimes. March 27, 2008.
 2. Burg MM, Lampert R, Joska T, et al. Psychological Traits and Emotion-Triggering of ICD Shock-Terminated Arrhythmias. *Psychosomatic Medicine* 66(6):898–902.
 3. Gullette ECD, Blumenthal JA, Babyak M, et al. Effects of Mental Stress on Myocardial Ischemia During Daily Life. *JAMA*. May 1997;277(19):1521-1526.
 4. Phillips DP, Liu GC, Kwok K, et al. The Hound of the Baskervilles Effect: Natural Experiment on the Influence of Psychological Stress on Timing of Death. *BMJ*. December 2001;323:1443-1446.
 5. Wittstein IS, Thiemann DR, Lima JAC, et al. Neurohumoral Features of Myocardial Stunning Due to Sudden Emotional Stress. *NEJM*. February 2005;352(6):539-548.
 6. Bybee KA, Kara T, Prasad A, et al. Systematic Review: Transient Left Ventricular Apical Ballooning: A Syndrome that Mimics ST-Segment Elevation Myocardial Infarction. *Annals of Internal Medicine*. December 2004;141(11):858–865.
 7. Balién MR, de Hoyos EA, Martinez, AL, et al. Reversible Myocardial Dysfunction in Critically Ill, Noncardiac Patients: A Review. *Critical Care Medicine*. June 2002;30(6):1280–1290.
 8. Donohue D and Movahed M. Clinical Characteristics, Demographics and Prognosis of Transient Left Ventricular Apical Ballooning Syndrome. *Heart Fail Rev*. December 2005;10(4):311–316.
 9. Gianni M, Dentali F, Grandi AM, et al. Apical Ballooning Syndrome or Takotsubo Cardiomyopathy: A Systematic Review. *European Heart Journal*. 2006;27(13):1523-1529.
 10. Afonso L, Bachour K, Awad K, and Sandidge G. Takotsubo Cardiomyopathy: Pathogenetic Insights and Myocardial Perfusion Kinetics Using Myocardial Contrast Echocardiography. *European Journal of Echocardiography*. November 2008;9:849-854.
 11. Dorfman TA and Iskandrian AE. Takotsubo Cardiomyopathy: State-of-the-Art Review. *Journal of Nuclear Cardiology*. January 2009;16(1):122-134.
 12. Ibanez B, Navarro F, Cordoba M, et al. Tako-tsubo Transient Left Ventricular Apical Ballooning: Is Intravascular Ultrasound the Key to Resolve the Enigma? *Heart*. 2005; 91:102–104.