Recurrent Direct Current Cardioversion Induced Takotsubo Cardiomyopathy. A Case Report and Literature Review

Athanasios Smyrlis MD, Dmitry Yaranov MD, Shazad Khan MD, Ira Galin MD, Jonathan Alexander MD

Danbury Hospital, Western Connecticut Health Network, Yale School of Medicine

Abstract

Stress cardiomyopathy (SCM), also called broken heart syndrome and Takotsubo cardiomyopathy is an increasingly reported syndrome generally characterized by transient systolic dysfunction of the apical and or mid segments of the left ventricle that mimics myocardial infarction, in the absence of obstructive coronary artery disease. Typically patients present within a few hours of exposure to physical or emotional stress. However, the mechanism by which these stressors result in myocardial dysfunction is unclear. Proposed factors include catecholamine excess and coronary vasospasm. We present the case of a 61-year-old female who experienced acute pulmonary edema secondary to stress cardiomyopathy, on two occasions immediately after undergoing elective direct current cardioversion (DCCV) for atrial fibrillation (Afib). After an urgent hospitalization for management of acute left ventricular failure, she made a complete clinical and echocardiographic recovery. The incidence, clinical implications and prognosis of DCCV induced SCM is unknown. Given DCCV for Afib is a common outpatient procedure and DCCV induced SCM can lead to acute clinical deterioration it is important that physicians are vigilant about this newly recognized DCCV complication.

Key words: Cardiomyopathy, direct current cardioversion, LV dysfunction, cardiogenic shock, acute pulmonary edema, takotsubo cardiomyopathy, broken heart syndrome.


Case Report

A 61-year-old female with past medical history significant only for hypertension on treatment with an ACE-I presented with palpitations and was found to have new onset Afib. Initial workup including an echocardiogram and TSH were unremarkable. Beta blocker therapy and rivaroxaban were initiated and outpatient DCCV was scheduled for one month later. She underwent successful DCCV with one 120J shock. Within 6 hours of DCCV she experienced severe shortness of breath at rest for which she did not seek medical attention as it was short lived (less than 12 hours). On follow up office visit one week later she was found to have recurrent Afib. She was started on flecanide and was scheduled for repeat DCCV. Within 4 hours of repeat DCCV she experienced severe shortness of breath. In the emergency room she was noted to be in severe respiratory distress with a respiratory rate of 40, hypoxic saturating 82% on room air. She denied chest pain and review of systems was otherwise negative. Her heart rate was 130 while and blood pressure 110/70. She had elevated JVP and bilateral crackles on lung exam as well as an S3 gallop. Her EKG revealed sinus tachycardia at 129 beats per minute with new Q waves in V1-V4 and non-specific ST changes (Fig. 1). Laboratory tests, including initial cardiac troponin-I levels, were within normal limits. An urgent echocardiogram revealed severe LV dysfunction with apical ballooning, hypercontractile basal segments and an ejection fraction of 20-25% (Figs. 2, 3). After 4 hours of positive pressure ventilation and adequate diuresis with 160mg of IV lasix her symptoms significantly improved. She was admitted to the intensive care unit for monitoring. Repeat ECG revealed A-fib with rapid ventricular response and new anterior T wave inversions. (Fig. 4) Troponin I level peaked at 0.95 ng/mL. Coronary angiogram on day two of hospitalization revealed minimal coronary artery disease. LVEDP was 8mmHg. (Figs. 5, 6, 7)

The final diagnosis of DCCV induced stress cardiomyopathy was made. Upon hospital discharge on day three, her medications included carvedilol, an angiotensin receptor blocker and rivaroxaban. Echocardiogram 2 weeks later on follow up office visit revealed complete resolution of her LV dysfunction while her ECG returned to baseline (Figs. 8, 9, 10).
Discussion and Literature Review

In the case presented, the development of acute pulmonary edema in close temporal relationship with DCCV with characteristic echocardiographic findings of apical ballooning followed by complete recovery within two weeks, strongly support the diagnosis DCCV induced stress cardiomyopathy. It is more than likely that the acute onset of shortness of breath two weeks prior to hospitalization, immediately after the first DCCV represents a milder short lived incidence of stress cardiomyopathy.

We conducted a Pubmed search and identified three case reports of DCCV induced TCM. The first case report by Eggleton et al is that of a 76-year-old woman who presented with acute pulmonary edema and cardiogenic shock 10 hours after elective electrical cardioversion for A-fib. She had complete resolution of LV dysfunction within 6 days. The second case is reported in an 81 year old female who presented with acute pulmonary edema, cardiogenic shock and neurologic deficits 24 hours after electrical cardioversion; she had a full clinical and echocardiographic recovery within one week. Finally, a case of a 67-year-old woman who experienced cardiogenic shock and ventricular tachycardia immediately after DCCV for Afib followed by a prompt clinical recovery was described by Siegfried et al. A common theme in all four cases of DCCV induced TCM described in the literature, is that of an elderly female with a dramatic clinical presentation of acute LV failure with associated shock and or pulmonary edema within hours of elective DCCV for Afib. All patients had a full clinical and echocardiographic recovery in one week on average. SCM has been reported consequent to various forms of electrical stimulation including DCCV, electroconvulsive therapy, and electrocution caused by lightning strike. The pathophysiology of SCM remains unclear. Several mechanisms have been proposed including catecholamine induced myocardial stunning with an apical preference because of a natural increasing gradient of catecholamine receptors from base to apex, ischemia mediated stunning due to epicardial coronary artery disease or spasm of the microvascular coronary bed. Physical or emotional stress may trigger an exaggerated release of catecholamines in certain genetically predisposed patients. A catecholamine surge triggered by the DCCV is quite possible; however the electrical current itself could have a direct effect on the myocardium resulting in stunning. Isolated atrial myocardial stunning has been described after Afib cardioversion.

Although TC is commonly self-limited with an overall good long term prognosis, it can result in acute hemodynamic decompensation that if not promptly treated can lead to death. Among patients with SCM regardless of precipitant the reported in-hospital mortality rates have ranged from 0 to 8% and appear to be unrelated to LV dysfunction at its most severe. Elective DCCV for Afib is a common outpatient procedure with a current recommendation for a brief post procedure patient monitoring for neurologic, arrhythmic and sedation related complications. The incidence of DCCV induced SCM is not well defined, however as delineated in the above cases electrical cardioversion has the potential to result in acute LV failure and rapid clinical deterioration, thus it is important that physicians are vigilant of this newly recognized DCCV complication.

Statement of ethical publishing

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Address for correspondence:

Athanasios Smyrlis
Western Connecticut Health Network
Yale School of Medicine
University of Vermont School of Medicine, United States
E-mail: athan.smyrlis@gmail.com
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