Northwell Health℠

Journal Articles

2014

Takotsubo Cardiomyopathy as a Sequela of Elective Direct-Current Cardioversion for Atrial Fibrillation

J. S. Siegfried
Northwell Health

S. Bhusri
Hofstra Northwell School of Medicine

N. Guttenplan
Northwell Health

N. L. Coplan
Hofstra Northwell School of Medicine

Follow this and additional works at: https://academicworks.medicine.hofstra.edu/publications

 Part of the Cardiology Commons

Recommended Citation

Siegfried J, Bhusri S, Guttenplan N, Coplan N. Takotsubo Cardiomyopathy as a Sequela of Elective Direct-Current Cardioversion for Atrial Fibrillation. . 2014 Jan 01; 41(2):Article 1824 [p.]. Available from: https://academicworks.medicine.hofstra.edu/publications/1824. Free full text article.

This Article is brought to you for free and open access by Donald and Barbara Zucker School of Medicine Academic Works. It has been accepted for inclusion in Journal Articles by an authorized administrator of Donald and Barbara Zucker School of Medicine Academic Works. For more information, please contact academicworks@hofstra.edu.

Case Reports

Takotsubo Cardiomyopathy as a Sequela of Elective Direct-Current Cardioversion

for Atrial Fibrillation

Jonathan S. Siegfried, MD
Satjit Bhusri, MD
Nils Guttenplan, MD
Neil L. Coplan, MD

In takotsubo cardiomyopathy, the clinical appearance is that of an acute myocardial infarction in the absence of obstructive coronary artery disease, with apical ballooning of the left ventricle. The condition is usually precipitated by a stressful physical or psychological experience. The mechanism is unknown but is thought to be related to catecholamine excess. We present the case of a 67-year-old woman who experienced cardiogenic shock caused by takotsubo cardiomyopathy, immediately after undergoing elective direct-current cardioversion for atrial fibrillation. After a course complicated by left ventricular failure, cardiogenic shock, and ventricular tachycardia, she made a complete clinical and echocardiographic recovery. In addition to this case, we discuss the possible direct effect of cardioversion in takotsubo cardiomyopathy. ***(Tex Heart Inst J 2014;41(2):184-7)***

Takotsubo cardiomyopathy (TC) is characterized by the clinical appearance of an acute myocardial infarction and by left ventricular (LV) apical ballooning in a patient who has no obstructive coronary artery disease.[1] Patients typically present after a precipitating physical or emotional stressor. However, the mechanism by which these stimuli lead to cardiac decompensation is unclear—proposed factors include catecholamine excess and coronary vasospasm.[2] We present the case of a patient whose TC was precipitated by elective direct-current (DC) cardioversion for atrial fibrillation.

Key words: *Cardiomyopathies/etiology; electric countershock/adverse effects; recovery of function/physiology; shock, cardiogenic/therapy; takotsubo cardiomyopathy/complications/diagnosis/etiology/physiopathology; treatment outcome; ventricular function, left/physiology*

From: *Department of Cardiovascular Medicine, Lenox Hill Hospital, New York, NY 10021*

Address for reprints: *Jonathan Siegfried, MD, Department of Cardiovascular Medicine, Lenox Hill Hospital, 100 E. 77th St., New York, NY 10021*

E-mail: *JSiegfried@nshs.edu*

© 2014 by the Texas Heart® Institute, Houston

Case Report

In January 2012, a 67-year-old woman was admitted to our hospital after a syncopal episode. She had been cooking breakfast and was able to catch herself before falling. During the event, her shirt caught on fire, but she was not burned. She reported no palpitations or chest pain; however, she had experienced dizziness and diaphoresis minutes after the event.

The patient's medical history included paroxysmal atrial fibrillation and hypertension, treated with spironolactone and metoprolol succinate. Two years before the patient's current presentation, a computed tomographic angiogram had revealed normal coronary arteries. Her metoprolol succinate dose had recently been increased from 12.5 mg/d to 50 mg/d, and her warfarin therapy had recently been discontinued because of hematuria in the presence of a supratherapeutic international normalized ratio.

Upon the patient's presentation, examination revealed an irregular pulse of 126 beats/min and a blood pressure of 106/72 mmHg. An electrocardiogram showed atrial fibrillation and low voltage (Fig. 1A). Laboratory values, including cardiac troponin I levels, were within normal limits. Echocardiograms showed normal LV function, normal wall motion, and a mildly dilated left atrium.

The decision was made to establish sinus rhythm by means of DC cardioversion. The patient was sedated with propofol, and electrical cardioversion via biphasic energy at 200 J was performed, with a resultant rhythm of sinus bradycardia at 30 beats/min. After cardioversion, the patient was hypotensive and lethargic, despite the correction of the bradycardia with atropine and epinephrine. She was intubated for respiratory

protection and was given norepinephrine, vasopressin, and dobutamine for hemodynamic support.

Serial electrocardiograms revealed sinus rhythm with low voltage, diffuse T-wave inversions, and a prolonged QT interval (Fig. 1B). Echocardiograms revealed an LV ejection fraction of less than 0.15. Contraction in only the basal segments of the LV and akinesis in the remaining segments resulted in a ballooning appearance (Fig. 2). The patient's troponin I level peaked at 3.39 ng/mL the next day. Swan-Ganz catheter readings revealed a cardiac index of 1.3 L/min via the thermodilution method. The patient's central mixed venous saturation was 45%. The diagnosis was TC with clinical cardiogenic shock.

The patient underwent hemodynamic support and intravenous diuresis for several days. Six days after the cardioversion, she sustained one minute of monomorphic ventricular tachycardia with associated hypotension, and electrical cardioversion was performed to re-establish sinus rhythm. By hospital day 9, she no longer needed hemodynamic support and was extubated. Her rhythm returned to atrial fibrillation with rapid ventricular response, with intermittent periods of relative bradycardia and associated pauses (the longest, 3 sec). On day 11, a single-lead permanent pacemaker was implanted, and the patient's cardiac rhythm remained atrial fibrillation. An echocardiogram on that day showed improvement in LV systolic function (ejection fraction, 0.35–0.40). Warfarin and β-blocker therapy was resumed before her discharge from the hospital on day 17. An echocardiogram 2 weeks later showed normal LV function and wall motion (Fig. 3). She was doing well and had returned to her baseline functional status.

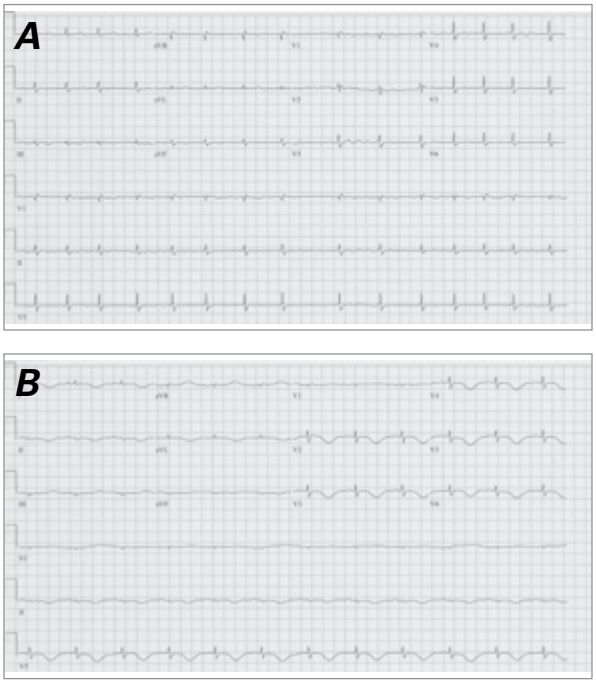

Fig. 1 ***A****) Admission electrocardiogram shows atrial fibrillation with rapid ventricular response, low voltage, and nonspecific ST-T–wave changes.* ***B****) Electrocardiogram after conversion shows sinus rhythm with low voltage, diffuse T-wave inversions, and prolonged QT interval.*

Discussion

Our patient's clinical presentation of sudden LV failure and cardiogenic shock, along with characteristic echocardiographic findings and her subsequent complete recovery, support the diagnosis of TC. Although patients with this clinical course often undergo cardiac catheterization for the evaluation of coronary artery disease, coronary angiograms were not obtained in this patient, in view of the normal evaluation 2 years earlier and the clinical findings.

Takotsubo cardiomyopathy has occurred after numerous precipitating events,[1] including DC cardiover-

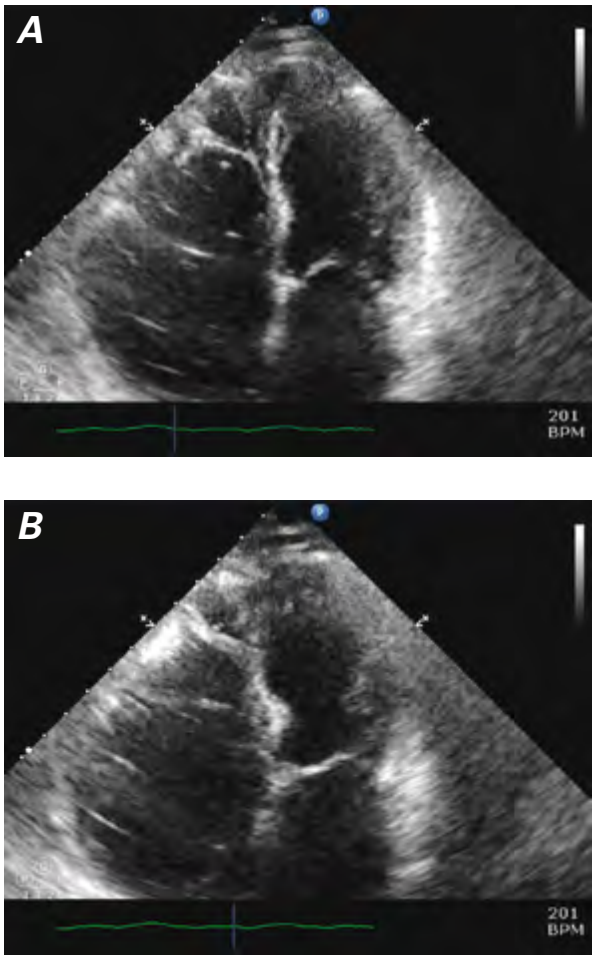

Fig. 2 *Post-cardioversion echocardiogram (apical 4-chamber view) shows a ballooning appearance of the left ventricular apex in* ***A****) diastole and* ***B****) systole.*

sion.[3,4] However, this case differs from previous reports (in which the patients presented with symptoms 10–24 hr after the procedure) in that our patient immediately experienced LV dysfunction and cardiogenic shock—which suggests a more direct pathophysiologic link between electrical cardioversion and TC.

The relevant medical literature provides some insight into how cardioversion might lead to the TC clinical complex. Although the precipitating events vary widely, there is often an association between TC and an elevated catecholamine level.[1,2,5,6] Intense psycho-emotional precipitants that have been associated with TC presumably induce a catecholamine surge. However, the mechanism by which catecholamine excess leads to TC is poorly defined. Results of animal studies suggest that high levels of serum epinephrine directly signal myocardial depression, with an apical preference because of a natural gradient of myocardial catecholamine receptors from base to apex.[6] Other postulated mechanisms include coronary vasospasm, which has been documented in several cases of TC,[7-9] as well as the effect of myocardial bridging.[10] It is unknown whether these mechanisms are the end result of catecholamine excess or are independent causes. Perhaps TC is a heterogeneous disorder with several plausible mechanisms, or perhaps it is the result of several precipitants interacting closely.

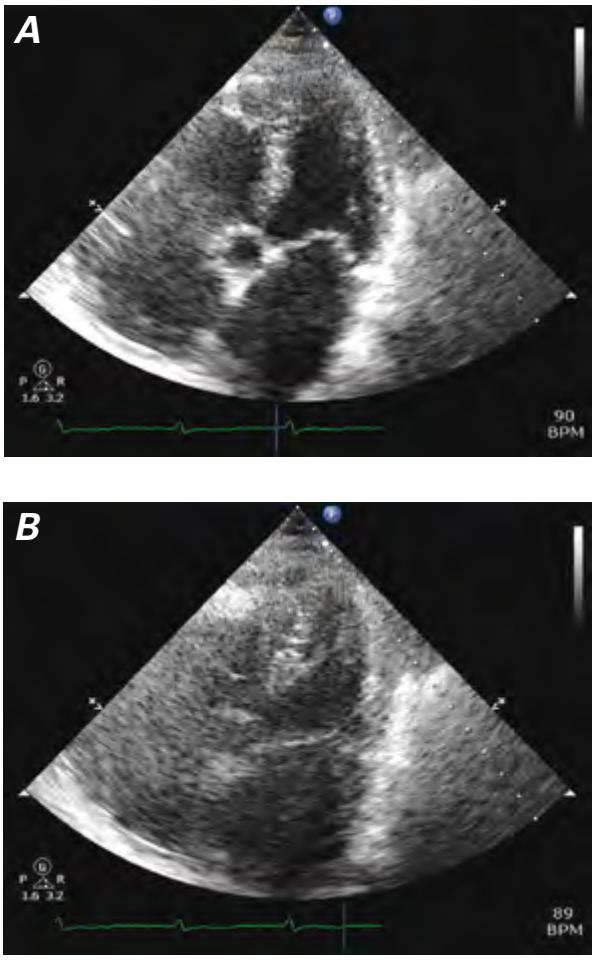

Fig. 3 *Echocardiogram (apical 4-chamber view) 20 days after cardioversion shows resolution of apical ballooning and normal left ventricular ejection fraction in* ***A****) diastole and* ***B****) systole.*

In our patient, catecholamine stimulation during cardioversion might have played a role in the development of TC and cardiogenic shock; however, the cardioversion itself also might have had an effect. Takotsubo cardiomyopathy has been reported consequent to various forms of electrical stimulation: electrical cardioversion,[3,4] electroconvulsive therapy,[11,12] and electrocution caused by lightning strike.[13] To our knowledge, no one has investigated whether electrical stimulation has a direct role in the development of the cardiomyopathy, or whether the cardiomyopathy is merely the result of catecholamine overload.

Although TC is usually self-limited, this case illustrates additional sequelae that can arise in the disease. In general, the sequelae are similar to those in myocardial infarction. Our patient had a fairly rapid and complete recovery, which is also typical of most TC cases. Whereas early reactions can lead to early death, long-term mortality rates tend to be low and appear to be unrelated to LV dysfunction at its most severe.[14,15] It is important to remember that TC can recur years later, although this is unusual.[16,17]

This is not the first report of TC precipitated by DC cardioversion; however, it differs from previous reports in that the procedure was immediately followed by a clinical TC syndrome. This does not rule out other causes of TC, particularly medications that were administered during the procedure; however, a growing body of evidence indicates that electrical stimulation and catecholaminergic stimulus contribute to the pathogenesis of this disorder. Our patient experienced severe sequelae (including LV failure, cardiogenic shock, and ventricular tachycardia) and made a complete recovery, both clinically and echocardiographically. This case underscores the need for further investigation of the effect of myocardial electrical stimulation on the development of TC.

References

1. Prasad A, Lerman A, Rihal CS. Apical ballooning syndrome (tako-tsubo or stress cardiomyopathy): a mimic of acute myocardial infarction. Am Heart J 2008;155(3):408-17.
2. Balkin DM, Cohen LS. Takotsubo syndrome. Coron Artery Dis 2011;22(3):206-14.
3. Eggleton S, Mathur G, Lambros J. An unusual precipitant of tako-tsubo cardiomyopathy. Heart Lung Circ 2008;17(6):512-4.
4. Vizzardi E, Rovetta R, Bonadei I, Quinzani F, D'Aloia A, Salghetti F, et al. A case of tako-tsubo cardiomyopathy after electrical cardioversion. Minerva Med 2013;104(1):115-7.

5. Akashi YJ, Nakazawa K, Sakakibara M, Miyake F, Sasaka K. Reversible left ventricular dysfunction "takotsubo" cardiomyopathy related to catecholamine cardiotoxicity. J Electrocardiol 2002;35(4):351-6.
6. Paur H, Wright PT, Sikkel MB, Tranter MH, Mansfield C, O'Gara P, et al. High levels of circulating epinephrine trigger apical cardiodepression in a β2-adrenergic receptor/Gi-dependent manner: a new model of takotsubo cardiomyopathy. Circulation 2012;126(6):697-706.
7. Nojima Y, Kotani J. Global coronary artery spasm caused takotsubo cardiomyopathy. J Am Coll Cardiol 2010;55(9):e17.
8. Haghi D, Suselbeck T, Wolpert C. Severe multivessel coronary vasospasm and left ventricular ballooning syndrome. Circ Cardiovasc Interv 2009;2(3):268-9.
9. Makaryus AN, Patrick C, Patcha R, Maccaro P. Cardiac arrest in a patient with normal coronary arteries. Am J Emerg Med 2008;26(6):734.e5-7.
10. Boktor M, Mansi IA, Troxclair S, Modi K. Association of myocardial bridge and takotsubo cardiomyopathy: a case report and literature review. South Med J 2009;102(9):957-60.
11. Celano CM, Torri A, Seiner S. Takotsubo cardiomyopathy after electroconvulsive therapy: a case report and review. J ECT 2011;27(3):221-3.
12. Sharp RP, Welch EB. Takotsubo cardiomyopathy as a complication of electroconvulsive therapy. Ann Pharmacother 2011; 45(12):1559-65.
13. Hayashi M, Yamada H, Agatsuma T, Nomura H, Kitahara O. A case of takotsubo-shaped hypokinesis of the left ventricle caused by a lightning strike. Int Heart J 2005;46(5):933-8.
14. Parodi G, Bellandi B, Del Pace S, Barchielli A, Zampini L, Velluzzi S, et al. Natural history of tako-tsubo cardiomyopathy. Chest 2011;139(4):887-92.
15. Regnante RA, Zuzek RW, Weinsier SB, Latif SR, Linsky RA, Ahmed HN, Sadiq I. Clinical characteristics and four-year outcomes of patients in the Rhode Island Takotsubo Cardiomyopathy Registry. Am J Cardiol 2009;103(7):1015-9.
16. Fabbiocchi F, Montorsi P, Bartorelli AL. Seven-year recurrence of left ventricular apical ballooning. Int J Cardiol 2010; 145(3):e86-7.
17. Carigi S, Santarelli A, Baldazzi F, Grosseto D, Santoro D, Piovaccari G. Recurrent takotsubo cardiomyopathy with similar clinical and instrumental signs. Int J Cardiol 2011;148(2): 256-7.