Shock begets shock: A case of direct current cardioversion–induced takotsubo cardiomyopathy

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Introduction

Takotsubo cardiomyopathy (TCM) is a recently emerging syndrome characterized by transient left ventricular wall motion abnormalities unexplained by obstructive coronary artery disease (CAD).[1] Emotional or physical stress is usually illustrated in most cases, with catecholamine excess often playing a mechanistic role.[1] We present a case of TCM induced by an unusual trigger: elective direct current (DC) cardioversion for atrial flutter (AF).

Case report

A 73-year-old male patient with past medical history of hypertension and hyperlipidemia presented to our hospital with symptoms of lightheadedness and dizziness for the past week. His chronic medications included olmesartan 40 mg daily, simvastatin 40 mg nightly, and minoxidil 10 mg twice a day, which were prescribed by an outside provider. Electrocardiogram (ECG) revealed he was in typical AF with high-grade atrioventricular block and variable ventricular rate of 30–50 beats per minute (bpm). The patient did not have history of atrial arrhythmias and did not undergo any prior cardiac procedures. He underwent a transesophageal echocardiogram that showed normal cardiac function with no thrombus, followed by a flutter ablation procedure and permanent dual-chamber pacemaker placement. After the ablation, he was in sinus rhythm, ventricularly paced at a rate of 80 bpm. His pacemaker mode was set as DDD with base rate of 60 bpm, paced atrioventricular delay of 200 ms, and maximum track rate of 130 bpm and he was discharged home on anticoagulation with dabigatran 150 mg twice a day.

Three weeks after discharge he presented to the electrophysiology clinic complaining of recurrence of initial symptoms of dizziness, lightheadedness, and palpitations for the last 3 days. Pacemaker interrogation revealed recurrence of persistent typical AF starting with the onset of symptoms (he maintained sinus rhythm with normal heart rate (HR) for about 18 days, with 90% ventricular pacing). During the last 3 days, the patient was ventricularly paced at the upper tracking rate (130 bpm) 90% of the time, and self-pacing with variable ventricular response otherwise, with average HR running in the 120s for the last 3 days. Given persistence of symptoms and recurrence of the arrhythmia, the decision was made to perform a DC cardioversion to attempt to restore sinus rhythm. Cardioversion was performed using a single 200 J biphasic synchronized shock under moderate sedation with propofol 40 mg and fentanyl 25 mcg with no intraprocedural complications. He did not receive any antiarrhythmic medications prior to the cardioversion. Following the cardioversion, he was in sinus rhythm, ventricularly paced, with a HR in the 70s (bpm). He was observed for 2 hours and discharged home afterward.

Three hours after discharge, emergency medical services were called for severe respiratory distress. The patient was found extremely tachypneic with increased work of breathing, requiring intubation, and was readmitted to the intensive care unit. History was obtained from his wife, who stated that her husband felt sudden-onset retrosternal chest pain and was breathing heavily, along with continuous coughing. On presentation his HR was 105 bpm (regular) and blood pressure was 110/70 mm Hg. Chest radiograph revealed acute pulmonary edema and congestion (Figure 1). ECG showed sinus tachycardia to 100s bpm, in ventricular pacing with

KEY TEACHING POINTS

- Takotsubo cardiomyopathy is a rare but possible complication of direct current cardioversion.
- Patients often suffer severe sequalae, mostly in the form of acute pulmonary edema or cardiogenic shock.
- The condition is usually quickly reversible but requires adequate supportive care with vasopressors and/or mechanical ventilation if needed.

KEYWORDS Cardioversion; Cardiomyopathy; Direct current; Stress-induced; Takotsubo

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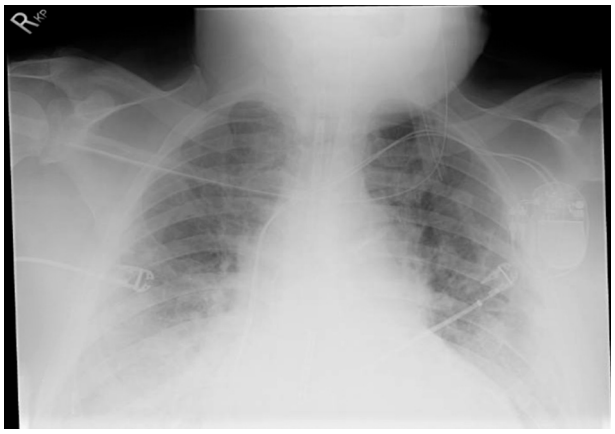

Figure 1 Presenting chest radiograph showing pulmonary edema and congestion.

no ST changes. Blood work revealed elevated cardiac troponin to a peak of 1.3 ng/mL. A transthoracic echocardiogram showed severe left ventricular dysfunction with severe hypokinesis and ballooning of the apex along with hyperdynamic basal segments, with estimated ejection fraction of 25%, highly suggestive of TCM (Figure 2A, Video 1). Coronary angiography showed no obstructive CAD supporting the diagnosis.

He was then managed with furosemide for preload reduction, and his oxygen requirements improved dramatically in the next 24 hours; he was successfully extubated the following day and weaned off oxygen support. Repeated ECGs showed diffuse T-wave inversion and QT interval prolongation (Figure 3) and newly diagnosed atrial fibrillation. He was started on carvedilol with resumption of his home medications. Repeated transthoracic echocardiogram 3 days later revealed normalization of the ejection fraction (60%) with resolution of the wall motion abnormalities (Figure 2B, Video 2). He was thus diagnosed with DC cardioversion–induced TCM and discharged home in a stable condition. Currently, the patient is still being followed in our clinic for various conditions and all of his subsequent echocardiograms showed normal function, with no recurrence of the TCM.

Discussion

Diagnosis

TCM diagnosis is challenging owing to the heterogeneity of symptoms and limited understanding of its pathophysiology. The modified Mayo Clinic criteria remain the most widely used criteria to establish the diagnosis.[2] The following 4 criteria should all be met to diagnose TCM: (1) transient left ventricular wall motion abnormality (hypokinesis, akinesis, or dyskinesis) extending beyond a single epicardial coronary vessel distribution, (2) absence of an obstructive CAD in the territory of the wall motion abnormality to explain the degree of dysfunction, (3) ECG changes suggestive of ischemia (ST elevation or T-wave inversions) or cardiac troponin elevation, and (4) the absence of pheochromocytoma or myocarditis. A recently published expert consensus proposed refined criteria that broaden the diagnostic scope and suggest the use of a diagnostic scoring system.[3]

Pathophysiology

The pathophysiology of TCM remains unclear, with multiple proposed mechanisms. Deleterious cardiac effects of excess catecholamines in times of stress is one of the widely accepted concepts.[3] Elevated catecholamines and reversible

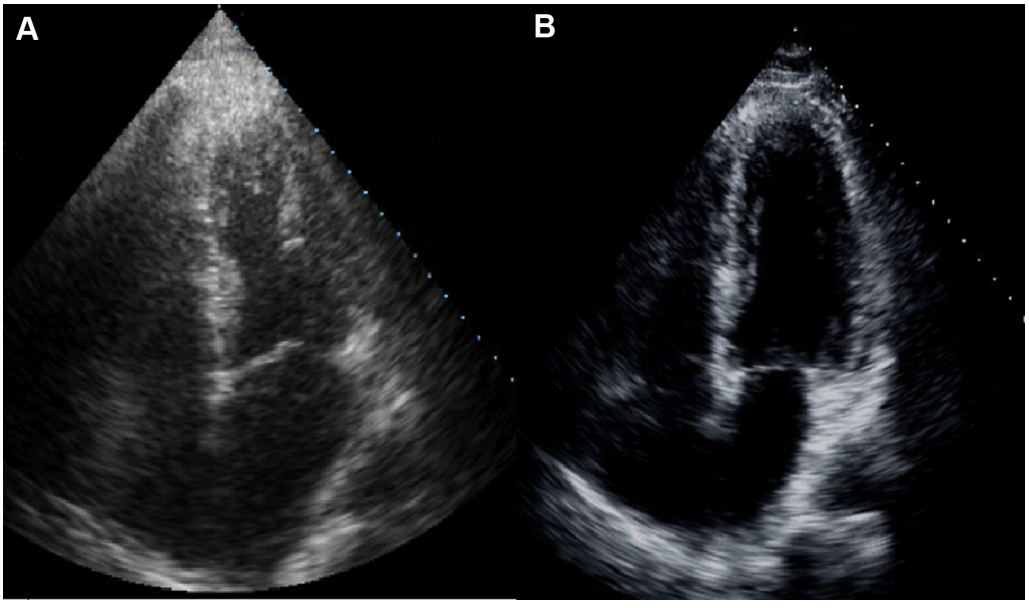

Figure 2 Transthoracic echocardiograms showing **A:** presenting left ventricular apical ballooning and **B:** resolution.

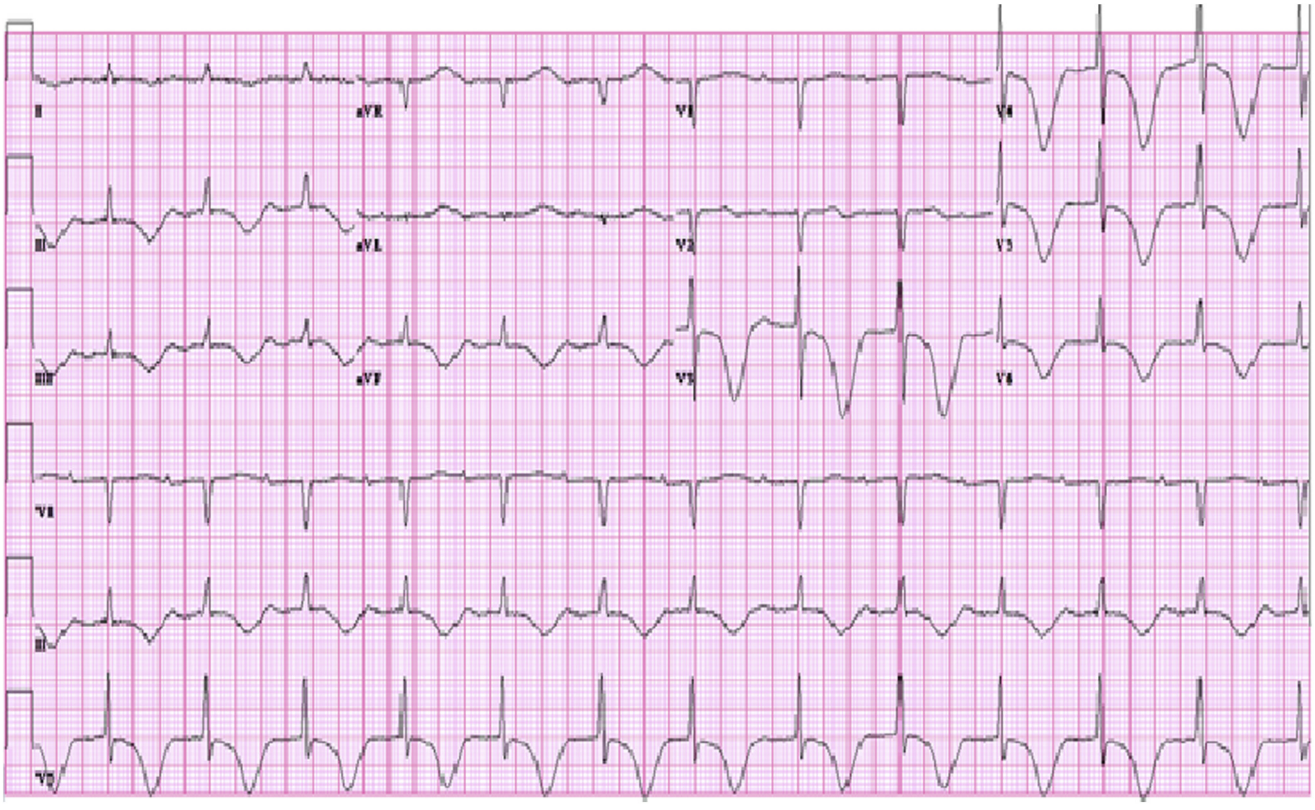

Figure 3 Electrocardiogram showing diffuse T-wave inversions and QT interval prolongation.

left ventricular ballooning consistent with TCM was demonstrated in a rat model with immobilization-induced stress.[4] The exact mechanism of catecholamine-induced cardiac injury remains speculative, with direct cardiotoxicity[5] and coronary vasospasm[6] being among several proposed theories. At the molecular level, it was suggested that high levels of epinephrine result in the paradoxical conversion of apical myocyte beta-2 adrenergic signaling protein from the stimulatory Gs to the inhibitory Gi form, resulting in negative inotropic effect. Those observations, coupled with the differential increased density of beta-2 adrenergic receptors on the heart apex, might help explain the characteristic apical wall motion abnormalities seen in most cases of TCM.[7]

Electrical injury

Electrical shocks such as lightning strikes[8] and electroconvulsive therapy[9] have been previously described as rare

Table 1 Prior case reports

Study	Year	Age	Sex	Underlying rhythm	Presenting symptom / disease process	ECG changes	Complications	Time to resolution echocardiogram
Eggleton et al	2008	76	Female	Atrial fibrillation	SOB/pulmonary edema	TWI and QT prolongation	Cardiogenic shock-vasopressor support	6 days
Vizzardi et al	2013	81	Female	Atrial fibrillation	SOB/pulmonary edema	TWI	Cardiogenic shock-vasopressor support	7 days
Siegfried et al	2014	67	Female	Atrial fibrillation	Lethargy/shock	TWI and QT prolongation	Cardiogenic shock-vasopressor support, mechanical ventilation, sustained ventricular tachycardia, cardiac arrest	14 days
McCutcheon et al	2016	84	Female	Atrial fibrillation	SOB/pulmonary edema	Left bundle branch block	Mechanical ventilation	14 days

ECG = electrocardiogram; SOB = shortness of breath; TWI = T-wave inversion.

triggers of TCM. Our understanding of cardiac effects of electrical injuries comes mostly from victims of lightning strikes. In those patients several mechanisms of cardiac injury have been described, including epinephrine surge, coronary vasospasm, and direct myocardial contusion.[10,11] The overlap of the deleterious cardiac mechanisms between electrical injuries and TCM might explain the pathogenesis.

In our patient, although high volume of right ventricular pacing, rapid pacing during AF, and sedation during the cardioversion could have contributed to some of his cardiac dysfunction, the relatively short duration of the tachyarrhythmia and ventricular pacing, low dose of administered sedatives, classical apical wall motion abnormalities seen in TCM, elevated troponin, ECG changes suggestive of ischemia, and the complete, rapid resolution highly favors the diagnosis of TCM as a cause for his presentation. To our knowledge, we present the fifth case in the literature of DC cardioversion–induced TCM.[12–15] More details regarding prior reports are shown in Table 1. Our case is the first report of DC cardioversion–induced TCM in a male patient despite strong female predominance of TCM in almost 90% of cases.[1] It is also the first case where DC cardioversion was performed for AF, as opposed to atrial fibrillation in prior reports. Our patient suffered severe sequalae and required intensive care and intubation, similar to prior reports, possibly suggesting a severe phenotype of TCM occurring in this particular trigger.

Conclusion

TCM is a rare but possible complication of DC cardioversion that can result in severe sequalae. Awareness of this entity is needed among physicians for prompt diagnosis, treatment, and appropriate patient counseling. Longer monitoring time following cardioversion may be needed to detect such possible complication to provide early supportive care to prevent serious sequalae.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2019.02.013.

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