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Recurrent Post-Cardioversion Takotsubo Syndrome

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ABSTRACT

Takotsubo syndrome (TTS) is characterised by transient regional wall motion abnormalities of the left or right ventricles often associated with new ECG changes (including ST-segment elevations, ST-segment depressions, T-wave inversions and QTc prolongation) in the absence of a culprit atherosclerotic artery which could explain the ventricular dysfunction. In this article we present an unusual case of recurrent TTS following electrical cardioversion in a 61 year old female patient who was admitted to our unit.

Keywords: TTS; ECG

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Case Presentation

A 61-year-old female was admitted to our hospital for an elective cardioversion of atrial flutter. Her past medical history included multiple previous cardioversions for atrial fibrillation and flutter and a radiofrequency ablation. Her chronic medications included sotalol 80mg twice daily, bumetanide 1mg twice daily, ramipril 5mg once daily, acenocoumarol according to international normalised ratio (INR), lormetazepam 1mg once daily, duloxetine 60mg once daily, diosmine/hesperidin 450/500mg once daily, ibandronic acid 150mg once a month and ipratropium bromide/fenoterol puffs when needed.

On admission her electrocardiogram (ECG) showed an atrial flutter with 1:2 conduction. Her only symptom was mild exertional shortness of breath and her blood results showed no evidence of infection and normal renal, hepatic and thyroid functions. The pre-cardioversion echocardiogram showed mixed aortic and mitral valve diseases with a good systolic left ventricular function and no visible blood clots.

The cardioversion was carried out the morning following her admission to hospital. She was sedated using 20mg of etomidate and returned to sinus rhythm after two consecutive shocks: the first at 30J following which the flutter converted into atrial fibrillation and the second shock at 70J following which she switched to sinus rhythm. There were no immediate post-procedural complications and the patient was kept in the cardiology ward for overnight surveillance as per hospital procedure.

In the evening following cardioversion, the patient started experiencing sudden onset severe dyspnoea following which she suffered a cardiac arrest. Upon arrival of the cardiac arrest team, cardiopulmonary resuscitation (CPR) had already been commenced. The patient was in pulseless electrical activity, which appeared to be sinus rhythm and CPR was continued for a total of 5 minutes during which she received 1mg of adrenaline in total. Upon return of spontaneous circulation (ROSC) she was rapidly intubated and transferred to the intensive care unit (ICU).

Her initial chest radiograph showed only mild vascular congestion and blood results revealed a small transient rise in cardiac troponins. Serial ECGs made following ROSC showed progressive QTc prolongation and diffuse inversion of T waves in the anterior leads.

Post-resuscitation echocardiography showed a greatly diminished systolic function of the left ventricle with diffuse apical akinesia typical of a Takotsubo syndrome (TTS) and an estimated ejection fraction of 20%. Subsequent coronarography showed non-significant stenotic lesions of the right coronary artery, the left anterior descending and the left circumflex arteries with no obstructive coronary artery disease (CAD) supporting the initial diagnosis.

She remained intubated and required mechanical ventilation for 20 days. On two occasions during her stay in ITU, the patient developed septic shock requiring up to 20mcg/kg/min of noradrenaline secondary to an infected line and a respiratory infection. Her cardiac rhythm reverted back to atrial fibrillation and following unsuccessful chemical cardioversion using amiodarone she was electrically cardioverted with a single shock of 50J without any procedural complications.

She also suffered from an ischemic cerebrovascular accident (CVA) secondary to the episodes of hypotension with a post-ICU MRI showed a small 3mm ischemic lesion in the right parietal cortex.

A control echocardiography 8 weeks after the initial presentation showed improved contractility of the middle and apical segments of the left ventricle with an ejection fraction at 69%. This permitted the reintroduction of the ACE-inhibitor (ramipril) and commencement of a betablocker (bisoprolol) and spironolactone. Therapeutic anticoagulation was continued using apixaban in view of the CVA.

Retrospective analysis of the patient's past medical history revealed a second episode suggestive of a mild Takotsubo syndrome, which had occurred a year earlier. The patient had been admitted for an electric cardioversion of atrial fibrillation. She had been briefly sedated using

etomidate and returned to sinus rhythm after a single 70J shock with no immediate post-procedural complications.

A few hours after the cardioversion, the cardiac arrest team was called in as she experienced a presyncopal episode following which she was in severe respiratory distress requiring intubation and mechanical ventilation. During this initial episode she remained in sinus rhythm and her initial ECG showed widespread flattening of the T waves. She rapidly recovered and was extubated less than 24 hours later. The following day she had a second presyncopal episode associated with severe respiratory distress. On examination she was found to be in atrial fibrillation and had florid pulmonary oedema. Echocardiography showed moderate left ventricular dysfunction with regional wall hypokinesia. Blood results revealed a small transitory rise in troponins and a bacteremia due to *Moraxella catarrhalis,* secondary to a respiratory infection for which she was put on antibiotics. She was managed using non-invasive ventilation and diuretics and was started on amiodarone in view of persistent atrial fibrillation. She rapidly recovered and was discharged home the following week. A control echocardiogram done a month later revealed resolution of the left ventricular wall hypokinesia and a normal ventricular function. The transitory changes seen at the time on echocardiography were attributed to the sepsis for which the patient was successful treated. Retrospectively, this first episode fits into the clinical presentation of a mild Takotsubo syndrome that had not been correctly identified at the time.

Discussion

TTS primarily affects post-menopausal women with the main diagnostic criteria being transient regional wall motion abnormalities of the left or right ventricles often associated with new ECG changes (including ST-segment elevations, ST-segment depressions, T-wave inversions and QTc prolongation) in the absence of a culprit atherosclerotic artery which could explain the ventricular dysfunction or conditions like infectious myocarditis. An emotional, physical or combined trigger can precede the TTS but is not obligatory [1]. Although transient myocardial dysfunction is considered to be essential for the diagnosis of TTS there have been recent reports showing that full myocardial recovery can take more than 4 months after the acute event [2].

There have already been a number of TTS cases following electrical cardioversion that have been described in the literature [3-7] and a first episode of TTS is known to predispose for recurrences of the same condition [8]. This is however the first case of recurrent TTS following cardioversion that has to our knowledge been described in the literature.

Although the exact pathophysiology of TTS remains unclear, several studies implicate the sympathetic nervous system and severe catecholamine surges or “sympathetic storm” in the myocardial stunning observed. All catecholamine levels are markedly higher in patients with TTS when compared to patients with coronary occlusion myocardial infarction [9]. The regional wall motion abnormalities observed in TTS might be explained by the anatomical sympathetic innervation of the heart from the left and right stellate and caudal ganglia [10]. A recent study provided histologic evidence showing β-adrenoreceptor desensitization resulting in myocardial stunning which could have a cardioprotective role against catecholamine toxicity [11].

In our patient the first episode of TTS was not correctly identified but was nonetheless treated successfully. She was also successfully cardioverted to sinus rhythm following recurrence of her atrial flutter on multiple occasions prior to her first episode of TTS and prior to the second episode of TTS. The reasons why she developed TTS during these two particular instances are not clear.

Conclusion

Although rare, TTS can present following electrical cardioversion and should make part of the differential diagnosis during the workup of patients who present in acute heart failure in the hours after successful cardioversion. Correctly identifying each such episode is important, as longer monitoring following cardioversion may be needed in these patients to provide early

supportive care and prevent serious complications in case of recurrence.

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