ABSTRACT

Introduction: Takotsubo cardiomyopathy (TTC) is a condition with a good long-term prognosis. However, when the TTC is due to a life-threatening arrhythmia, such as atrioventricular block (AVB), several considerations must be made regarding treatment.

Case Presentation: A 71-year-old woman with a history of ischemic stroke presented after a syncopal episode. Before passing out, the patient was walking, nauseous, lightheaded, dizzy, and short of breath. In the emergency department, the blood pressure was 230/120 mmHg, and the heart rate was 38 beats per minute, but the patient was asymptomatic. An electrocardiogram showed a new-onset 2:1 AVB, bifascicular block, and prolonged PR and corrected QT intervals. An echocardiogram revealed a new-onset ejection fraction of 30% to 35%; hypokinesia of the apex, mid-inferoseptum, mid-anterolateral, apical to mid-inferior, and apical to mid-anterior walls; and hyperkinesis of the basal segments. The cardiac catheterization illustrated normal coronary arteries without significant stenosis. Therefore, the patient was diagnosed with TTC and 2:1 AVB. She was treated with lisinopril and metoprolol succinate and received a dual-chamber pacemaker. At the follow-up visit, the patient’s ejection fraction and hypokinetic segments improved. She denied any recurrence of syncope, and her pacemaker was functioning appropriately.

Conclusion: When AVB or other arrhythmias initiate a TTC, the patient can experience sudden cardiac death and decompensate quickly. Therefore, clinicians should understand this rare but fatal complication because these patients require pacemakers and beta blockers.

INTRODUCTION

Takotsubo cardiomyopathy (TTC) is a disease in which a patient develops transient hypokinesis, akinosis, or dyskinesis with compensatory excessive contraction in the other areas. Usually, this is preceded by a stressor. Despite the possibility of acute life-threatening symptoms and hemodynamic compromise at its onset, the long-term prognosis is generally favorable, and the wall motion abnormality improves within 2 weeks.1,2

In this case presentation, we report a 71-year-old woman who developed new-onset TTC, 2:1 atrioventricular block (AVB), bifascicular block, and prolonged PR and corrected QTc intervals after a syncopal episode. The prevalence of AVB in TTC has been reported at 2.2%3 and 2.9%.4 Although TTC resolves, the comorbid bradyarrhythmias are typically persistent and require the placement of a pacemaker.4,5 This case report was prepared following the CARE guidelines.6

CASE PRESENTATION

A 71-year-old woman with a history of ischemic stroke and chronic lymphocytic leukemia in remission presented after a syncopal episode. The patient stated before passing out that she had walked about 10 feet and was nauseous, lightheaded, dizzy, and short of breath. The patient denied preceding chest pain and palpitations. At first, the patient denied any recent emotional stress or possible triggers, but on further questioning, she admitted to a tense verbal conversation with her son earlier that morning. She was not taking any antiplatelet, anticoagulant, anti-arrhythmic, or beta-blocker medication. In the emergency department, the patient’s vital signs included a blood pressure of 230/120 mmHg and a heart rate of 38 beats per minute, but she was otherwise stable and asymptomatic. Moreover, the physical examination, laboratory investigations, and pan imaging were unremarkable (Table 1). However, an electrocardiogram showed a new-onset 2:1 AVB, bifascicular block and prolonged PR and QTc intervals (Figure 1). An echocardiogram revealed an ejection fraction of 30% to 35%; hypokinesia of the apex, mid-inferoseptum, mid-anteralateral, apical to mid-inferior, and apical to mid-anterior walls; and hyperkinesis of basal segments. The cardiac catheterization illustrated normal coronary arteries without significant stenosis. Finally, the left ventriculography confirmed the 30% to 35% ejection fraction, apical hypokinesis, and basal hyperkinesis. Therefore, the patient was diagnosed with TTC and 2:1 AVB. Subsequently, she was started on lisinopril and metoprolol succinate. The patient underwent an emergent implantation of a dual-chamber pacemaker. The patient was discharged home on daily lisinopril and instructed to follow up in the office after obtaining an echocardiogram in 6 weeks. This echocardiogram showed an improved ejection fraction of 50% and improvement in the mid-to-apical hypokinetic segments and device lead in the right heart chambers.

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Abbreviations: AVB, atrioventricular block; QTc, corrected QT interval; TTC, takotsubo cardiomyopathy
Moreover, the patient had not had a recurrence of syncope or AVB, and her pacemaker was functioning appropriately.

**DISCUSSION**

The differential diagnosis for acute myocardial infarction includes TTC, named after the resemblance of the left ventricular apical ballooning in systole to a Japanese octopus trap. The left ventricular mid-segments, with or without the apex, had transient hypokinesis, akinesis, or dyskinesia, with compensatory excessive contraction in the other areas. Other diagnostic criteria are regional wall motion abnormalities extending beyond a single coronary vascular bed, absence of obstructive coronary disease or angiographic evidence of acute plaque rupture, and presence of new-onset electrocardiographic abnormalities or modest elevations in cardiac troponin. Typically, the trigger of these symptoms is a physical or emotional stressor; thus, it is also known as broken-heart syndrome or stress-induced cardiomyopathy.\(^1\)\(^,\)\(^7\) Therefore, TTC is unlikely to be the precipitating factor, but rather the effect of the AVB. This is proposed to be through an increased catecholamine secretion that produces a vagal response and eventually AVB and TTC. Possible etiologies of this are blood flow disruption by alpha-receptors and multiple branch coronary artery spasm.\(^2\)\(^,\)\(^8\)

With this in mind, beta blockers are potentially indicated to stunt the catecholamine surge, but are limited by the patient’s hemodynamics.\(^9\) Beta blockers have even been reported to prevent the development of arrhythmias.\(^10\) Our patient was not on beta blockers before admission, but based on the above recommendations, we discharged her on metoprolol succinate.

Two studies evaluating AVB among patients with TTC reported a rare prevalence of 2.2% (4/178)\(^3\) and 2.9% (24/816).\(^4\) The difficult task in these patients is determining its pathophysiology. One hypothesis is based on the fact that although TTC resolves rather quickly, the bradyarrhythmias are usually present long term.\(^2\)\(^,\)\(^4\)\(^,\)\(^5\)

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The bradyarrhythmias seen with TTC are: AVB (2.2% [4/178] and 2.9% [24/816]), sinus node dysfunction (1% [3/178] and 1.3% [11/816]), asystole (1.7% [5/178] and 0.5% [4/816]), and pulseless electrical activity (0.3% [2/816]).

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### Table 1. Timeline: relevant medical history and interventions for a 71-year-old woman presenting with a history of ischemic stroke and chronic lymphocytic leukemia in remission

<table>
<thead>
<tr>
<th>Date</th>
<th>Summaries from initial and follow-up visits</th>
<th>Diagnostic testing</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>12/23/2019</td>
<td>The patient presented to the emergency department with acute syncope with prodromal symptoms.</td>
<td>Blood pressure, 230/120 mmHg; heart rate, 38 beats per minute; temperature, 98.2°F</td>
<td>Started lisinopril and metoprolol succinate</td>
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<td></td>
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<td>Electrocardiogram: 2:1 atrioventricular block; bifascicular block; PR interval, 293 ms; QTc interval, 514 ms</td>
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<td>Echocardiogram: ejection fraction, 30%-35%; hypokinesis of the apex, mid-inferoseptum, mid-anteralateral, apical to mid-inferior, and apical to mid-anterior walls; and hyperkinesis of basal segments</td>
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<td></td>
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<td>Cardiac catheterization: normal coronary arteries without significant stenosis</td>
<td>A dual-chamber pacemaker was placed emergently</td>
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<td>Left ventriculography: ejection fraction, 30%-35%, apical hypokinesis, and basal hyperkinesis</td>
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<td></td>
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<td>White blood cells, 12.5 10^3/μL</td>
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<td></td>
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<td>Troponin I, 7 and 10 pg/mL</td>
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<td></td>
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<td>Serum potassium, 3.9 meq/L</td>
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<td></td>
<td>Serum magnesium, 2.0 mg/dL</td>
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<td>Thyroid-stimulating hormone, 7.01 u[IU]/mL</td>
<td></td>
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<tr>
<td></td>
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<td>Free T4, 0.90 ng/dL</td>
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<tr>
<td>12/24/20</td>
<td>The patient presented to the office for a follow-up echocardiogram and denied any recurrence of syncope or atrioventricular block.</td>
<td>Follow-up echocardiogram: ejection fraction of 50%, improvement in the mid-to-apical hypokinetic segments, and device lead in the right heart chambers</td>
<td>Discharged home</td>
</tr>
<tr>
<td>2/7/2020</td>
<td></td>
<td></td>
<td>Instructed to follow up in 6 weeks</td>
</tr>
</tbody>
</table>
Although these bradyarrhythmias have been reported to resolve, the fatal bradyarrhythmia can reoccur and cause sudden cardiac death. Thus, lifelong pacemakers should be considered in these patients, even if the patient becomes asymptomatic. Furthermore, life-threatening arrhythmias have been illustrated to have a lower ejection fraction (39.7% ± 11.0%), longer QTc interval (476.5 ± 74.4 ms and 491 ± 81 ms), longer PR interval (207 ± 96 ms), and longer maximal R-R interval (30.6 ± 14.5 ms). Our patient had an ejection fraction of 30% to 35%, QTc interval of 516 ms, PR interval of 293 ms, and R-R interval of 1452 ms. We placed a dual-chamber pacemaker in our patient. At her follow-up appointment, she had not had a recurrence of syncope, AVB, or TTC, but the decision was made to keep the pacemaker. Because this is a case report, it has the limitation that it can only indicate associations, not causations. Moreover, the association inferred from our patient may not be generalizable to a larger population.

Although bradyarrhythmias should be actively monitored for and expectantly treated, patients with TTC can also develop other dire complications that require urgent intervention. It should be noted that these recommendations are only suggestions based on retrospective and literature reviews, because prospective and clinical trials have yet to be completed. In those who develop heart failure, one should initiate a typical heart failure regimen of beta blockers, angiotensin-converting enzymes or angiotensin receptor blockers, and diuretics. Beta blockers are also indicated to prevent the TTC sequelae of developing a cardiac rupture. Additionally, patients with a history of left ventricular thrombus or embolic complications who develop extensive mid-apical ballooning should receive anticoagulation treatment for a minimum of 2 to 3 months. Finally, cardiogenic shock can be seen in patients with TTC. When due to left ventricular outflow tract obstruction, one should start parenteral beta blockers and intravenous fluids, with phenylephrine being a second-line medication. Due to the obstruction, diuretics, nitroglycerin, and intra-aortic balloon pumps are contraindicated. However, venoarterial extracorporeal membrane oxygenation, left ventricular assist device, or noncatecholamine inotropes such as levosimendan may be indicated in patients with primary pump failure.

CONCLUSION

Increased catecholamine secretion is likely to cause AVB and subsequently TTC. TTC is a rare but life-threatening condition that should be managed expectantly, even if the patient’s symptoms resolve. Furthermore, beta blockers may help decrease the catecholamine surge and prevent the onset or recurrence of an arrhythmia.

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Author Contributions

Rohan Prasad, DO, wrote the Case Narrative and Discussion, conducted the literature review, obtained consent, and edited final draft. Mohammad Fahad Salam, XX, wrote the Abstract and Introduction, assisted in writing the Discussion, conducted the literature review, and edited final draft. Shaurya Srivastava, XX, wrote the Conclusion and References, formatted the table, and edited the final draft. FNU Samreen, XX, obtained the figure, formatted the table, and edited final draft. Zulfiqar Utroo Baloch, XX, assisted in writing the Discussion and literature review, and edited the final draft.

References