Takotsubo Cardiomyopathy
Complicating Sick Sinus Syndrome

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ABSTRACT

Takotsubo cardiomyopathy presents with diverse electrocardiographic changes, possibly masquerading as an acute coronary syndrome, and may occur after psychological, medical, or surgical stress. We describe a case of a 69-year-old woman with sick sinus syndrome, repolarization abnormalities, akinesia of the left ventricular apex, and normal coronary arteries. The patient’s electrocardiographic and echocardiographic abnormalities normalized after the implantation of a permanent pacemaker; cardiac events did not recur within a 5-year follow-up period.

Key words: • pacemaker • sick sinus syndrome • takotsubo cardiomyopathy

Introduction

Takotsubo cardiomyopathy may occur after diagnostic or therapeutic cardiovascular procedures, as well as following emotional and medical stress, in susceptible patients. Patients exhibiting this cardiomyopathy are at risk for the development of torsades de pointes. Takotsubo cardiomyopathy can occur in association with a complete atrioventricular block, but its presence in a patient with newly diagnosed sinus node dysfunction has not previously been reported. We describe the case of a 69-year-old hypertensive woman with sick sinus syndrome and takotsubo cardiomyopathy in whom the electrocardiographic and wall-motion abnormalities were reversed after the implantation of a pacemaker.

Case

A 69-year-old woman was admitted following progressive dyspnea over the previous month. She reported the absence of angina, prolonged chest pain, dizziness, and syncope. Her medical history included chronic hepatitis C and hypertension, which was under control with amlodipine and ramipril; she was not taking any sympathomimetic drugs. She had not experienced any emotional or
physical stress that was likely to be related to stress cardiomyopathy. The results of her physical examination were unremarkable, with the exception of bradycardia; focal neurologic symptoms and signs were absent. A 12-lead electrocardiogram revealed sinus arrest with a junctional escape rhythm, a retrograde P wave, and a repolarization abnormality (Figure 1A); her QT and QTc intervals were 1,120 ms and 844 ms, respectively. The patient had a creatine kinase MB (myocardial band) isoenzyme level of 0.6 ng/mL (upper limit of normal, 3.1 ng/mL) and a troponin I level of 0.16 ng/mL (upper limit of normal, 0.06 ng/mL), but the levels of creatinine, sodium, potassium, calcium, magnesium, bilirubin, and transaminase were within normal limits. An echocardiogram indicated akinesia of the mid- to apical left ventricle (Figure 2A), and coronary angiography was normal. Left ventriculography indicated akinesia of the apex (Figure 3A and B); Holter monitoring revealed a persistent junctional escape rhythm and multiple episodes of sinus pause of >3.0 seconds during the day.

After excluding reversible causes of bradycardia, the patient underwent implantation of a dual chamber pacemaker. Thereafter, her QT interval decreased to 560 ms (Figure 1B) and focal akinesia of the apex was observed after atrial pacing (Figure 2C and 2D). The wall-motion abnormalities in the apex, however, normalized 2 months after the procedure (Figure 2E and 2F). Her electrocardiogram showed an atrial-paced rhythm of 60 bpm and a QT interval of 440 ms at 5 months after the procedure (Figure 1C). The patient was diagnosed with Child-Pugh class A liver cirrhosis caused by hepatitis C at 3 years after the event. Imaging follow-up with computed tomography and ultrasonography did not reveal the presence of an adrenal mass. Over a 5-year follow-up period, the patient did not exhibit any further cardiac events.

Discussion

Takotsubo cardiomyopathy is characterized by wall–motion abnormalities involving the apex or mid–ventricular wall; normal coronary arteries; electrocardiographic changes mimicking acute myocardial infarction; and the elevation of cardiac markers following emotional, physical, or medical stress. In addition to emotional stress, medical or surgical procedures can induce takotsubo cardiomyopathy; catheter ablation of cardiac arrhythmias is one of the precipitating causes of takotsubo cardiomyopathy. Few cases of takotsubo cardiomyopathy after the implantation or malfunction of a pacemaker have been reported. However, to our knowledge, this is the first report of the occurrence of takotsubo cardiomyopathy in a patient with newly diagnosed sick sinus syndrome in whom wall–motion and electrocardiographic abnormalities resolved after atrial pacing.

Similar to a patient in a previous report, the current patient was an elderly woman without a history of cardiac disease. Given that emotional and other medical stresses and the intake of sympathomimetic drugs were ruled out during her detailed history and routine examination, sick sinus syndrome may be regarded as a trigger of takotsubo cardiomyopathy in this patient. Stimulation of the sympathetic nervous system is known to play a critical role in the pathogenesis of takotsubo cardiomyopathy. Therefore, reflex stimulation of the sympathetic nervous system to overcome bradycardia may have accounted for the development of takotsubo cardiomyopathy in this patient. Prolongation of a corrected QT interval is a risk factor for the development of torsades de pointes in patients with takotsubo cardiomyopathy. Considering the role of sympathetic stimulation in the pathogenesis of takotsubo...
cardiomyopathy and that detrimental effects may occur in the presence of bradycardia upon repolarization heterogeneity, urgent cardiac pacing, rather than administration of sympathomimetic drugs, may help reduce the risk of developing torsades de pointes. However, no apparent stressors were found in 11% of patients with takotsubo cardiomyopathy; therefore, the sinus node dysfunction and stress cardiomyopathy observed in the present patient may only be a coincidental phenomena.

Transient electrocardiographic and wall-motion abnormalities can result from diverse causes. Although the ergonovine provocation test was
Figure 2. Apical 4-chamber views of the left ventricle. End-systolic and end-diastolic images at initial presentation (A, B), 2 days after pacemaker implantation (C, D), and 2 months after pacemaker implantation (E, F).

Figure 3. Left ventriculogram of the patient: (A) end-systolic frame and (B) end-diastolic frame.

not performed, the absence of chest pain, gradual improvement of the electrocardiographic abnormality, improvement of the wall-motion abnormality with pacing therapy, and the absence of angina attacks during treatment with a beta blocker suggested that vasospastic angina was
unlikely to be the cause of the electrocardiographic and echocardiographic abnormalities in this patient. Spontaneous recanalization of an infarct-related artery may have caused the wall-motion and electrocardiographic abnormalities. However, the absence of a typical rise and fall in the patient’s cardiac markers was not consistent with an acute myocardial infarction. Acute myocarditis can be excluded in the absence of antecedent viral illness, the absence of elevated cardiac enzymes, and the presence of a typical wall-motion abnormality.

Hidden pheochromocytoma is another well-known cause of takotsubo cardiomyopathy in the absence of an apparent stressful condition. In this case, even though a hormonal assay for pheochromocytoma was not performed, the absence of an intra-abdominal mass during an imaging examination of the abdomen for cirrhosis evaluation and the rapid resolution of clinical manifestations following pacing treatment likely excludes hidden pheochromocytoma as the cause of takotsubo cardiomyopathy.

In conclusion, we report a case of takotsubo cardiomyopathy in a patient with sick sinus syndrome. After implantation of a permanent pacemaker, the patient did not experience any subsequent cardiac events during a 5-year follow-up period.

References