Pacemaker Induced Takotsubo Cardiomyopathy

Jalaj Garg MD
Lehigh Valley Health Network, jalaj.garg@lvhn.org

Kailyn Mann DO
Lehigh Valley Health Network, kailyn.mann@lvhn.org

James Kimber DO
Lehigh Valley Health Network, James.Kimber@lvhn.org

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In 2004, the Mayo clinic identified diagnostic criteria for takotsubo cardiomyopathy, which include: a transient hypokinesia/disksynkinesis of the left ventricle, the absence of obstructive coronary disease, new EKG abnormalities including diffuse ST elevation or T wave inversion, often strongly resembling ST elevation myocardial infarction.1 Oftentimes also include elevated troponin, and the absence of pheochromocytoma and myocarditis.1 Our patient old demonstrate appreciable akinesia via angiography and new elevation of Troponins during this admission. Takotsubo cardiomyopathy has been previously shown to occur status post pacemaker implantation, and presents typically with symptoms of chest pain and dyspnea, and EKG changes resembling acute myocardial infarction.2 In current theory, takotsubo cardiomyopathy may be precipitated by catecholamine toxicity, microvascular dysfunction, or coronary artery vasospasm.3 As in this case, our patient is an elderly female with significant cardiac risk factors, and having experienced recent pacemaker implantation, is predisposed to increased catecholamine release, a favored theory in recent literature.

In a recent review conducted by Potenza et al. in 2014, the onset of takotsubo cardiomyopathy with documented heart failure by echocardiography occurred 10 minutes to 3 days following implantation of pacemaker device.4 In this aspect, this patient’s onset of symptoms was significantly delayed in comparison to the seven cases analyzed.4 Notably, the implantation of a Dual chamber pacemaker in this patient was complicated by a 30% left pneumothorax, which may have contributed to the development of Takotsubo-like symptoms. However, the patient had documented resolution of pneumothorax. Given this complication, the presence of dyspnea on exertion and shortness of breath with new orthopnea may have led early to early misdiagnosis of this condition. Additionally, the complication of pneumothorax during pacemaker implantation may have contributed to the developing pathology of Takotsubo cardiomyopathy and further predisposed our patient to develop these symptoms. In this patient, there was troponin spil as well as angiography demonstrating hypokinesia of the left ventricle in the absence of significant CAD, which were consistent with Mayo Clinic criteria.5 Other evaluation of the patient showed an ejection fraction of 20-25% echocardiogram, and of 25% by other evaluation. Echocardiography was useful in guidance of further clinical decision making for this patient, having demonstrated a severe reduction in Left Ventricular systolic function and status post pacemaker implantation.

References:

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Jalaj Garg, MD, Kailyn Mann, DO, and Jim Kimber, DO
Lehigh Valley Health Network, Allentown, PA

Abstract
Takotsubo cardiomyopathy, also known as stress-induced cardiomyopathy, apical balloon syndrome and broken heart syndrome is an increasing reported syndrome that is generally characterized by transient systolic dysfunction of the left ventricular, usually affecting the apex or mid segments, with absence of obstructive coronary artery disease. The exact pathogenesis remains unknown, however postulated mechanisms include catecholamine excess, coronary artery spasm and microvascular dysfunction. The most common presenting symptom is acute sub-acute chest pain, however some patients present with dyspnea, syncope, shock or electrocardiographic abnormalities. We present a case of an 83-year-old female who developed Takotsubo cardiomyopathy after undergoing a pacemaker implantation. This case provides evidence that TCM should be in the differential when patients develop dyspnea following pacemaker implantation.

Case Report
An 83-year-old female with a past medical history significant for dyslipidemia, hypertension and symptomatic bradycardia associated with 2nd degree heart block status post recent St. Jude’s dual chamber pacemaker presented a day prior to our emergency room with shortness of breadth and dyspnea on exertion that have been worsening over the past week. Over the course of her hospital stay, the patient’s ejection fraction was 25% on echocardiogram. Her global LV function was severely depressed and her ejection fraction was 25%. Coronary angiography demonstrated minor luminal irregularities and no MR or TR was noted. Patient was diagnosed with severe Takotsubo cardiomyopathy short post pacemaker implantation. This case provides evidence that TCM should be in the differential when patients develop dyspnea following pacemaker implantation.

Discussion
In 2004, the Mayo clinic identified diagnostic criteria for takotsubo cardiomyopathy, which include: a transient hypokinesia/disksynkinesis of the left ventricle, the absence of obstructive coronary disease, new EKG abnormalities including diffuse ST elevation or T wave inversion, often strongly resembling ST elevation myocardial infarction. Oftentimes also include elevated troponin, and the absence of pheochromocytoma and myocarditis. Our patient old demonstrate appreciable akinesia via angiography and new elevation of Troponins during this admission. Takotsubo cardiomyopathy has been previously shown to occur status post pacemaker implantation, and presents typically with symptoms of chest pain and dyspnea, and EKG changes resembling acute myocardial infarction. In current theory, takotsubo cardiomyopathy may be precipitated by catecholamine toxicity, microvascular dysfunction, or coronary artery vasospasm. As in this case, our patient is an elderly female with significant cardiac risk factors, and having experienced recent pacemaker implantation, is predisposed to increased catecholamine release, a favored theory in recent literature.

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