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Pacemaker Induced Takotsubo Cardiomyopathy

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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-sternal 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 implantation presented the emergency room c/o shortness of breath and dyspnea on exertion that have been worsening over the past week.

About one week prior, the patient had a St. Jude's Dual chamber pacemaker with fluoroscopy implanted for symptomatic bradycardia associated with 2nd degree AVB, that was complicated by a 30% left sided pneumothorax, which subsequently required a chest tube. Several chest x-rays were done that admission and the patient was sent home once there was radiographic evidence the pneumothorax resolved. Several days following her initial discharge, the patient began complaining of fatigue, shortness of breath and weakness but did not have any associated chest pain. Her shortness of breath progressed to the point where she was no longer able to lie flat, which subsequently brought her to the emergency room for further evaluation.

In the emergency room, the patient was tachycardic and had an oxygen saturation of 90% on room air. Labs revealed a troponin of 0.13 and a BNP of 2433. The other labs were all WNL. The patient had no prior history of cardiac or pulmonary disease and according to her she had a stress test in the past that was negative for ischemia. An EKG was performed (Figure 1). A 2D echocardiogram was performed prior to pacer insertion that showed a preserved LV function with LVH. An echocardiogram performed on re-admission revealed severe LV dysfunction with an estimated EF of 20-25%. The best contracting segment was that of the base of the septum and the lateral all. There was also evidence of moderate MR and moderate to severe TR with pulmonary hypertension.



Figure 1. EKG post pacemaker

The patient was transferred to tertiary facility for further management. She underwent a cardiac catheterization, which demonstrated patent coronary arteries (Figure 2). Ventriculogram revealed anterolateral akinesis, apical akinesis, diaphragmatic akinesis, apical septal akinesis, inferolateral akinesis and posterolateral akinesis (Figure 3). 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. She was subsequently started on a systolic heart failure regimen, which included lisinopril 5mg daily, metoprolol 50 mg BID, simvastatin 20 mg daily and aspirin 81 mg. Over the course of her hospital stay, the patient's symptoms of dyspnea improved. The patient was discharged home in stable condition.

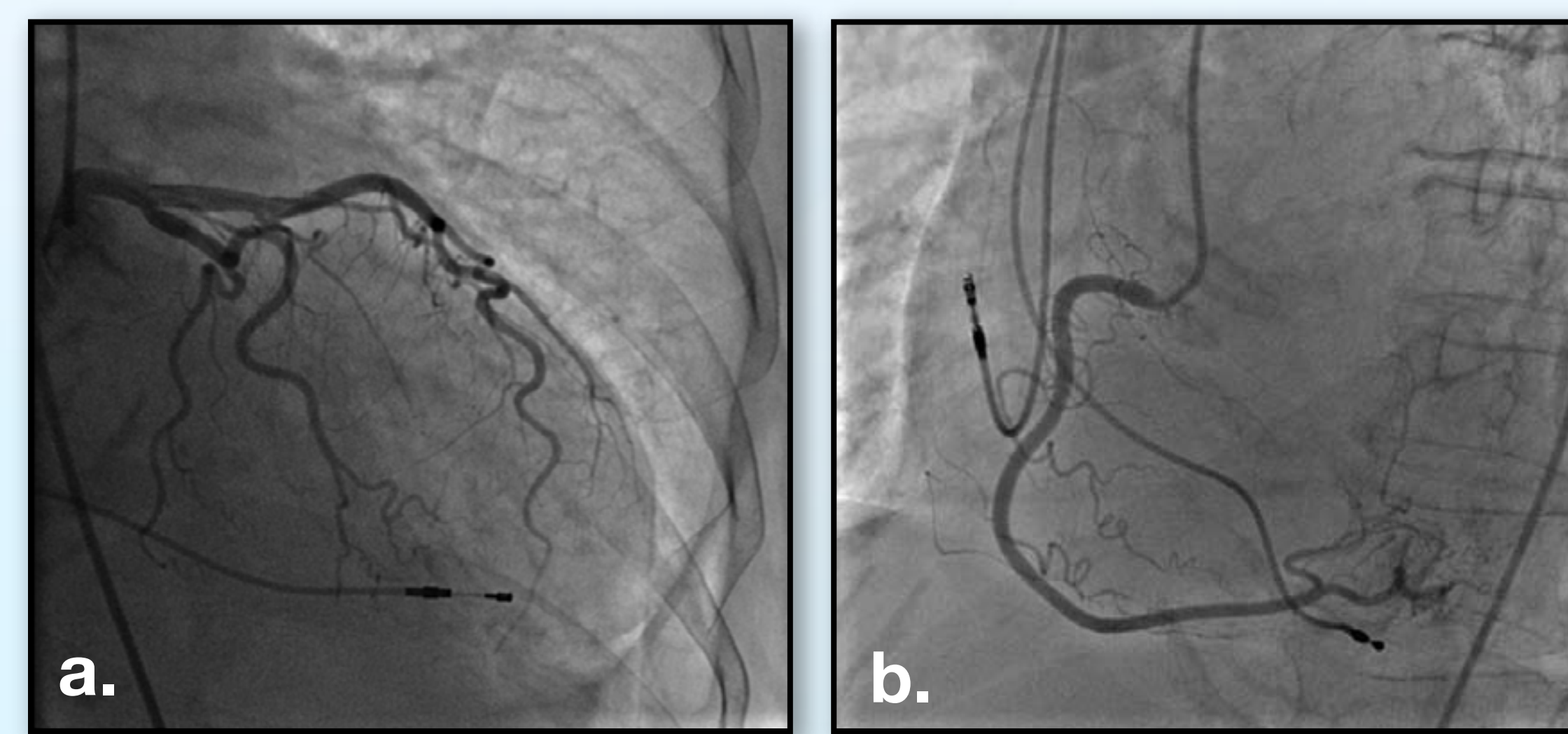


Figure 2. Coronary Angiography showing a) left coronary artery arterial system with patent LAD, Circumflex, and b) right coronary artery.

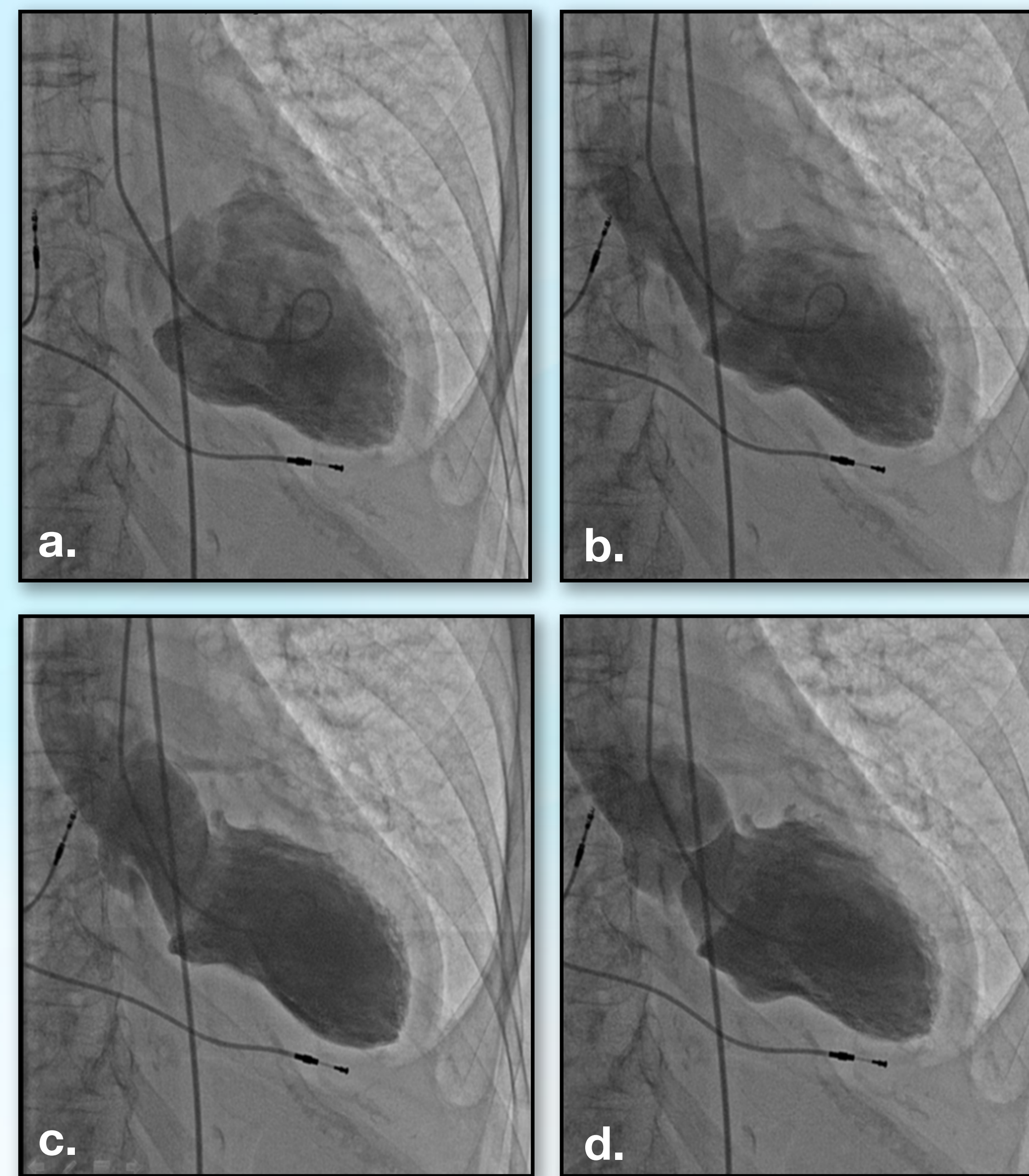


Figure 3. Left Ventriculogram demonstrating apical akinesis, apical ballooning characteristic of TCM. a) End Diastole, b) Early Systole, c) Late Systole, d) Early diastole.

Discussion

In 2004, the Mayo clinic identified diagnostic criteria for takotsubo cardiomyopathy, which include: a transient hypokinesis/dyskinesis 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.⁴ Criteria also include elevated troponin, and the absence of pheochromocytoma and myocarditis.¹ Our patient did demonstrate appreciable akinesis 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.^{3,4} In current theory, takotsubo cardiomyopathy may be precipitated by catecholamine cardiotoxicity, microvascular dysfunction, or coronary artery vasospasm.² 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.

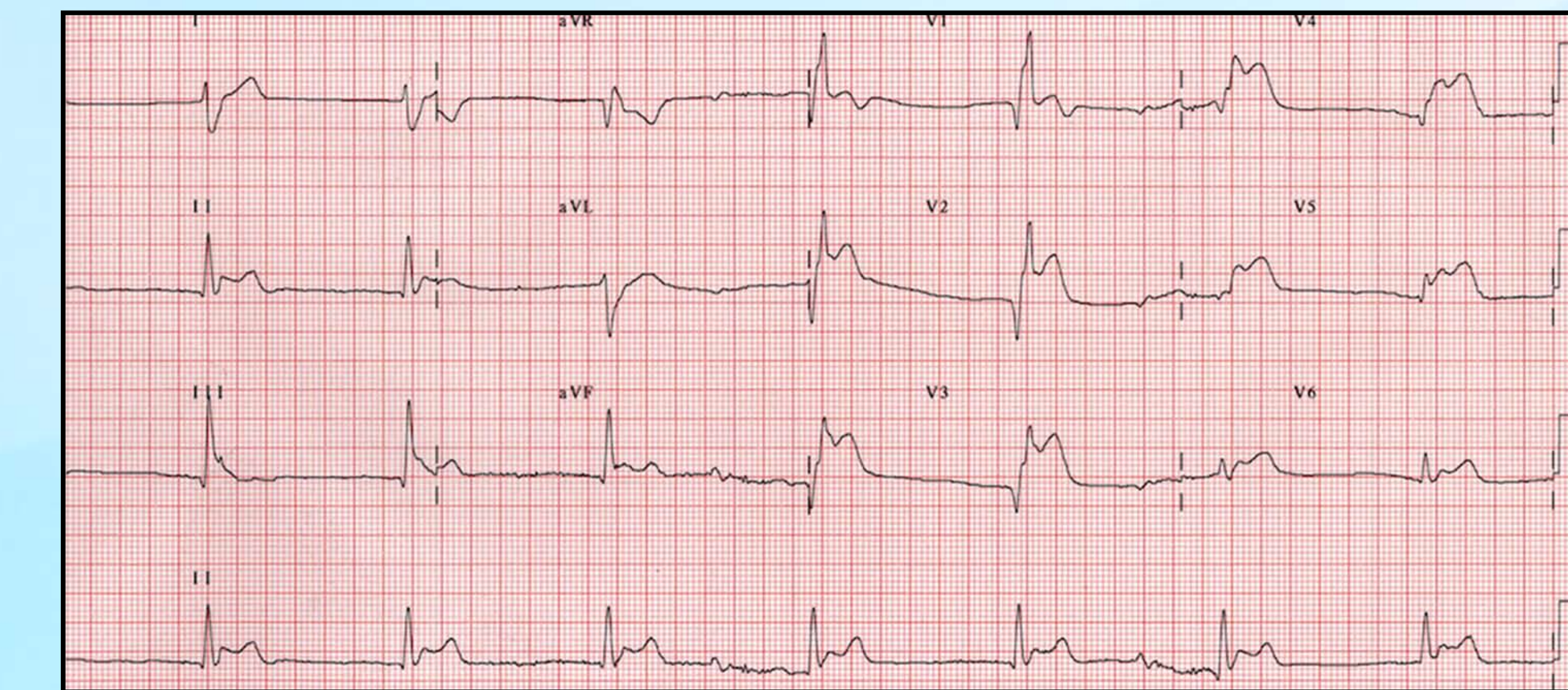


Image obtained from <http://lifeinthefastlane.com/ecg-library/tako-tsubo/>

In a recent review conducted by Postema 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.³ In this aspect, this patient's onset of symptoms was significantly delayed in comparison to the seven cases analyzed.³

Notably, the implantation of a Dual chamber pacemaker in this patient was complicated by a 30% left pneumothorax, which may have contributed to delayed recognition 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 spill as well as angiography demonstrating hypokinesis of the left ventricle in the absence of significant CAD, which were consistent with Mayo Clinic criteria.¹ Other evaluation of the patient showed an ejection fraction of 20-25% on echocardiogram, and of 25% by angiography. Echocardiography was useful in guidance of further clinical decision making for this patient, having demonstrated a severe reduction in Left Ventricular systolic function status post pacemaker implantation.

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