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Internal Medicine

A Case of Focal Atrial Tachycardia Not Detected by the Implanted Device

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Abstract **Case Report**

Implantable cardioverter-defibrillators (ICD) can be placed to reduce the risk of sudden cardiac death. Dual chamber ICDs have the additional benefit of identifying atrial arrhythmias. However, depending on the arrhythmia as well as the programmed settings for detection, the device may not detect rhythm abnormalities. Relying on a device check report alone to rule out arrhythmias may result in a missed diagnosis. In our case, an 81-year-old male who had a dual chamber ICD presented to the hospital due to worsening congestive heart failure with shortness of breath, weight gain, and lower extremity edema. His electrocardiogram readings were concerning for sinus tachycardia versus atypical atrial flutter versus atrial tachycardia. His ICD interrogation did not record the sustained atrial tachycardia as an abnormal rhythm since the rate was below the tachycardia detection threshold. His evaluation by an electrophysiologist determined that he had focal atrial tachycardia. The electrophysiologist pace-terminated the atrial tachycardia through the ICD. He presented 2.5 months later due to recurrence of atrial tachycardia and underwent successful ablation.

Keywords: Focal atrial tachycardia, Implantable cardioverter-defibrillator, electrocardiogram.

INTRODUCTION

In the United States, there are more than 800,000 fatalities associated with heart disease, and more than half are due to sudden cardiac death [1]. Implantable cardioverter-defibrillators (ICD) are placed to prevent sudden cardiac death [2]. In addition to delivering shocks in the setting of ventricular fibrillation or ventricular tachycardia, dual chamber ICDs can help with distinguishing among arrhythmias, assess atrial arrhythmias, and provide "antitachycardia pacing" when arrhythmias arise [3]. Some dual chamber ICDs can provide shocks in the setting of atrial tachyarrhythmias [3]. Devices can record events based on criteria set by the doctor or manufacturer [4]. Identification of arrhythmias by ICDs can depend on factors such as the programmed detection rate, sensitivity of the leads, refractory time frame of the heart tissue, and configuration of the leads [5]. Our case illustrates the importance of recognizing that atrial tachycardia may not be detected by a dual chamber ICD if the rate of the atrial tachycardia is lower than the pre-programmed atrial arrhythmia detection rate.

CASE PRESENTATION

An 81-year-old male with history of coronary artery disease treated with coronary artery bypass grafting 24 years prior, aortic stenosis treated with transcatheter aortic valve replacement 2 years prior, heart failure with reduced ejection fraction with left ventricular ejection fraction (LVEF) of 35-40% three months prior, ischemic cardiomyopathy with dual-chamber ICD in place, paroxysmal atrial fibrillation, peripheral vascular disease, orthostatic hypotension, hyperlipidemia, chronic anemia, chronic obstructive pulmonary disease, and obstructive sleep apnea presented to the hospital due to shortness of breath that had been worsening over the course of 24 hours. He also noted increasing bilateral lower extremity and an 8-pound increase in weight over the past week. He denied any chest pain associated with these symptoms.

He had recently been admitted to the hospital 3 weeks prior to presentation where he was treated for reported atrial fibrillation with rapid ventricular response and acute exacerbation of congestive heart failure with reduced ejection fraction. His B-type natriuretic peptide (B-NP) level was 794.1 pg/mL (reference range: < 100 pg/mL). At this time, he underwent direct current

cardioversion to an atrial-paced rhythm. His amiodarone was increased from 200 mg once daily to twice daily for one month before returning to once daily.

He reported compliance with his outpatient medications, including amiodarone dosed at 200 mg twice daily, atorvastatin dosed at 20 mg once daily, bumetanide dosed at 2 mg in the morning and 1mg in the evening, clopidogrel dosed at 75 mg once daily, ezetimibe dosed at 10 mg once daily, midodrine dosed at 5 mg up to three times daily as needed, ranolazine dosed at 500 mg twice daily, and apixaban dosed at 2.5 mg twice daily.

On presentation, his blood pressure was 130/81 mmHg, heart rate was 118 beats per minute, and oxygen saturation was 98% on room air. Physical examination was notable for diminished breath sounds and bilateral 1+ lower extremity edema. His electrocardiogram (EKG) was concerning for sinus tachycardia versus atrial flutter versus atrial tachycardia (Figure 1). However, his ICD interrogation report in the emergency room did not reveal any device-detected atrial arrhythmia. His atrial tachycardia/atrial fibrillation detection rate had been set at 171 beats per minute. A chest radiograph was notable for a small right-sided pleural effusion. His B-NP was 918.2 pg/mL. He was started on intravenous bumetanide dosed at 2 mg twice daily. An echocardiogram revealed a left ventricular ejection fraction of 20-25%, which was a decrease in LVEF as compared to three months prior. His diuresis was changed to IV bumetanide dosed at 2 mg in the morning and 1 mg in the evening.

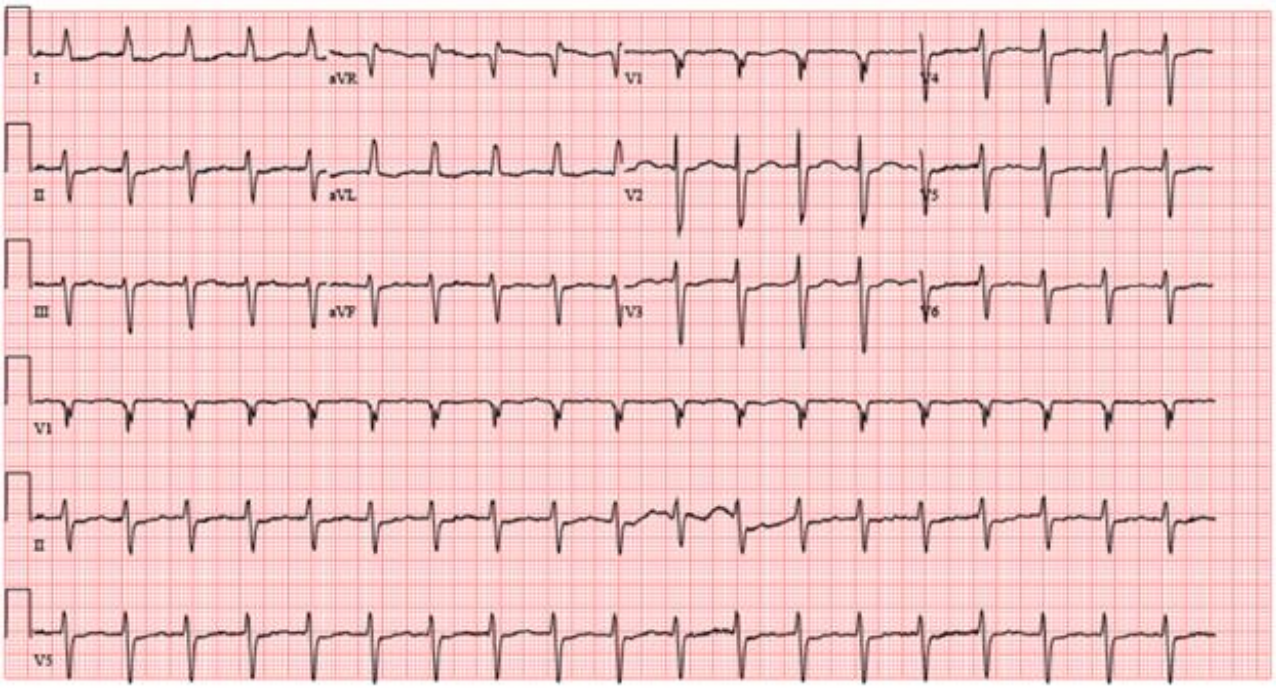

Figure 1: First Electrocardiogram

The following day, his heart rates remained elevated, around 100 – 115 beats per minute. Another EKG revealed similar findings (Figure 2). Once again, his ICD interrogation report did not reveal any atrial arrhythmia. The electrophysiology team was consulted to evaluate the patient. A formal diagnosis of focal atrial tachycardia was made. His ICD interrogation confirmed that the device was not recording the atrial tachycardia since it was outside of its rate detection parameters. The focal atrial tachycardia was subsequently pace-terminated through the ICD. His atrial tachycardia was slow enough such that ICD diagnostics would not be able to detect any events if they recurred. Thus, his ICD would be unreliable in determining if the atrial tachycardia appeared again in the future. Up until the pace-termination, his heart rate was between 100 and 120 beats per minute. Afterwards, his heart rate remained in the 60s. Given the patient's left ventricular dysfunction, renal insufficiency, and hypotension requiring midodrine, there were limited options for medication management. His metoprolol succinate was increased to 12.5 mg twice daily. Additionally, ablation was recommended if the focal atrial tachycardia returned. His EKG, the next day, revealed atrial-paced rhythm with prolonged atrioventricular conduction (Figure 3). He was subsequently discharged with instructions to follow-up with cardiology as an outpatient. He was also advised to return to the emergency department if his resting heart rate exceeded 100 beats per minute.

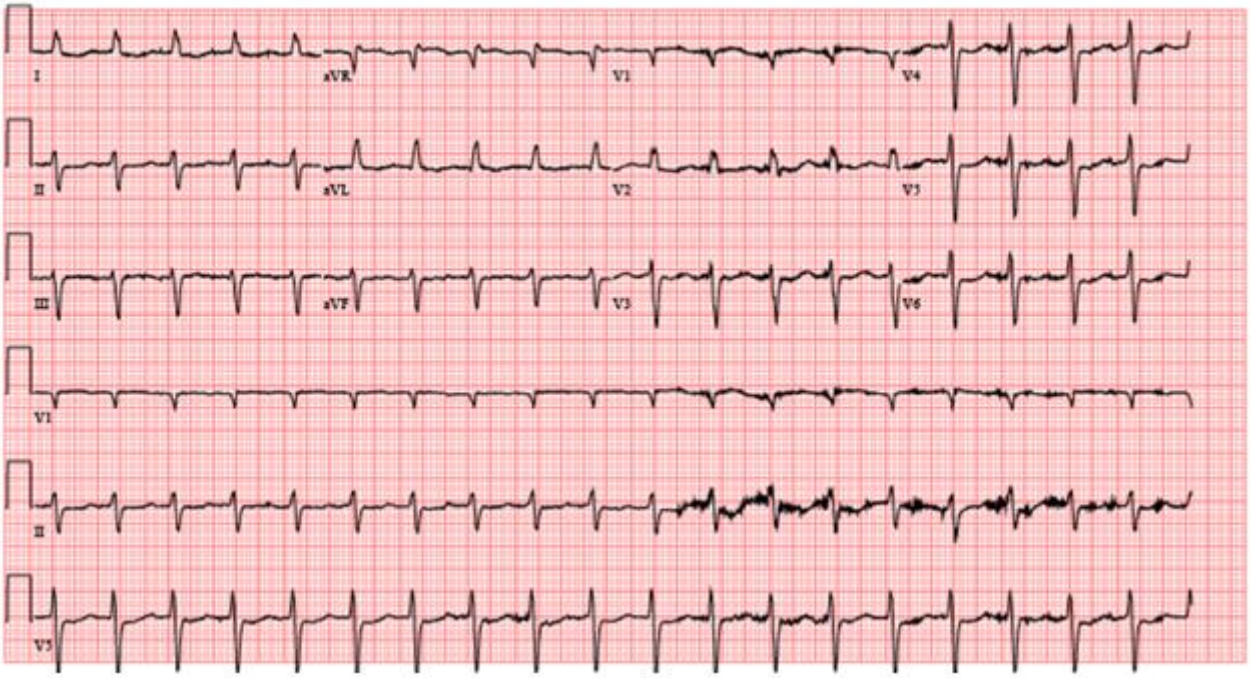

Figure 2: Second Electrocardiogram

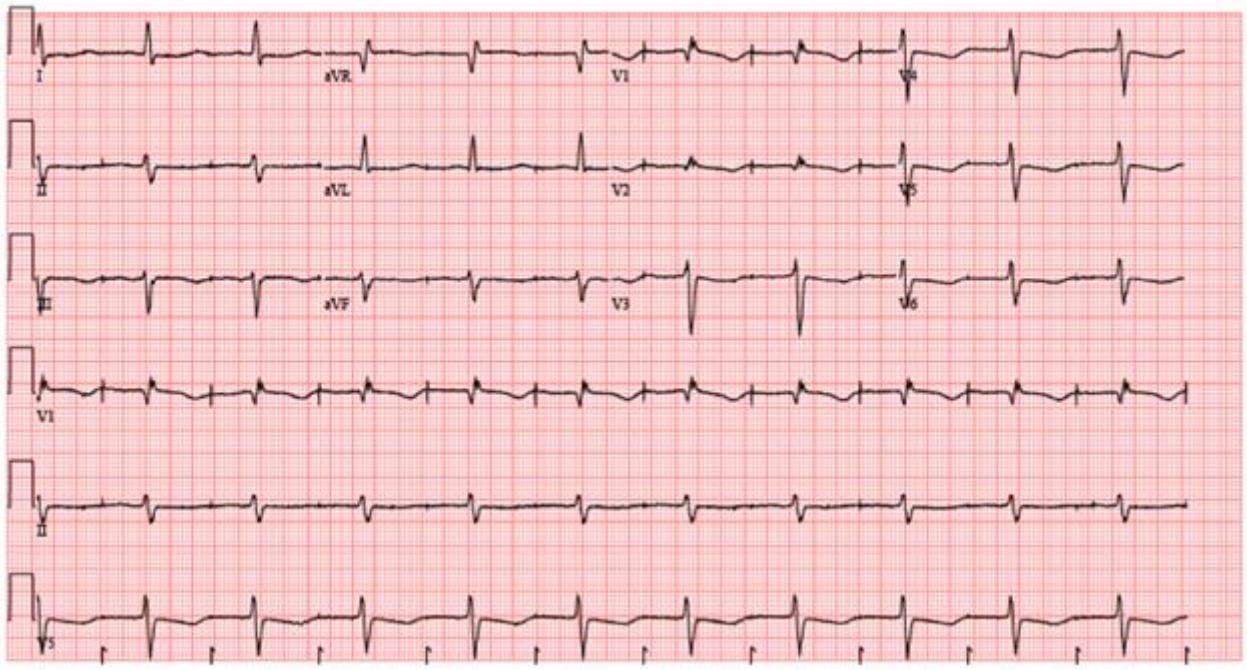

Figure 3: Third Electrocardiogram

Two and a half months later, the patient returned to the emergency department due to recurrent atrial tachycardia. His B-NP was 844.1 pg/ml. He underwent successful atrial overdrive pacing of the atrial tachycardia with restoration of sinus rhythm. Two days later, he underwent an electrophysiologic study and was found to have two focal right atrial tachycardias. One was arising from the inferolateral right atrium in the lateral cavotricuspid isthmus area. The second was arising from the inferior region of the right atrium at the lip of the inferior vena cava approximately halfway between the septal and lateral walls. The cycle lengths were 500 ms and 530 ms, respectively. Both were successfully ablated. Radiofrequency energy application at the first site resulted in sinus rhythm. Atrial burst pacing on isoproterenol resulted in an easily inducible focal atrial tachycardia at the second site and radiofrequency application at this site resulted in sinus rhythm. Repeat atrial burst pacing on isoproterenol did not induce any tachycardia and the procedure was terminated.

DISCUSSION

Excluding atrial fibrillation and atrial flutter, paroxysmal supraventricular tachycardia (PSVT) refers to anomalies of automaticity and reentry [6]. In this context, PSVT is estimated to have a prevalence of 168/100,000 and incidence of 73/100,000 in the United States [6]. Risk factors of PSVT include underlying cardiac disease, elevated age, and female gender [6].

An uncommon type of PSVT, atrial tachycardia (AT), accounts for approximately 10% of cases of PSVT [7]. In AT, the atrial rate is generally between 130 and 250 beats per minute; however, the rate can decrease to 100 beats per minute or increase to 300 beats per minute [8]. Symptoms of AT include chest pain, shortness of breath, palpitations, and fatigue [8].

Distinguishing AT from sinus tachycardia, atrioventricular nodal reentrant tachycardia (AVNRT), and atrioventricular reentrant tachycardia (AVRT) on electrocardiogram may be challenging [8]. There also may be challenges differentiating AT from atrial flutter on electrocardiogram [9]. The P waves of AT and sinus tachycardia generally appear different, although AT arising from the crista terminalis may yield a similar appearing P wave [8]. A tachycardia that starts and ends suddenly is more likely to be AT; on the other hand, a tachycardia that rises and lowers over the course of half a minute to several minutes is more likely to be sinus [8]. To distinguish between AT and AVNRT or AVRT, it is important to look at the P wave and R-P interval [8]. In both typical AVNRT and AVRT, the P wave morphology cannot generally be easily identified, and the R-P interval is short and usually constant [8]. Though a short R-P interval can be seen in AT, AT is more commonly associated with a long R-P interval and a variable R-P interval may be seen [8]. There are a few features which can help differentiate between AT and atrial flutter [9]. AT generally has variable P waves, and P waves that are aligned closely with the QRS [9]. In atrial flutter, flutter waves may occur at a rate that is not consistent with the QRS [9].

Detecting tachyarrhythmias early on is important [10]. Addressing a tachyarrhythmia can help improve heart failure attributed to the tachyarrhythmia [10]. Treatment of cardiomyopathies associated with arrhythmias can improve medical outcomes, quality of life, while also reducing costs of medical care and need for inpatient hospitalizations [11]. Additionally, correctly identifying of arrhythmias as AT rather than atrial fibrillation permits antitachycardia pacing that is considered painless as opposed to cardioversion that can be painful [12].

Dual-chamber devices give the clinician valuable information about atrial arrhythmias, but it is essential to look at the device settings to ensure accuracy [13]. Episodes of atrial tachycardia may not be detected if the cycle length of the AT is longer than the programmed detection interval [12]. Our case serves as an important reminder that ICDs do not always detect or record the AT, especially if rates are lower than the programmed detection parameters. Arrhythmias can be a contributing factor in the exacerbation of symptoms even at lower rates. We were able to recognize that the patient was having AT, which led to the subsequent pace-termination and ablation. This case serves as an important reminder that device checks alone should not be relied upon for detection of arrhythmias. It is important for the clinician to identify and manage such scenarios.

CONCLUSION

AT is an uncommon type of PSVT that can be identified by an ICD. It is often difficult to distinguish AT from other abnormal rhythms on EKG but there are some notable differences which can help provide clarity. In some cases, such as ours, AT may not be detected by the ICD if rates are below the tachycardia threshold. EKG analysis with close scrutiny may help detect abnormal rhythms. It is necessary for the clinician to identify the AT and address it appropriately. This may involve pace-termination of the AT for immediate treatment and ablation for permanent treatment.

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