Open Journal of
Clinical & Medical
Case Reports

Volume 4 (2018)
Issue 16

ISSN 2379-1039

An unlikely cause of decompensated heart failure: Repetitive non-reentrant ventriculoatrial synchrony

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Abstract

A 65-year-old male with non-ischemic cardiomyopathy and a dual-chamber Implantable Cardioverter-Defibrillator (ICD) was admitted to the Coronary Care Unit (CCU) for an acute decompensated heart failure exacerbation. On initial presentation he was hypotensive and in moderate respiratory distress.

In the CCU, he had repetitive episodes of hypotension during dual chamber pacing. Device interrogation revealed a rare form of pacemaker-mediated tachycardia, Repetitive Non-Reentrant Ventriculoatrial Synchrony (RNRVAS). Although initially identified incidentally during device interrogations, this case demonstrates the potential for RNRVAS to cause hemodynamic compromise and decompensated heart failure.

Keywords

pacemaker mediated tachycardia; arrhythmia; repetitive non-reentrant ventriculoatrial synchrony; implantable cardioverter defibrillator

Abbreviations

ICD: Implantable cardioverter-defibrillator; CCU: Coronary care unit; RNRVAS: Repetitive non-reentrant ventriculoatrial synchrony; ADHF: Acute decompensated heart failure; NYHA: New york heart association; ECG: Electrocardiogram; VPDs: Ventricular premature depolarizations; PVARP: Post-ventricular atrial refractory period; PMT: Pacemaker-mediated tachycardia; BPM: Beats per minute; V-A: Ventriculo-atrial; A-V: Atrio-ventricular; NCAP: Non-competitive atrial pacing; MS: milliseconds; V-V: ventriculo-ventricular

Case Presentation

A 65-year-old male with a long-standing history of non-ischemic cardiomyopathy, an ejection fraction of 25%, and a primary prevention dual-chamber Implantable Cardioverter-Defibrillator (ICD) (Boston Scientific, Marlborough, MA, USA) was admitted to the Coronary Care Unit (CCU) for Acute Decompensated Heart Failure (ADHF). Initial symptoms included a two-week history of palpitations, lower extremity edema, and worsening dyspnea on exertion. During this time frame his weight increased by approximately 10 pounds and his New York Heart Association (NYHA) functional class progressed from II to III. Past medical history was significant for hypertension and obesity. Prior to presentation he

was compliant with all of his heart failure medications, which included Lisinopril, Carvedilol, and Spironolactone. He denied any recent illnesses or dietary indiscretions, and there were no other potential exacerbating factors identified.

In the emergency department his initial Electrocardiogram (ECG) demonstrated an atrial-paced, ventricular-sensed rhythm (**Figure 1**). He was hemodynamically stable but in moderate respiratory distress, and was therefore admitted for treatment of ADHF. He was treated with intravenous nitroglycerin, diuretics, and non-invasive ventilation.

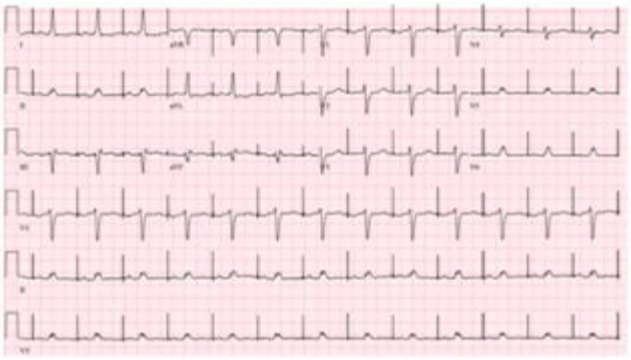

Figure 1. Initial ECG on arrival to CCU.

During his admission, his heart rhythm varied between sinus rhythm with intermittent Ventricular Premature Depolarizations (VPDs), a trial paced-ventricular sensed rhythm, and dual-chamber pacing. Initial device evaluation revealed a dual-chamber ICD with leads in the right atrial appendage and the right ventricular apex. Device interrogation failed to identify any high rate events or therapies delivered, and all pacing parameters were normal (**Table 1**).

Table 1: Baseline Programmed Device Parameters

Programmed Parameters	Setting
Mode	DDDR
Lower rate limit	60 beats/min
Maximum tracking rate	120 beats/min
Paced A-V delay	300 – 400 ms
Sensed A-V delay	300 – 400 ms
PVARP	200 – 300 ms
Ventricular refractory period	240 – 250 ms
Ventricular blanking after atrial pacing	65 ms

Telemetry and continuous arterial blood pressure monitoring revealed that during dual-chamber pacing the patient consistently became hypotensive and appeared to lose atrial capture. An ECG was able to partially capture an interval of dual-chamber pacing **(Figure 2)**. Evaluation of this ECG shows that although the first four beats are apparently dual-chamber paced, there was atrial non-capture. Recognizing this abnormality prompted repeat interrogation of the device during one of these episodes. During the second device interrogation, there was evidence of ventricular-paced events that were followed by retrograde atrial sensed activity which fell in the Post-Ventricular Atrial Refractory Period (PVARP). Despite these sensed atrial events, the device attempts to pace the atria but fails to capture. This sequence then continues to repeat itself creating a rare form of Pacemaker-Mediated Tachycardia (PMT) known as Repetitive Non-Reentrant Ventriculoatrial Synchrony (RNRVAS) **(Figure 3)**.

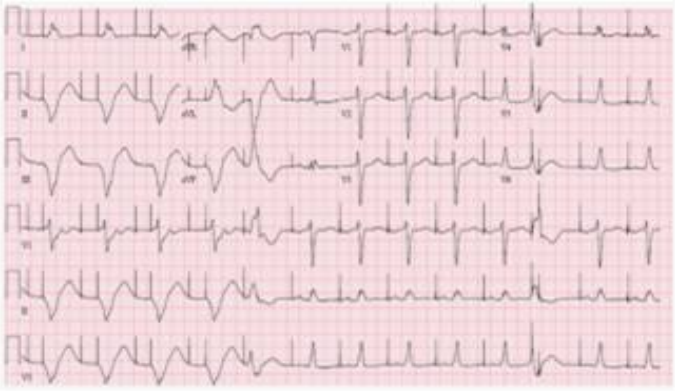

Figure 2: ECG during episode demonstrating dual-chamber pacing in the first four beats with atrial non-capture. The sequence is interrupted by a VPD, and then atrial pacing with ventricular sensing resumes.

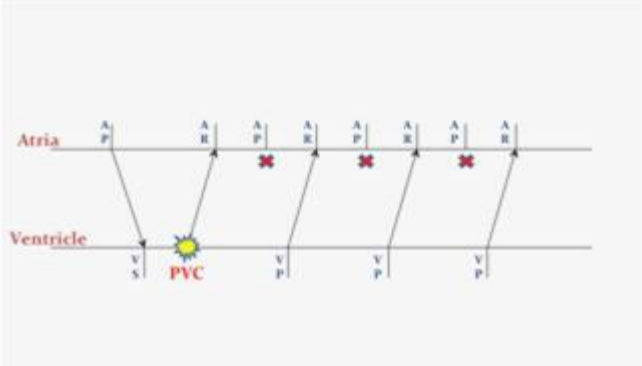

Figure 3: **A – B.** Telemetry rhythm strip (panel A) and device electrograms (EGMs, panel B) from initial device interrogation in sinus rhythm with normal pacemaker function. **C – D.** Telemetry rhythm strip (panel C) and device EGMs (panel D) from second device interrogation demonstrating repetitive non-reentrant ventriculoatrial synchrony (RNRVAS). Ventricular pacing (VP) is followed by an atrial sensed (AS) event, and subsequently an atrial paced (AP) event that fails to capture.

A = right atrial lead electrogram, V = right ventricular lead electrogram, L = leadless (far-field) electrogram, AS = atrial sensed, VS = ventricular sensed, AP = atrial paced, VP = ventricular paced.

Our patient's device was reprogrammed to a lower rate limit of 50 beats per minute (bpm). Throughout his hospital course he continued to experience VPDs with retrograde ventriculo-atrial (V-A) conduction, however developed no further episodes of RNRVAS. During his routine device interrogation follow-up 6-months later, he reported no further episodes of palpitations and had no further heart failure hospitalizations.

Discussion

In the initial evaluation of patients presenting with ADHF it is essential to search for potential triggers, as evidence suggests that over 60% of patients will have an identifiable precipitant [1]. In one cohort of patients, acute respiratory infections, myocardial ischemia, and cardiac arrhythmias were the most frequently identified triggers, with dietary and medication non-compliance, uncontrolled hypertension, and worsening renal function occurring less commonly. As discussed in the case presentation, our patient reported compliance with his medication regimen and dietary restrictions, and had no recent acute illnesses. Initial evaluation also ruled out worsening renal function and active ischemia. Therefore, the only identifiable trigger for his presentation was the presence of intermittent RNRVAS.

PMT is a well-known clinical syndrome that has traditionally been used to describe an endless-loop reentrant tachycardia, occurring in patients with dual-chamber pacemakers and retrograde V-A conduction [2]. A far less common and under-recognized form of PMT, RNRVAS, can have equally important clinical consequences, yet may be much more difficult to diagnose.

Similar to the endless-loop PMT, RNRVAS is a non-reentrant PMT that is typically initiated with a VPD causing retrograde V-A conduction. However, due to PVARP, which is set to prevent endless-loop PMT, retrograde atrial depolarization is not sensed due to functional under-sensing. This then leads to attempted atrial pacing, which fails to capture the myocardium due to atrial tissue refractoriness, causing functional loss of capture. Finally, after the set atrio-ventricular (A-V) delay, ventricular pacing is initiated, and once again due to retrograde V-A conduction the cycle becomes repetitive (**Figure 4**). This can lead to unnecessary ventricular pacing and loss of A-V synchrony. Interestingly, RNRVAS has arisen as a consequence of the programming strategies used to minimize ventricular pacing and to prevent the more traditional endless-loop PMT.

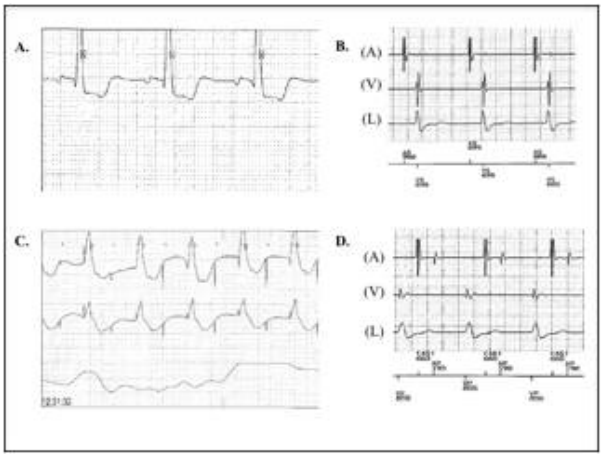

Figure 4: Diagrammatic representation of repetitive non-reentrant ventriculoatrial synchrony (RNRVAS). A Ventricular Premature Depolarization (VPD) creates retrograde Atrial conduction that falls in the Refractory period (AR), followed by Atrial Pacing (AP) with functional non-capture (red x). After the programmed A-V delay, ventricular pacing (VP) resumes with subsequent retrograde atrial conduction. RNRVAS is initiated and the cycle repeats.

Although early case reports have identified this malfunction in asymptomatic patients, more recently RNRVAS has been found to be clinically relevant, particularly, as in the case of our patient, in individuals with clinical heart failure, left ventricular dysfunction, and decreased left ventricular compliance [3].

Due to the intermittency with which RNRVAS may occur and the relatively low heart rates, most pacemakers fail to recognize it as a malfunction, and consequently fail to log the events [4]. St. Jude devices (Abbott, Abbott Park, IL, U.S.A.) will log the events as atrial high-rate events. Consequently, standard device interrogation cannot be relied upon for diagnosis. Instead, clinicians must have a high clinical suspicion in patients whose symptoms do not correlate with interrogation reports and must carefully review inpatient cardiac monitoring or consider obtaining outpatient Holter monitoring for symptomatic patients.

There are several device-programming strategies that can be employed in order to prevent further episodes of RNRVAS once it has been recognized. Reducing the lower rate limit of the device and shortening the A-V delay will prevent the device from attempting to pace during the atrial refractory period. Decreasing the PVARP will allow the device to recognize the retrograde atrial activity and again prevent atrial pacing during the atrial refractory period. However, this will in turn make the patient more susceptible to endless-loop PMT [4].

The most sophisticated strategy for preventing RNRVAS comes in the form of newer device technology such as Medtronic's (Minneapolis, MN, U.S.A.) non-competitive atrial pacing (NCAP). NCAP is a device algorithm that is triggered whenever a sensed p-wave falls within the PVARP. It functions to prevent competitive atrial pacing during the vulnerable atrial refractory period, which is accomplished by inhibiting atrial pacing for 300 milliseconds (ms) once NCAP has been triggered [5]. After NCAP has been initiated the ventriculo-ventricular (V-V) interval will be maintained through shortening the paced A-V delay. By delaying any scheduled atrial pacing events by 300 ms, the algorithm reduces the chance of atrial refractoriness at the time of atrial pacing, and theoretically eliminates the possibility of RNRVAS. However, if the atrial tissue remains refractory following the 300 ms delay, the atrial paced beat that follows will fail to capture and RNRVAS may still occur [5]. There has also been a report of NCAP leading to ventricular tachycardia due to a ventricular-sensed event taking place during atrial blanking, following far-field over-sensing in the atrial lead [6].

Conclusion

Although it is far less well-known than the traditional endless-loop PMT, RNRVAS is a clinically important syndrome that may exacerbate congestive heart failure. Programming strategies to prevent endless-loop PMT or ventricular pacing (such as long A-V delays) may actually promote RNRVAS. Clinicians must understand the limitations to standard device interrogation, and consider other forms of continuous cardiac monitoring, such as Holter monitors, in patients who present with symptoms of palpitations, shortness of breath, or heart failure despite the absence of device-logged events.

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Manuscript Information: Received: April 02, 2018; Accepted: August 28, 2018; Published: August 31, 2018

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Citation: Marbach JA, Sadek MM. An unlikely cause of decompensated heart failure: Repetitive non-reentrant ventriculoatrial synchrony.. Open J Clin Med Case Rep. 2018; 1454.

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