Atrial lead malfunction presenting as new onset pacemaker-mediated tachycardia

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This report describes the de novo occurrence of pacemaker-mediated tachycardia (PMT) in a patient with a dual-chamber implantable cardioverter-defibrillator and stable retrograde ventriculo-atrial conduction time. The same rate-adaptive post-ventricular atrial refractory period (PVARP) duration had previously prevented PMT. Oversensing of atrial false signals from a defective lead shortened the PVARP with consequent sensing of retrograde conduction.

Introduction

This report describes how a defective atrial lead in a patient with a DDDR implantable cardioverter defibrillator (ICD) resulted in a shortened rate-adaptive post-ventricular atrial refractory period (PVARP) which, in turn, precipitated de novo pacemaker-mediated tachycardia (PMT). Prior to the lead abnormality, the propensity to PMT had been controlled by the same PVARP duration.

Case report

A 65-year-old man received dual-chamber ICDs in 2001, 2005, and 2010, the last being a St Jude, Current Accel DR.1 In July 2011, he developed many asymptomatic PMT episodes (Figure 1). The atrial lead impedance was normal. The stored atrial electrogram (A) revealed oversensed false signals sometimes difficult to distinguish from atrial depolarizations. The patient had always exhibited DDDR. Low rate = 50 ppm, upper rate = 120 ppm, paced AV delay = 170 ms, sensed AV delay = 110 ms, rate adaptive PVARP = 275 ms (minimum 225 ms high setting), ventricular intrinsic preference = 200 ms; 3 intervals (terminating with VP as above: AS-VP = 110 + 200 = 310 ms). False signals detected in the atrial refractory period are shown by small vertical upward markers with no label. AS = atrial sensed event, AP = atrial paced event, VS = ventricular sensed event, VP = ventricular paced event, V = ventricular electrogram.

Figure 1 Pacemaker-mediated tachycardia. V, ventricular electrogram. See text for details.
constant retrograde conduction of 240 ms. The rate responsive PVARP had remained unchanged at 275 ms. No PMT had occurred until 2011. A false signal probably initiated the PMT (*) by permitting retrograde conduction. During the PMT, the longest PVARP measured 500 -- 262 or 500 -- 258 = 238 to 242 ms.

**Discussion**

The rate responsive PVARP in St Jude devices automatically changes the PVARP in response to increases or decreases in the Atrial Fibrillation Suppression™ Algorithm rate, sensor-indicated rate, or the filtered atrial rate interval (FARI) used for automatic mode switching (AMS).1,2 The algorithm begins to operate when the intrinsic rate exceeds 90 b.p.m. A high setting changes the PVARP settings faster than a low setting. Thus, as pacing rates rise, the PVARP decreases until the maximum sensor rate, maximum tracking rate, or the shortest PVARP setting is reached.

In our case the sensed atrial false signals created short atrial intervals which decreased the FARI and eventually caused inappropriate AMS. The shorter FARI decreased the PVARP so that the device became capable of sensing retrograde P waves resulting in PMT. De novo PMT (and AMS) episodes without programming changes should raise the suspicion of a defective atrial lead. Such PMT may be the first clue of a defective atrial lead because atrial false signals tend to occur irregularly and may not be apparent or suspected at the time of follow-up.

**Conflict of interest:** none declared.

**References**


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**Sudden reversible pacemaker failure in a patient with cardiac sarcoidosis: an unfortunate case of ventricular septal pacing**

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We report a case of sudden marked deterioration of ventricular stimulation threshold resulting in pacemaker failure 16 months after a ventricular septal lead implantation for atrioventricular block. Echocardiography revealed septal wall thinning at the electrode–tissue interface, which was not detected pre-operatively. Endomyocardial biopsy confirmed cardiac sarcoidosis. The increased threshold was reversible with prednisolone.

**Clinical case**

A 30-year-old man was hospitalized with syncope. He was found to have complete left bundle branch block at age 28. The physical examination was unremarkable. A 12-lead electrocardiogram showed complete atrioventricular block (AVB). An echocardiogram demonstrated hypokinesis of the septal wall (Figure 1E) and apex [left ventricular ejection fraction (LVEF), 0.58].2 Thallium myocardial scintigraphy showed severe hypoperfusion of the entire left ventricle except for the lateral wall (Figure 1C). A whole-body computed tomography scan and serum angiotensin-converting enzyme were normal. Endomyocardial biopsy showed non-specific interstitial fibrosis and inflammatory cell infiltration. After a diagnosis of AVB due to unexplained myocarditis, he received a dual-chamber pacemaker with the ventricular lead placed in the septum (Figure 1B).

He had been well until 16 months after discharge, when he fainted again and was rehospitalized. A 12-lead electrocardiogram showed failure of ventricular sensing and pacing. Pacemaker interrogation revealed a marked rise in the ventricular capture threshold to 6.5 V (Figure 1A). The ventricular lead impedance and lead position were unchanged. An echocardiogram demonstrated dyskinesis and thinning of the septal wall near the electrode–tissue interface (Figure 1F) and deterioration of left ventricular function (LVEF, 0.37).2 Thallium myocardial scintigraphy revealed the progression of hypoperfusion in the anteroseptal wall (Figure 1D). A whole-body gallium scan was unremarkable. The second endomyocardial biopsy showed epithelioid cells and a few Langhans giant cells in granulation tissue. Thus, a diagnosis of cardiac sarcoidosis (CS) was made and treatment with oral prednisolone 30 mg/day was initiated. The stimulation threshold fluctuated widely between 0.7 and 6.5 V in the initial 6 days after the commencement of prednisolone but eventually became stable at <1.0 V (Figure 1A). Placement of the second ventricular lead or up-grading to implantable cardioverter-defibrillator was recommended but was refused by the patient. He is currently doing well 5 years after steroid initiation, with no evidence of CS progression.

**Conflict of interest:** none declared.

**References**