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A case report of lead dysfunction presenting as high ventricular premature complex burden

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ABSTRACT

Pacemaker-troubleshooting is an important step in the evaluation of a patient with syncope post-pacemaker-implantation. The basic functions of sensing, pacing and impedance may remain spuriously normal in the case of lead-microfracture or insulation break. We report a case in which the lead dysfunction was diagnosed based on multiple episodes of premature ventricular beats.

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1. Clinical problem

A 79-yr-old-female, a case of complete-heart-block, underwent a dual-chamber pacemaker (Boston Scientific, Marlborough, Massachusetts, USA) in 2012. She was asymptomatic since implantation. She was on routine follow-up with regular interrogations which revealed normal pacing, sensing, and impedance on interval plot. There were a few atrial high rate episodes with appropriate mode switches. The patient started having multiple episodes of syncope (5–6 episodes in the last 9 months) after 7 years of implantation, for which she came for the consultation to the outpatient department (OPD). The clinical evaluation did not reveal any cause for recurrent syncopal events, including normal postural blood pressure and heart rate values. The ECG showed regular-paced rhythm (intermittent atrial-sensed ventricular-paced & atrial-paced ventricular-paced, with no evidence of loss of capture). The interrogation at this point showed a battery life of more than a year and normal sensing & impedance, with a marginal increase in pacing threshold in the ventricular lead (A-0.6V@0.4ms, V-1.7V@0.4ms). The impedance graph showed a marginal decrease in impedance from 500Ω to 450Ω over the last 3 months (Fig. 1).

The interrogation revealed multiple premature ventricular complexes (PVCs) (215800 episodes over the last 82 days). It also revealed ventricular tachycardia episodes (6000 episodes in total over the last 9 months), with the fastest episode at 245 bpm for 5 beats (Fig. 1). The electrogram (EGM) is as shown in Fig. 2. Given the stable lead impedance, what do you think can be the cause of the syncope: lead dysfunction or hemodynamically unstable VT episodes?

1.1. Reply to the clinical problem

A closer look at the electrograms (EGM) during each of these events revealed multiple high amplitudes, high-frequency EGMs on the ventricular channel (RV_{tip}→Ring). The marker channel annotations show all these EGMs as ventricular events (Fig. 2).

The ECG and Holter readings did not reveal any VPCs or VT episodes. There was no particular pattern to these EGMs and no corresponding deflections were seen on the atrial channel. There was no continuous high-frequency noise seen on the ventricular sensing electrode to suggest a lead fracture. Lots of the ventricular sensed events were seen, which were annotated as VT episodes. The VV intervals were not regular and occurring frequently with different coupling intervals, varying inter-VV intervals, and varying amplitudes (Fig. 2). This was considered as lead noise and a diagnosis of probable lead dysfunction (LD) was made. The ventricular timing cycles were also reset by these over-sensed events. Few of

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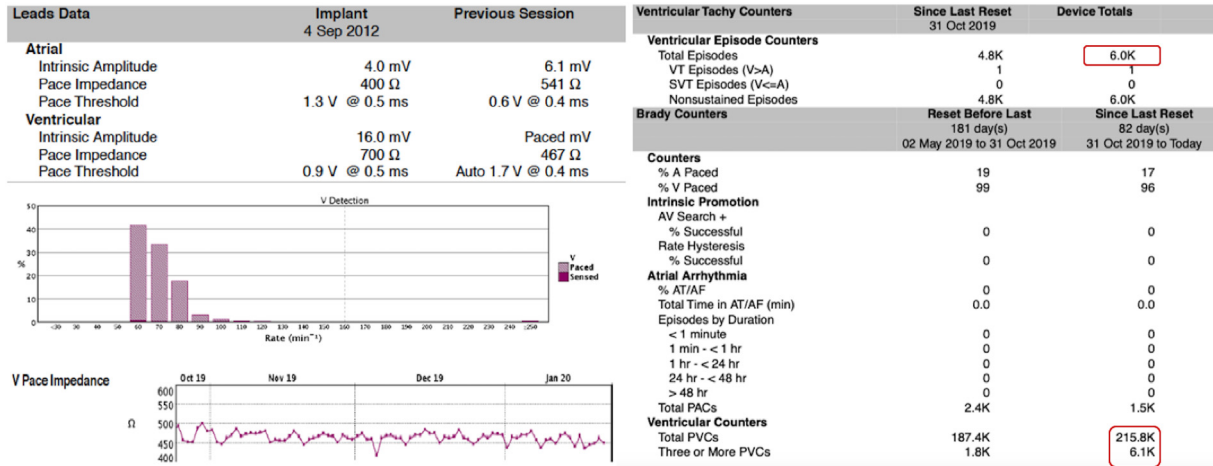


Fig. 1. Interrogation summary.

Event V-5950: 21 Jan 2020 21:56

NonSustV Event Onset

Avg A Rate: 30 min⁻¹

Avg V Rate: 131 min⁻¹

Event Ended 00:00:55

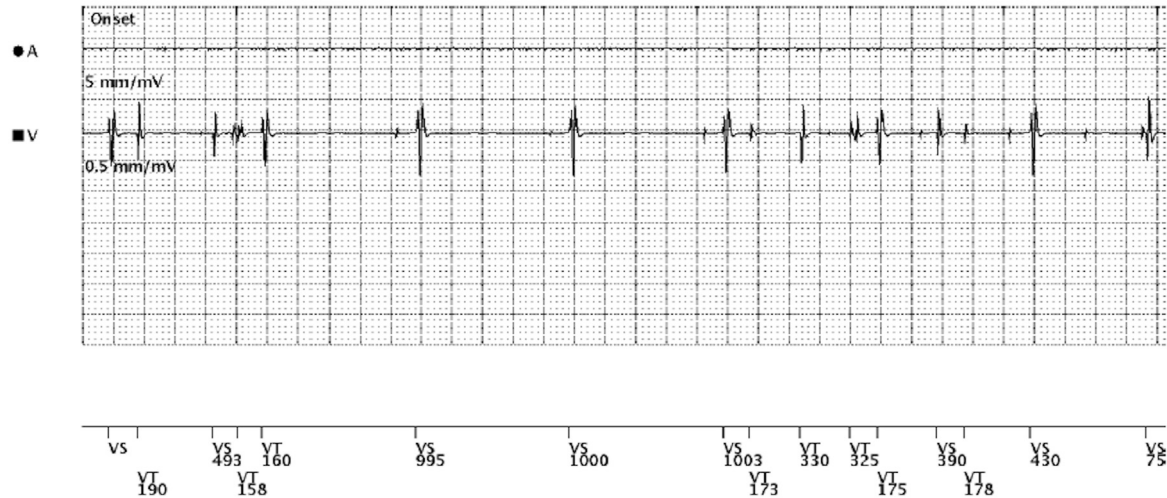


Fig. 2. Ventricular high rate event.

the ventricular sensed events were erroneously marked as falling into the ventricular refractory period and visa-versa (Fig. 2). The differential diagnoses at this point were – lead fracture, loose set screw, or insulation failure.

The arm on the side of the implant was moved to reproduce these EGMs on the OPD, but there was no effect of arm movement on the EGMs. So, fracture or insulation failure in the extra-thoracic part of the lead was ruled out. Chest X-ray did not show any evidence of loose set screw, insulation break, or any obvious lead fracture along the lead length. All the obvious causes were ruled out, but the patient was symptomatic and lead noise was very clear.

Since the lead noise was only seen in the ventricular channel, it was suspected to be due to microfracture or insulation break, which may be missed in routine radiological evaluation. Due to the significant symptoms and evidence of oversensing on the ventricular channel, the ventricular lead was replaced at the time of the pulse generator change. The old lead was not extracted. After replacement, there was no noise on the ventricular channel and the patient

is completely asymptomatic on 12 months follow-up.

2. Discussion

Transvenous mechanical leads of pacemakers are expected to operate in a chemically hostile environment and under high mechanical stress [1]. So, these can get damaged, which is termed lead dysfunction (LD) [2].

Appropriate follow-up evaluation of pacemakers is essential to ensure patient safety, provide appropriate physiological pacing, and maximize device longevity. The pacing-sensing testing and impedance analysis may sometimes be spuriously normal even with significant electrical LD. In our case, multiple ventricular ectopics and ectopic burden suggested the possibility of lead dysfunction but there was no specific finding to pinpoint the diagnosis to lead microfracture or insulation break. So, there should be a high degree of suspicion, especially if the patient is having significant symptoms.

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