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

Saurabh Ajit Deshpande a, *, Ameya Udyavar b

a CTVS, Jag Jivan Ram Western Railway Hospital, Mumbai, India
b Cardiology, Institution: P D Hinduja Hospital, Mumbai, India

Article history:
Received 22 August 2021
Received in revised form 7 October 2021
Accepted 15 October 2021
Available online 16 October 2021

Keywords:
Lead dysfunction
Ventricular tachycardia
Normal lead impedance
Lead revision

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 (RVtip--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 TV 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
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.
Lead noise may be seen in LD due to electromagnetic interference (EMI), myopotentials (diaphragmatic/skeletal muscle), or lead-related problems viz. lead fracture, insulation failure, or connector problems (Table 1) [3]. EGMs related to EMI have a significant history and are generally detected in all the pacing channels with some specific exceptions, but there is no change in the lead hardware parameters. Also, the noise associated with EMI is a high-frequency continuous signal. EGMs related to myopotentials are noted on the lead spatially closer to the muscles (e.g. diaphragmatic – right ventricular lead, pectoral – unipolar leads), can be reproduced by specific maneuvers, and have high frequency with low amplitude [4]. EGM’s due to LD are noted in the specific lead channels, especially in the case of a bipolar lead. LD is generally associated with abnormal lead parameters and may show clues on a chest X-ray. In our patient, the lead was programmed as bipolar (for pacing and sensing), X-ray was not helpful, and the noise was irregular with low frequency, detected only in the ventricular channel.

Despite these general assumptions sometimes lead fracture or insulation break can have a lead threshold and impedance in the normal range. Ellenbogen et al. [5] have shown that the diagnosis of lead fracture may be missed if we rely solely on lead impedance. Even insulation breaks can present with near-normal impedance [6]. Our patient also showed similar findings. In such cases, we need to look for other signs suggestive of LD. In our case, there were several episodes of ventricular tachycardia events and PVCs, which were increased over the last few months. Also, there was evidence of reset of the ventricular timing cycles, which may have caused loss of pacing and syncope during a few of these events. This is a rare finding and highlights the importance of an in-depth analysis of the EGMs.

Noise reversion algorithms can be useful to protect against prolonged inhibition of pacing from oversensing due to noise and to ensure continuous pacing, which is very important in pacing dependant patients. In ICDs, noise reversion algorithms ensure that the high frequency EGM’s are not classified as tachyarrhythmia which prevents inappropriate shocks [6]. These noise events are detected during noise sampling window which is programmed just after blanking period in that chamber. Devices have different responses to first detection of noise (e.g. resetting of entire refractory period, resetting of noise sampling window only or reversion to asynchronous pacing) but repeated detection shifts the pacemaker to asynchronous mode [7].

Repeat pacemaker procedures have a significant risk of complications [1]. So, a clinician should weigh the risks and benefits of a repeat procedure. Our patient had significant symptoms due to LD. So, it was planned to change the lead. The patient has had no complaints on the follow-up.

3. Conclusions
A thorough evaluation of the patient is important when there is suspected pacemaker and/or lead dysfunction causing significant symptoms. It is possible to have a near-normal threshold and normal impedance in a patient with a lead microfracture or insulation failure. It may be prudent to change the damaged lead to avoid any other complications associated with syncope in these patients.

Informed consent
Informed consent has been obtained from the patients regarding the use of case details and images for publication. It has been discussed to keep all the data anonymized during all the publications.

CRediT statement
Ameya Udyavar: Conceptualization, Methodology, Investigation, Resources, Writing – Review and Editing, Supervision; Saurbh A Deshpande: Methodology, Formal Analysis, Writing - Original Draft, Visualization.

Funding
None.

Disclosure
Nothing to disclose.

References
[1] Kusumoto FM, Schoenfeld MH, Wilkoff BL, et al. 2017 HRS expert consensus statement on cardiovascular implantable electronic device lead management and extraction. Heart Rhythm 2017;14(12):e503–51.
[2] Nair SG, Swedlow CD. Monitoring for and diagnosis of lead dysfunction. Cardiac Electrophysiology Clinics 2018;10(4):573–99.
[3] Kowalski M, Ellenbogen KA, Wood MA, Friedman PL. Implantable cardiac defibrillator lead failure or myopotential oversensing? An approach to the diagnosis of noise on lead electrograms. Europace 2008;10(8):914–7.
[4] Pinski SL, Trohman RG. Interference in implanted cardiac devices, part II. Pacing and Electrophysiology Journal 2009;22(3):276–81.
[5] Ellenbogen KA, Gunderson BD, Stromberg KD, Swerdlow CD. Performance of Lead Integrity Alert to assist in the clinical diagnosis of implantable cardioverter defibrillator lead failures: analysis of different implantable cardioverter defibrillator leads. Circ Arrhythm Electrophysiol 2013;6(6):1169–77.

[6] Swerdlow CD, Koneru JN, Gunderson B, Kroll MW, Ploux S, Ellenbogen KA. Impedance in the diagnosis of lead malfunction. Circ Arrhythm Electrophysiol 2020;13(2):e008092.

[7] Pinski SL, Trohman RG. Interference in implanted cardiac devices, Part I. Pacing Clin Electrophysiol 2002;25(9):1367–81.