Case Report

Dizziness spells: Should one suspect the pacemaker?

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Abstract

A 52-year-old lady presented to the emergency department with recurrent episodes of dizziness and near-syncope. Initial rhythm strips demonstrated intermittently non-conducted p waves corresponding to inappropriate pacemaker inhibition and oversensing malfunction. Pacemaker was interrogated in the ED showing ventricular lead noise and decreased lead impedance over a one year period. The patient was diagnosed with pacemaker lead failure supported by correlating pacemaker lead variation with homolateral upper limb movement. The patient was referred to an electrophysiologist and underwent new right ventricular lead placement with the resolution of symptoms.

Keywords: Dizziness, Lead fracture, Pacemaker malfunction

1. Background

Lead failure has been recognized as a potential complication of pacemakers. Lead failure may occur due to fracture, insulation break or a problem between the header and the lead [1-3]. Although lead failure may be suspected radiologically on chest x ray when lead fracture is identified, clues to the diagnosis may include the clinical presentation, initial electrocardiogram and pacemaker interrogation tracings. We present a classic case of pacemaker lead failure along with few clinical and electrocardiographic keys to the appropriate diagnosis.

2. Case

A 52 year-old lady presented to the emergency department (ED) with multiple episodes of dizziness and near-syncope. Symptoms have been intermittent for a one-week duration. Patient reported symptoms to be worse upon awakening up or swinging her arm. She used to sleep on her left side with her arm flexed. Patient denied any associated palpitations or chest pain. Patient’s past medical history is significant for a high-grade atrioventricular block resulting in dual chamber pacing [1-3]. Some risk factors leading to lead failure have been identified in the literature including acute entry angle, sharp turn in the generator pocket, tight sutures and young age [3]. Limb or truncal movements may lead to pulling both ends of the insulation break resulting in myopotential sensing. Pacemaker placement 11 years prior. In the ED, Blood pressure was recorded 128/76 mmHg, HR 79 bpm with no orthostatic changes noted on sitting or standing up at 1 and 3 min. Physical exam was unremarkable. Labatory and chest X-ray didn’t show any abnormalities. Full disclosure of initial rhythm strips demonstrated oversensing malfunction and loss of capture (Fig. 1C). Pacemaker was interrogated in the ED and corresponding electrocardiogram tracing demonstrated ventricular lead noise initially misinterpreted as ventricular tachycardia (Fig. 1D). Right ventricular lead impedance was noted to decrease since last interrogation a year prior from 660 to 540 Ohms. The patient was diagnosed with pacemaker lead failure and definite diagnosis was made possible in our case by correlating pacemaker lead variation with homolateral arm movement. Patient was referred to an electrophysiologist and underwent new right ventricular lead placement with resolution of symptoms.

3. Discussion

Pacemaker consists of two major components; the generator or can, which include the battery and electronics, as well as the leads, which travels from the can to the myocardium. Leads sense the myocardium intrinsic activity and deliver the external electrical depolarizing stimulus via the lead tip electrodes. Such tip electrodes can be fixed in the myocardium by an electrically active helix or electrically inert tines that only serve to anchor the lead. Electrodes are separated from the conductor cables via insulation materials. Disruption of insulation materials may result in insulation break with low impedance (short-circuiting), as in our case, or lead fracture with high impedance [3,4]. It appears that both insulation break and lead fracture result in lead failure [3]. Lead failure may occur at any site though most commonly occurs distal to the site of subclavian venous entry [1,2]. Many risk factors leading to lead failure have been identified in the literature including acute entry angle, sharp turn in the generator pocket, tight sutures and young age [3]. Limb or truncal movements may lead to pulling both ends of the insulation break resulting in myopotential sensing.
considers myopotentials as spontaneous ventricular activity leading to an oversensing malfunction.

Sensing is a pacemaker function that allows recognition of the chamber cardiac cycle timing where the lead tip is placed. Effective sensing algorithms can signal out near-field repolarization signals (T-waves) and far-field signals (tissue signals not in contact with electrode) [3]. False sensing T waves or extra-cardiac electric activity leads to oversensing malfunction resulting in inappropriate inhibition of the pacing function. In our case, myopotentials by extra-cardiac tissues were misinterpreted by the pacemaker as ventricular activity (oversensing). Oversensing malfunction is evident on the initial rhythm strip by observing the intermittently non-conducted p waves corresponding to false sense of myopotentials as ventricular activity leading to inappropriate pacing inhibition (Fig. 1B). Pacemaker ECG tracing reafirms the aformentioned observation (Fig. 1D). Sensing frequency range is usually set for atrial channels between 80 and 100 Hz, and for ventricular channels between 10 and 30 Hz range [5,6]. The above-optimized parameters typically result in recorded signal amplitudes on electrocardiogram between 1.5 and 5 mV in the atrial leads and 5 to 25 mV in the ventricular leads [3,5,6]. Lead noise doesn’t reach the pulse voltage as ventricular waves and shouldn’t be misinterpreted as ventricular electric activity usually set at 5 to 25 mV as we previously mentioned.

Fig. 1. Chest X-ray (AP view) demonstrating dual chamber pacemaker (Panel A). Rhythm strip shows atrial sensed ventricular paced rhythm with intermittently non-conducted p waves (Panel B) and intermittent loss of capture (Panel C). Pacemaker interrogation electrocardiogram showing ventricular lead noise, which may be misinterpreted as ventricular tachycardia (Panel D).

Lead failure diagnosis may as well be recognized by loss of capture as evident on the initial rhythm strip (Fig. 1C), which indicates that affected lead is not delivering appropriate electric output to initiate myocardial depolarization. Finally, it’s imperative to check the chest X-ray to rule out any visible lead fractures and confirm proper position of pacemaker leads [3,7] (Fig. 1A).

4. Conclusion

Lead failure should be suspected when pacemaker oversensing is evident even in absence of radiological evidence. Clinical presentation, loss of capture and change of lead impedance are all other significant clues to confirm the diagnosis.

Conflict of interests

Authors declare no Conflict of Interests for this article.

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References


