Phrenic nerve stimulation, a rare complication of pacemaker
A case report

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Abstract
Rationale: The phrenic nerve stimulation (PNS) is a rare complication after pacemaker setting. We report a case report that describes this complication and how it can be resolved.

Patient concerns: An 88-year-old man presented himself to the emergency geriatric unit with intermittent painless abdominal contraction due to phrenic nerve stimulation. He has a history of transcatheter aortic valve implantation with cardiac resynchronization therapy pacemaker due to persistent left bundle branch block.

Diagnoses: All the usual causes for abdominal spasms were eliminated and the possibility of a link with the pacemaker was considered. The phrenic nerve stimulation is a rare complication of a pacemaker implantation. It can be clinically nonrelevant but challenging to diagnose for those not familiar with cardiac devices technology.

Interventions: Initial setting was an axis of stimulation between distal left ventricular (LV) and right ventricular. It was changed to LV and D1-M2.

Outcomes: This noninvasive procedure managed to eradicate the involuntary abdominal spasms.

Lessons: PNS could be challenging to diagnose for those not familiar with cardiac devices technology but easy to manage with noninvasive methods.

Abbreviations: CRT-P = cardiac resynchronization therapy pacemaker, CT = computed tomography, LV = left ventricular, PNS = phrenic nerve stimulation.

Keywords: complication, older people, pacemaker, phrenic nerve stimulation

1. Introduction
Pacemaker implantation is a frequent procedure in older people.[1] Complications can occur in 6% to 12.6% of cases. Common complications include hematomas, pneumothorax, cardiac injury or tamponade, lead dislodgement, deep venous thrombosis, infection and lead or device malfunctions.[2] However rarely, phrenic nerve stimulation (PNS) can occur.
2. Case report

The patient, an 88-year-old man, was admitted to the Emergency Geriatrics Unit, in January 2019, after an uncomplicated fall. He lives with his wife in an apartment and was walking with a cane, his frailty score was 4 out of 9 (vulnerable). He was hemodynamically stable.

He has a history of atrial fibrillation, heart failure with normal ejection fraction, hypercholesterolemia, and transcatheter aortic valve implantation with cardiac resynchronization therapy pacemaker (CRT-P) due to persistent left bundle branch block in May 2017 (Quadra Allure MP, St Jude Medical).

At admission, cardiac, lung, neurological examination was unremarkable. However, we noticed visible and palpable intermittent painless abdominal contractions in the left hypochondrium without any evidence of underlying mass or pulses on palpation. The contraction was less intense when he held his breath. The patient had noticed this spasm for a long time, but never bothered him. This spasm was present in decubitus and did not change with position. We did not notice any dyspnea or loss of consciousness.

Blood sample analyses showed no inflammatory syndrome (c-reactive protein < 10 mg/L), normal calcium and magnesium levels (2.33 mmol/L [2.2–2.52] and 0.63 mmol/L [0.59–0.83] respectively). Electrocardiogram was showing an atrial and ventricular pacing. No sign of pulmonary infection or nodal were detected on chest X-ray and pacemaker leads were in place.

After excluding ruptured abdominal aortic aneurysm with a computed tomography, the most likely diagnosis was PNS.

CRT-P, in our patient, was a quadripolar device with 3 leads: 1 in the right atrium (auriculus), 1 in right ventricle (septal), and the last 1 in the left ventricle (lateral coronary sinus vein). Left ventricular (LV) pacing was unipolary mode (2.5 Volts with pulse width of 0.4 mSec). Pacing mode was DDD with basal frequency and metabolic disorders such as hypercalcemia, hypocalcaemia, and hypomagnesemia can cause diaphragmatic contractions.

PNS is frequently reported after CRT-P (around 30%) but clinical impact is relevant in only 3% to 26% of patients. The electrical impulse delivered by the LV lead may incidentally cause PNS. Anatomic explanation of this phenomenon is the left electrical impulse delivered by the LV lead may incidentally cause PNS.

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In our patient case, PNS appeared approximately 1 year after implantation. Reason for this late appearance was not very clear; minimal lead displacement, and/or cardiac remodeling could be an explanation. It was clinically significant for the patient but easy to manage with the change of pacing vector. Quadripolar devices allow more possibility to eradicate PNS. Early recognition and management is essential.

This clinical case demonstrates the importance of identifying this complication, because of its simplicity of resolution, specifically with noninvasive methods.

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Author contributions

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