

C A S E R E P O R T

Reverse Reel syndrome – An unusual case of myocardial perforation and liquidopneumothorax caused by migrated ventricular pacemaker lead

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Abstract. Reverse Reel Syndrome represents a rare but potentially life-threatening complication in patients with cardiac implantable electronic devices (CIEDs). This condition, characterized by the abnormal displacement and distal migration of pacemaker leads, can lead to severe adverse events due to tissue and organ damage caused by the sharp tip of an active lead. Our case report highlights a unique instance of Reverse Reel Syndrome in an elderly female patient with early ventricular pacemaker lead distal migration resulting in life-threatening right ventricular myocardial perforation and left-sided liquidopneumothorax. Discussing the patient's characteristics, clinical presentation, and therapeutic considerations as essential factors in managing this complication, we emphasized the importance of preventive measures during implantation to mitigate risks associated with CIED complications. (www.actabiomedica.it)

Key words: Reverse Reel syndrome, myocardial perforation, pacemaker lead displacement, cardiac implantable electronic devices (CIED), idiopathic lead migration, Twiddler's syndrome

Introduction

Cardiac implantable electronic devices (CIEDs), including pacemakers and implantable cardioverter-defibrillators (ICDs), have significantly improved life expectancy and quality of life for patients with cardiac arrhythmias and other conduction abnormalities. However, while CIEDs are generally safe and reliable, complications related to device implantation and lead stability can occasionally arise. Abnormal displacement, including retraction and advancement of the leads, also known as lead migration, occurs extremely rarely (1). Although it is usually caused by deliberate or inattentive manipulation of the pulse generator, as first described in 1968. as Twiddler's syndrome, sometimes an apparent cause is absent (2). Considering the causes

and mechanisms of lead migration, well-documented entities such as Twiddler's, Reel, and Ratchet syndromes, as well as their reverse variants, are reported in everyday clinical practice (3-6). Also, a new term, idiopathic lead migration syndrome (ILMS), is proposed, which encompasses unknown causes of lead dislodgement (7). The phenomenon known as Reverse Reel Syndrome, a rare complication involving the distal migration of pacemaker leads, can have profound mechanical and electrical effects due to tissue and organ damage. Mechanical consequences occur as a result of tissue and organ damage of cardiac, other thoracic or extrathoracic organs caused by the protruding sharp tip of the active lead (8). It includes pericarditis, pericardial effusion, cardiac tamponade, pleural effusion, pneumothorax or hemothorax, and abdominal and

visceral injury (9). The electrical consequences refer to the loss of sensing, loss of capture, and extracardiac stimulation, including stimulation of the pectoral, intercostal, and diaphragmatic regions (10). This paper will explore a specific case of Reverse Reel Syndrome leading to myocardial perforation and liquidopneumothorax, highlighting the unique clinical challenges in treatment decisions.

Case Report

A 91-year-old female with a medical history of arterial hypertension and permanent atrial fibrillation presented to the emergency department with chest discomfort and dyspnea. She was a retirement home resident with limited mobility and depended on other people's care. Other comorbidities included a suspected urinary bladder neoplasm that was not further evaluated due to the patient's advanced age. Six days earlier, she received a single-chamber VVI pacemaker due to intermittent complete atrioventricular block. A pulse generator (Abbott Endurity PM 1162 MRI) was placed subcutaneously into the right pectoral region, and an active screw-in ventricular lead (Tendril STS 2088 TC 58 cm) was placed into the right ventricular apex using the right subclavian transvenous approach. Previous medical therapy consisted of a calcium channel blocker (CCB), amiodarone, benzodiazepine, and a proton pump inhibitor (PPI). A 12-lead electrocardiogram (ECG) revealed a

loss of capture with a slow atrial fibrillation rhythm. A complete blood count revealed a normal leukocyte count with slightly elevated neutrophilic granulocyte count of $7,08 \times 10^9/L$ and CRP level of 53,4 mg/L. Hemoglobin and platelet count were normal. An arterial blood gas analysis confirmed mild hypoxemia with a borderline arterial oxygen saturation (SpO_2) of 94% and partial arterial oxygen (PaO_2) pressure of 8.02 kPa, while carbon dioxide and pH levels were normal. Serum biochemistry revealed marked NT-proBNP of 1089,6 ng/L. Liver and kidney function tests, as well as coagulation tests, were normal. Device interrogation revealed loss of sense and capture of the ventricular lead. Diagnostic imaging, including chest radiography and computed tomography, confirmed that the pacemaker lead had advanced through the myocardium and pleural parenchyma to the left costophrenic sinus, causing significant pleural effusion and lung collapse (Figure 1). The pulse generator was stationary, located inside the pocket, but in a flipped-around position along its transverse axis and without evident lead coiling. The patient denied deliberate and intense manipulation of the pacemaker. Ultimately, she was admitted to the ICU.

A heart ultrasound revealed a discrete zone of organized pericardial effusion along the right ventricle without hemodynamic influence. The left ventricle was moderately enlarged with mildly hypertrophic walls. The ejection fraction was estimated as mildly reduced due to global hypokinesis. The left atrium was enlarged with a left atrial volume index (LAVi) of

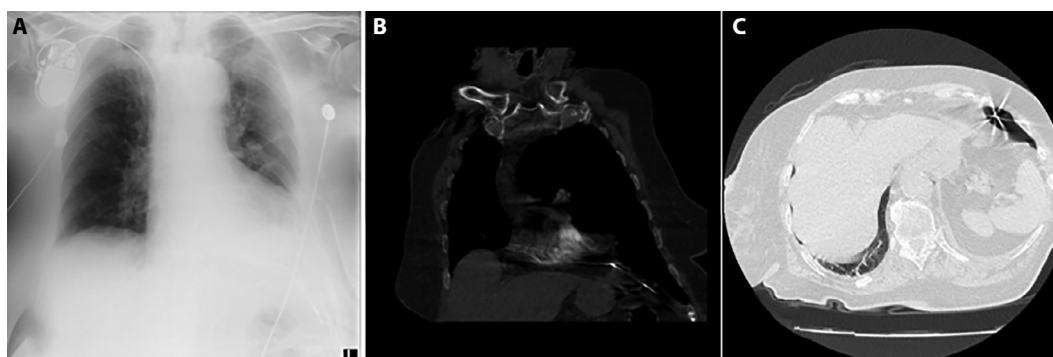


Figure 1. Chest radiogram (A): Notable left-sided pleural effusion. The pulse generator is rotated to the right side about its long axis, and collimation of the lead is absent. Notice the displacement of the ventricular lead with the tip located in the left phrenicocostal sinus. The chest CT scan shows a displaced ventricular lead in the coronal plane (B) and in a transverse plane (C).

36 mL/m². Moderate mitral and tricuspid regurgitation were present. Due to respiratory insufficiency, an urgent thoracentesis with negative pressure drainage was performed (Figure 2).

Drained hemorrhagic content was classified as transudative effusion using Light's criteria. Given the high risk of fatal bleeding associated with lead repositioning, we adopted a “cap and abandon” strategy, opting to leave the migrated lead in situ to maintain compression at the perforation site. A new ventricular lead was implanted via the same access site at the mid-septal level (Figure 3) (9,11).

Repeated chest radiograms verified significant regression of left-sided pleural effusion and lung re-expansion. The patient was treated with appropriate antibiotic therapy directed at gram-positive bacteria. Repeated laboratory exams confirmed the normalization of inflammation parameters. The patient made a

complete recovery and was free from the initial symptoms. Device interrogation confirmed appropriate sensing and capture parameters and lead impedance at follow-up 1 and 3 months after the hospitalization.

Discussion

CIED lead macrodislodgement and distal migration resulting in myocardial perforation and life-threatening mechanical injuries of other intrathoracic organs is an infrequent complication of transvenous pacing and cardioverter-defibrillator leads (9). The overall incidence ranges from 0.1 to 1.0% with pacemakers and 0.6 to 5.2% with ICD implantation, predominantly in the subacute phase (within one month) rather than the delayed phase after implantation (9,11,12). In the available literature, the proposed mechanism of such a phenomenon involves reverse Twiddlers and reverse Reel syndrome (8,12). Both syndromes are characterized by the distal advancement of leads due to the rotation of the PG. More precisely, the pulse generator is rotated about its long axis in reverse Twiddler's and about a transverse axis in reverse Reel syndrome. The dissection of myocardial fibers explains the advancement of the lead due to its fixed position and the constant pressure exerted during cardiac contractions (13,14). Furthermore, higher age (above 80 years), female sex, lower body weight, and the external characteristics of the lead, such as smaller diameter, stiffness, and active fixation mechanism, as well as the location of placement, represent risk factors for a higher

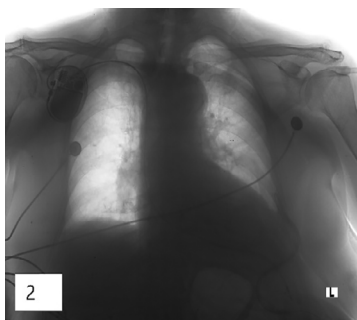


Figure 2. A negative chest radiogram (anteroposterior view) highlights a chest tube placed in the mid-axillary line of the sixth intercostal space.

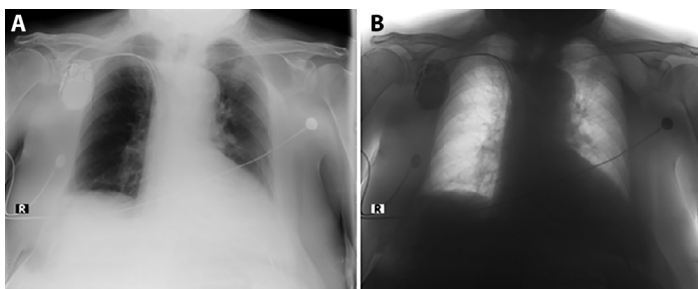


Figure 3. Chest radiogram (A) shows a new lead implanted via the same venous access site into the mid-septal position. Notice the regression of pleural effusion compared to image A. Negative chest radiogram (B) highlighted the newly implanted ventricular lead and the abandoned lead.

incidence of perforation (8). Treatment approaches in these conditions typically depend on the patient's clinical status, the risk of removing the lead, and the availability of surgical backup. Generally, percutaneous strategies, such as lead abandonment, repositioning, or retrieval, and open thoracic surgical procedures can be considered (8,11). Many of the aforementioned risk factors were recognized in our patients. Possible rotation of the pulse generator about the transverse axis without coiling of the advanced lead indicates possible reverse Reel syndrome. We decided to "cap and abandon" the old lead and implant new ventricular lead at a safer mid-septal level via the same access site. Our decision was emphasized on the potential for fatal bleeding after repositioning of the old lead that served as a compress of the cardiac perforation site. Also, the patient's higher age automatically excluded thoracotomy as a treatment option. The resolution of symptoms after thoracic drainage reaffirmed our strategy.

Reverse Reel syndrome is an unusual but serious complication of CIED implantation that necessitates timely diagnosis and a tailored therapeutic approach. The presented case highlights the importance of a comprehensive understanding of the risk factors and mechanisms underlying pacemaker lead migration. Clinicians should adopt preventive strategies, such as secure pulse generator and lead sleeve anchoring, cautious active lead fixation, and avoiding the formation of large pockets with excessive redundant space, as well as rigorous patient education, to reduce the likelihood of lead displacement and associated complications. As the use of CIEDs continues to grow, increased awareness of rare complications, such as Reverse Reel Syndrome, will be essential in improving patient outcomes and device longevity.

Ethical Approval: All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. This is an observational study. For this type of study, formal consent is not required because no personal data was contained, and there is no concern about identifying information.

Conflict of Interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity

interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

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References

1. Tseng AS, Shipman JN, Lee JZ et al. Incidence, patterns, and outcomes after transvenous cardiac device lead macrodislodgment: Insights from a population-based study. *Heart-Rhythm*. 2019 Jan;16(1):140-147. doi: 10.1016/j.hrthm.2018.07.026
2. Bayliss CE, Beanlands DS, Baird RJ. The pacemaker-twiddler's syndrome: a new complication of implantable transvenous pacemakers. *Can Med Assoc J*. 1968 Aug 24-31; 99(8):371-3. PMID: 4952398.
3. Vlay SC. Reverse Twiddler's syndrome. *Pacing Clin Electrophysiol*. 2009 Jan;32(1):146. doi: 10.1111/j.1540-8159.2009.02191.x
4. Carnero-Varo A, Pérez-Paredes M, Ruiz-Ros JA et al. Reel Syndrome: a new form of Twiddler's syndrome? *Circulation*. 1999 Aug 24;100(8):e45-6. doi:10.1161/01.cir.100.8.e45
5. Von Bergen NH, Atkins DL, Gingerich JC, Law IH. Ratchet syndrome, another etiology for pacemaker lead dislodgement: a case report. *HeartRhythm*. 2007 Jun;4(6): 788-9. doi:10.1016/j.hrthm.2006.12.029
6. Machraa A, Sidaty O, Fellat N, Fellat R. An unusual cause of pacemaker malfunction: A case report of an association of twiddler and reel syndrome. *Ann Med Surg (Lond)*. 2022 Nov 11;84:104857. doi: 10.1016/j.amsu.2022.104857
7. Morales JL, Nava S, Márquez MF et al. Idiopathic Lead Migration: Concept and Variants of an Uncommon Cause of Cardiac Implantable Electronic Device Dysfunction. *JACC Clin Electrophysiol*. 2017 Nov;3(11):1321-1329. doi: 10.1016/j.jacep.2017.02.015
8. Vlay SC. Complications of active-fixation electrodes. *Pacing Clin Electrophysiol*. 2002 Aug;25(8):1153-4. doi: 10.1046/j.1460-9592.2002.01153.x
9. Simsolo E, Wilkoff BL. A Shocking Case of Pacemaker Lead Perforation. *JACC Case Rep*. 2022 Sep 21;4(18): 1203-1205. doi: 10.1016/j.jaccas.2022.07.003
10. Salahuddin M, Cader FA, Nasrin S, Chowdhury MZ. The pacemaker-twiddler's syndrome: an infrequent cause of pacemaker failure. *BMC Res Notes*. 2016 Jan 20;9:32. doi:10.1186/s13104-015-1818-0

11. Hirschl DA, Jain VR, Spindola-Franco H, Gross JN, Haramati LB. Prevalence and characterization of asymptomatic pacemaker and ICD lead perforation on CT. *Pacing Clin Electrophysiol.* 2007 Jan;30(1):28-32. doi: 10.1111/j.1540-8159.2007.00575.x
12. Alla VM, Reddy YM, Abide W, Hee T, Hunter C. Delayed lead perforation: can we ever let the guard down? *Cardiol Res Pract.* 2010 Jul 25;2010:741751. doi: 10.4061/2010/741751
13. Forsell Caroline, Gasser T. Christian . Numerical simulation of the failure of ventricular tissue due to deep penetration: the impact of constitutive properties. *Journal of biomechanics* 44, no. 1 (2011): 45-51. doi.org/10.1016/j.jbiomech.2010.08.022
14. Archontakis, S, Sideris, K, Aggeli, K, et al. Percutaneous lead extraction and repositioning: An effective and safe therapeutic strategy for early ventricular lead perforation

with dislocation both inside and outside the pericardial sac following a cardiac device implantation. *J cardiovasc Electrophysiol.* 2019; 30: 299 307. <https://doi.org/10.1111/jce.13804>

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