Cureus
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Open Access Case Report

Early Right Ventricular Implantable Cardioverter-Defibrillator Lead Microdislodgement Associated With Active-Fixation Helix Retraction: A Case Report

Review began 09/24/2026
Review ended 10/16/2026
Published 10/21/2026

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DOI: 10.7759/cureus.118307

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Abstract

Lead dislodgement is an important early complication of cardiac implantable electronic device (CIED) implantation, and microdislodgement may be clinically significant despite little or no visible change in lead position on standard radiography.

We report the case of an 87-year-old man with ischemic dilated cardiomyopathy, previous transcatheter aortic valve implantation (TAVI), and a pre-existing right-sided pacemaker for high-degree atrioventricular block. A dual-chamber implantable cardioverter-defibrillator (ICD) was implanted for secondary prevention after syncope associated with ventricular tachycardia. The newly implanted right ventricular (RV) defibrillator lead had satisfactory acute electrical parameters. Within the following days, the patient developed recurrent symptoms, slow ventricular tachycardia, and dynamic RV lead dysfunction evident during upper-limb mobilization. Device interrogation before lead revision showed an elevated RV pacing threshold, reduced R-wave amplitude to 2-3 mV, and an increase in lead impedance to 1500 Ω. Retraction of the active-fixation screw was identified. A few days after implantation, a new RV defibrillator lead was positioned at the RV apex, after which the dysfunctional lead was removed. Subsequent device follow-up was reassuring, with no recurrent RV lead dysfunction documented at long-term follow-up.

This case highlights the diagnostic challenge of early ICD lead microdislodgement and the value of integrating symptoms, device interrogation, telemetry, imaging, and dynamic mechanical assessment.

Categories: Cardiology, Emergency Medicine, Healthcare Technology
Keywords: active-fixation lead, device interrogation, implantable cardioverter-defibrillator, lead dysfunction, lead microdislodgement, loss of capture, ventricular escape rhythm

Introduction

Lead dislodgement remains one of the most frequent early complications after implantation of a cardiac implantable electronic device (CIED). Contemporary expert guidance reports dislodgement in approximately 1.2%-3.3% of implantations, with most events occurring before discharge [1]. In a large real-world registry of 20,683 patients, lead dislodgement occurred in 1.69% of patients and involved 0.95% of implanted leads [2]. Dislodgement may be radiographically evident (macrodislodgement) or radiographically occult (microdislodgement); the latter may manifest primarily through changes in capture threshold, sensing, or intermittent loss of capture despite little or no visible lead-tip migration [1,3-5].

The right ventricular (RV) implantable cardioverter-defibrillator (ICD) lead senses ventricular electrical activity, provides pacing support when required, and delivers defibrillation therapy [1,6]. Active-fixation leads use a screw-in helix to anchor the lead tip to the myocardium; in retractable-helix designs, residual torque may promote helix retraction and compromise myocardial engagement and electrical stability [1,7]. Helix auto-retraction has previously been described in a pacemaker lead used for deep septal pacing [7]; the present case differs in involving an ICD defibrillation lead with clinically significant dynamic lead dysfunction, unmasked by upper-limb mobilization, despite the absence of obvious macrodislodgement. These events are particularly important in ICD recipients because lead dysfunction may compromise pacing and sensing and may interfere with appropriate detection or treatment of ventricular arrhythmias [1,6].

We report an early RV ICD lead microdislodgement occurring shortly after ICD implantation, associated with retraction of the active-fixation helix and clinically significant lead dysfunction. This case highlights the diagnostic importance of correlating device interrogation with dynamic assessment when significant electrical dysfunction occurs despite the absence of obvious macroscopic lead displacement.

Case Presentation

An 87-year-old man had a history of heart failure with reduced left ventricular ejection fraction, ischemic dilated cardiomyopathy with previous left anterior descending coronary artery stenting and subsequent in-stent restenosis, and severe aortic stenosis treated by transcatheter aortic valve implantation (TAVI), complicated by valve thrombosis. He had previously undergone right-sided permanent pacemaker implantation for high-degree atrioventricular block. Additional comorbidities included arterial hypertension, dyslipidemia, chronic kidney disease, and a 50-pack-year smoking history.

The patient underwent implantation of a dual-chamber ICD for secondary prevention after syncope associated with documented ventricular tachycardia. The pre-existing right-sided pacemaker generator was explanted, and the retained pacing leads were capped. A contralateral left-sided approach was used. Venography demonstrated significant left subclavian venous stenosis (Figure *1A*); nevertheless, venous access was successfully obtained. A dual-chamber ICD was successfully implanted. The new RV defibrillator lead was positioned in the RV septo-apical region, with satisfactory acute electrical parameters: pacing threshold 0.75 V, R-wave amplitude 15.3 mV, and RV pacing impedance 720 Ω. The right atrial lead also showed satisfactory acute sensing and pacing parameters.

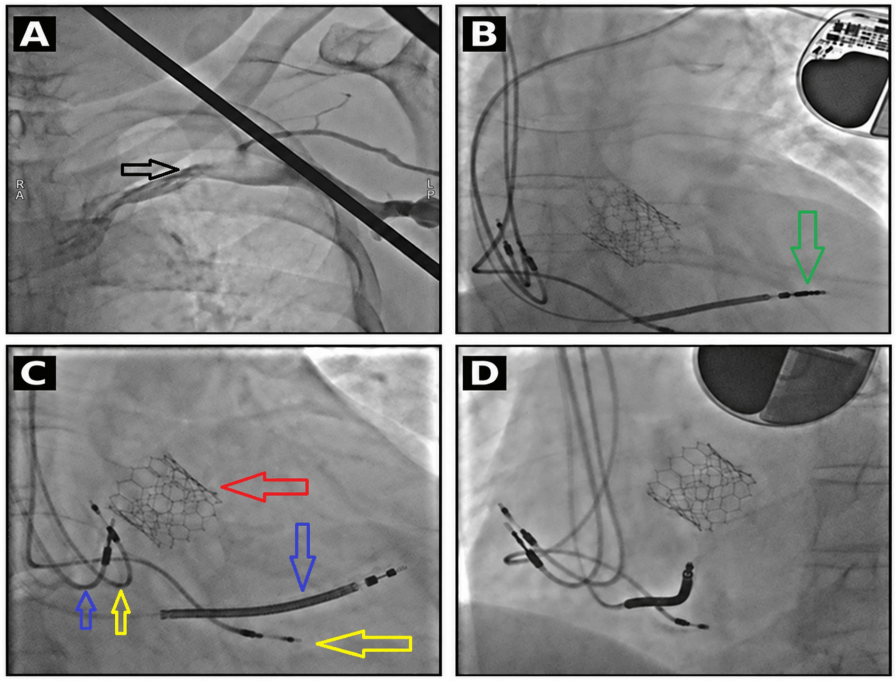


FIGURE 1: Fluoroscopic findings during implantable cardioverter-defibrillator implantation, right ventricular lead dysfunction, and subsequent lead revision.

(A) Left anterior oblique fluoroscopic venogram showing significant left subclavian venous stenosis (black arrow) encountered during left-sided implantable cardioverter-defibrillator implantation.

(B) Near-anteroposterior fluoroscopic view obtained at the time of right ventricular lead dysfunction, showing apparent retraction of the active-fixation helix (green arrow), without obvious macroscopic lead displacement.

(C) Right anterior oblique fluoroscopic view after lead revision, showing the ICD leads (blue arrows), the transcatheter aortic valve implantation prosthesis (red arrow), and the capped retained right-sided pacemaker leads (yellow arrows).

(D) Left anterior oblique fluoroscopic view obtained after lead revision, demonstrating the final position of the newly implanted right ventricular defibrillation lead.

The following day, the patient developed symptomatic tachycardia. ICD interrogation documented slow ventricular tachycardia, including an episode successfully terminated by antitachycardia pacing. No causal relationship was established between this episode of ventricular tachycardia and the subsequently documented lead dysfunction. On the second day after implantation, recurrent symptoms occurred during upper-limb mobilization; telemetry documented prolonged pauses of 6.6 s and 4 s with several non-conducted P waves. A 12-lead ECG obtained during RV lead dysfunction demonstrated a ventricular escape rhythm (Figure *2*). Evaluation raised concern for dysfunction of the newly implanted RV ICD lead. Before lead revision, device interrogation showed significant deterioration of the RV lead parameters, an elevated

RV pacing threshold, a decrease in R-wave amplitude from 15.3 mV to 2-3 mV, and an increase in RV pacing impedance from 720 Ω to 1500 Ω. The abnormality was dynamic and became evident during upper-limb mobilization. Clinical findings and device interrogation raised suspicion of mechanical lead instability. Fluoroscopic assessment of the lead tip identified retraction of the active-fixation helix despite the absence of obvious macroscopic lead displacement (Figure *1B*). Transthoracic echocardiography showed no pericardial effusion or other findings suggestive of lead perforation. On the fifth day after ICD implantation, lead revision was performed: a new RV defibrillator lead was first positioned at the RV apex, after which the dysfunctional lead was removed (Figures *1C*-*1D*). The procedure was uncomplicated. The subsequent course was favorable, with hemodynamic stability and no local complications.

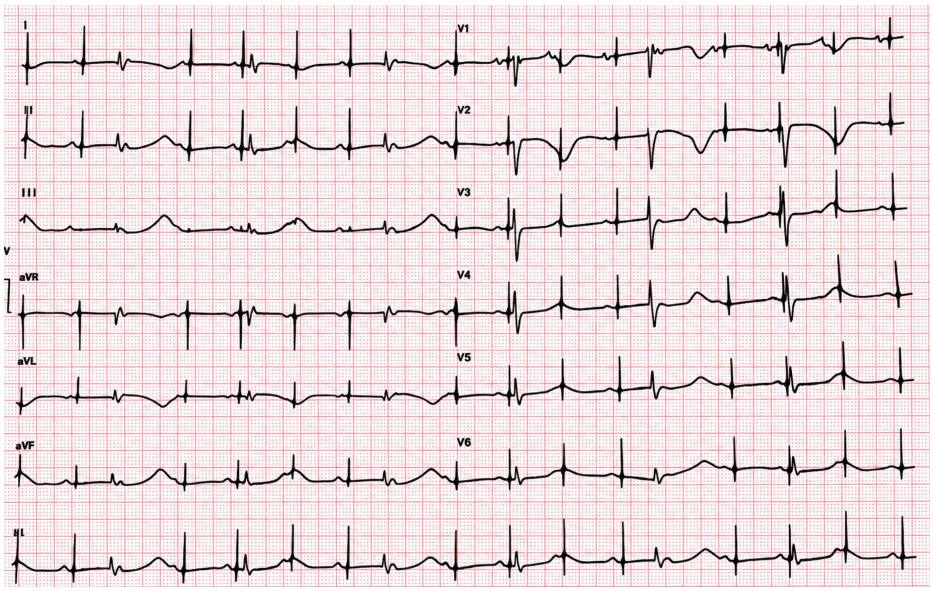


FIGURE 2: Ventricular escape rhythm during right ventricular lead dysfunction.

Twelve-lead electrocardiogram demonstrating a ventricular escape rhythm during right ventricular implantable cardioverter-defibrillator lead dysfunction.

The clinical timeline is summarized in Table *1*.

Timing	Clinical events and findings
Day 0	Dual-chamber ICD implantation with satisfactory RV lead parameters: pacing threshold 0.75 V, R-wave amplitude 15.3 mV, and impedance 720 Ω.
Day 1	Symptomatic tachycardia and documented slow ventricular tachycardia, including an episode terminated by antitachycardia pacing.
Day 2	Recurrent symptoms during upper-limb mobilization, prolonged pauses (6.6 and 4 s), and ventricular escape rhythm.
Pre-revision assessment	Elevated RV pacing threshold, decreased R-wave amplitude (2-3 mV), and increased pacing impedance (1500 Ω). Dynamic lead dysfunction without obvious macrodislodgement, with identification of helix retraction.
Day 5	Implantation of a new RV defibrillator lead at the apex, followed by removal of the dysfunctional lead.
Follow-up	Favorable clinical course without recurrent RV lead dysfunction during long-term follow-up.

TABLE 1: Clinical timeline of RV ICD lead dysfunction and management.

ICD: implantable cardioverter-defibrillator; RV: right ventricular

Discussion

Lead dislodgement is predominantly an early post-implant complication. The 2021 EHRA implantation

consensus reports a dislodgement rate of 1.2%-3.3% and emphasizes that most events occur before hospital discharge [1]. In the Product Surveillance Registry analyzed by Qin et al., 350 of 20,683 patients (1.69%) experienced a lead-displacement event, involving 371 of 39,060 implanted leads (0.95%) [2]. More recent reviews continue to identify lead dislocation as a clinically important mechanical complication of CIED therapy, particularly when it produces changes in sensing, capture, or pacing thresholds [3,4]. In our patient, advanced age (87 years), dual-chamber ICD implantation, and the presence of a pre-existing pacing system were notable characteristics potentially relevant to early lead dislodgement. However, their direct contribution to active-fixation helix retraction remains uncertain.

Microdislodgement must be distinguished from macrodislodgement. Macrodislodgement is visible on chest radiography or fluoroscopy, whereas microdislodgement denotes a small change in lead-tip position that may not be apparent on conventional imaging but is sufficient to alter the electrode-myocardium interface [3,5]. Electrical abnormalities can include an increased capture threshold or intermittent loss of capture, impaired sensing, and changes in lead impedance; these abnormalities may occur without obvious radiographic lead migration [2,5,6]. Consequently, a normal or apparently unchanged chest radiograph does not exclude clinically significant microdislodgement, and device interrogation is central to diagnosis [1,5,6]. Clinicians should suspect lead microdislodgement when new-onset syncope, bradycardia, prolonged pauses, or abnormal sensing and capture parameters occur shortly after ICD implantation, even in the absence of visible lead displacement [5,6]. In the present case, the change from satisfactory acute parameters (pacing threshold 0.75 V, R-wave amplitude 15.3 mV, impedance 720 Ω) to an elevated pacing threshold, R-wave amplitude of 2-3 mV, and impedance of 1500 Ω provided objective evidence of significant RV lead dysfunction despite the absence of macroscopic displacement. The differential diagnosis of early post-implant lead dysfunction includes lead dislodgement or malposition, lead maturation-related threshold changes, lead integrity abnormalities, and myocardial perforation [1,6]. In our patient, the very early onset, marked deterioration in sensing and pacing parameters, dynamic dysfunction during upper-limb mobilization, and documented retraction of the active-fixation helix favored mechanical lead instability and microdislodgement.

Several mechanisms may result in radiographically occult lead instability. Minimal alteration of the lead-tip-myocardial interface may impair capture despite an apparently unchanged lead position [5,6]. In active-fixation leads, incomplete helix engagement or subsequent helix retraction may compromise lead stability. EHRA guidance recommends fluoroscopic confirmation of helix deployment, avoidance of torque accumulation, and direct testing of lead stability after fixation [1]. Suzuki et al. reported spontaneous retraction of a retractable helix associated with residual torque, followed by lead displacement, providing a plausible mechanical mechanism whereby fixation-screw instability may lead to progressive loss of lead stability [7]. Mechanical tension related to lead slack and fixation may also influence lead stability, emphasizing the importance of adequate intravascular slack and secure fixation of the lead sleeve and generator to fascia [1,5]. The significant left subclavian venous stenosis observed in our patient may have increased procedural complexity; however, its direct contribution to lead instability or helix retraction remains uncertain.

In the present case, the documented retraction of the active-fixation screw strongly suggests loss of fixation as the principal mechanism of lead instability. The dynamic abnormality observed during upper-limb mobilization further suggests that mechanical tension may have transiently altered lead-tip contact. However, upper-limb movement should not be considered the proven cause of dislodgement. The EHRA consensus states that routine restriction of arm movement after implantation has not been shown to reduce lead dislodgement and may increase shoulder morbidity [1]. In this patient, movement is therefore better interpreted as a provocative maneuver that unmasked an already unstable lead-fixation system rather than as proof that normal mobilization caused the complication.

The clinical manifestations of lead microdislodgement depend on the affected device function. Acute loss of capture after implantation is most commonly related to lead dislodgement or malposition, and microdislodgement may remain radiographically occult [6]. In an ICD system, lead dysfunction is particularly relevant because the RV lead is responsible not only for pacing but also for ventricular sensing and delivery of defibrillation therapy. Device-related lead displacement can therefore present with bradycardia, pauses, syncope, sensing abnormalities, inappropriate therapies, or failure to deliver needed therapy, depending on the electrical consequence of the displacement [1,4]. The marked reduction in R-wave amplitude from 15.3 mV to 2-3 mV raised concern for potential ventricular arrhythmia undersensing, although the earlier episode of slow ventricular tachycardia was successfully detected and terminated by antitachycardia pacing. In our patient, recurrent symptoms, significant pauses, deterioration in RV lead electrical parameters, and dynamic lead dysfunction prompted urgent revision.

Management depends on timing, clinical consequences, pacing dependency, lead type, and the degree of electrical dysfunction [1,5]. When dislodgement is clinically significant, lead revision is generally required. The registry analysis by Qin et al. found that, among patients with lead dislodgement, implantation of a new lead was associated with a lower risk of subsequent dislodgement than repositioning the same lead (p=0.012) [2]. This observation is consistent with the strategy used in the present case: a new RV defibrillator lead was positioned at the RV apex, and the dysfunctional lead was subsequently removed rather than repositioned. A 2024 bicentric study of 249 patients undergoing lead reintervention found no evidence that delaying lead

revision beyond one week reduced CIED infection risk, supporting correction of clinically important dysfunction without an arbitrary delay [8].

Fixation strategy should also be considered in context. Active-fixation leads permit precise positioning and generally provide good stability, but their performance depends on correct helix deployment and avoidance of excessive torque [1,7]. A 2026 meta-analysis of 18 observational studies including 69,198 patients found a lower post-discharge dislodgement risk with active- versus passive-fixation leads (OR 0.71, 95% CI 0.54-0.94), although active fixation was associated with a higher risk of post-discharge cardiac perforation (OR 1.78, 95% CI 1.09-2.90) [9]. Thus, active fixation reduces but does not eliminate dislodgement risk, and meticulous implantation technique remains essential.

Finally, the indication for ICD implantation in this patient preceded the lead complication and was independent of it. The implantation report documents syncope associated with ventricular tachycardia as the indication for the dual-chamber ICD, consistent with a secondary-prevention strategy [10]. The key teaching point is therefore not that lead displacement created the indication for an ICD, but that an early mechanical complication of a newly implanted defibrillator lead produced clinically significant device dysfunction within days of implantation. At approximately 19 months of follow-up, device surveillance was reassuring, with no arrhythmic events recorded and satisfactory sensing, pacing thresholds, and impedances. Left ventricular ejection fraction was 35%, and no recurrent RV lead dysfunction was documented.

Conclusions

Early ICD lead microdislodgement can cause major clinical and electrical consequences despite minimal or absent radiographic change. In this patient, recurrent symptoms, prolonged pauses, and marked deterioration in RV lead parameters led to recognition of dynamic lead dysfunction, with fluoroscopic findings suggestive of active-fixation helix retraction. Lead revision with placement of a new RV defibrillator lead at the RV apex followed by removal of the dysfunctional lead resulted in a favorable outcome, with no recurrent RV lead dysfunction during follow-up. The case illustrates the importance of prompt device interrogation with comparison to baseline parameters, alongside clinical assessment, telemetry, dynamic assessment, and targeted imaging, while considering alternative causes of early lead dysfunction, including lead maturation-related threshold changes and myocardial perforation.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Drafting of the manuscript: Ayoub Moutaoakil, Wassim Beladel, Mohamed El Minaoui

Critical review of the manuscript for important intellectual content: Ayoub Moutaoakil, Wassim Beladel, Karim Hasni, Mohamed El Minaoui

Acquisition, analysis, or interpretation of data: Wassim Beladel, Karim Hasni, Mohamed El Minaoui

Supervision: Wassim Beladel, Karim Hasni, Mohamed El Minaoui

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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