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Dynamic Fluoroscopic Recognition of Chronic Subclavian Vein Occlusion During CIED Implantation: Mechanobiological Remodeling and Functional Venous Patency

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Abstract

Chronic thoracic venous obstruction is an increasingly encountered but frequently underrecognized challenge during cardiac implantable electronic device (CIED) implantation. Because progressive collateral venous development may preserve effective upper extremity drainage, severe venous obstruction can remain entirely asymptomatic and become apparent only during attempted transvenous instrumentation. We report a case of incidentally discovered chronic total left subclavian vein occlusion identified during permanent pacemaker implantation in a patient without clinical manifestations of venous obstruction. Initial left-sided venous access demonstrated successful aspiration of venous blood; however, fluoroscopic guidewire advancement revealed an abnormal superior and lateral trajectory inconsistent with normal central venous anatomy. Subsequent contrast venography confirmed chronic total left subclavian vein occlusion with extensive collateral venous circulation and delayed contralateral venous opacification. Following abandonment of the left-sided approach, contralateral right subclavian venography demonstrated an angiographically narrowed and medially tapered venous segment that initially raised concern for additional obstruction. Nevertheless, careful dynamic fluoroscopic assessment confirmed preserved functional luminal continuity, permitting successful right ventricular lead implantation without complications. This case highlights several important procedural and mechanobiological principles relevant to contemporary electrophysiology practice, including the remarkable adaptive capacity of thoracic collateral venous remodeling, the limited reliability of venous blood aspiration alone in confirming central venous patency, and the critical importance of abnormal guidewire trajectory as an early fluoroscopic marker of occult venous obstruction. Furthermore, the case emphasizes the distinction between static angiographic appearance and true functional venous traversability. Early recognition, procedural adaptability, and meticulous fluoroscopic assessment remain essential for safe and successful management of complex venous anatomy during CIED implantation.

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Introduction

Central thoracic venous obstruction involving the subclavian, axillary, or brachiocephalic venous systems represents a clinically significant challenge during implantation and revision of cardiac implantable electronic devices (CIEDs). Progressive expansion in the use of permanent pacemakers, implantable cardioverter-defibrillators, and cardiac resynchronization therapy systems has increased the frequency with which electrophysiologists encounter complex venous anatomy during transvenous procedures. Venous stenosis or chronic total occlusion may develop secondary to endothelial injury, thrombosis, fibrosis, and chronic inflammatory remodeling induced by transvenous leads or prior central venous instrumentation [1]. In addition to device leads, recognized predisposing factors include dialysis catheters, malignancy, thrombophilic disorders, trauma, prior thoracic surgery, and repeated central venous cannulation. However, clinically significant obstruction may also occur in patients without obvious predisposing history and may remain undetected for years because gradual venous narrowing allows the progressive formation of extensive collateral venous pathways that preserve effective upper extremity and thoracic venous drainage [1]. The true prevalence of asymptomatic thoracic venous obstruction is likely underestimated in routine clinical practice. Studies employing contrast venography before lead revision, extraction, or device upgrade procedures have demonstrated variable degrees of venous stenosis or complete occlusion in a substantial proportion of patients carrying transvenous systems [2]. Importantly, many affected individuals remain entirely asymptomatic despite advanced anatomical obstruction, highlighting the remarkable compensatory capacity of collateral venous circulation. Consequently, occult venous obstruction is frequently discovered only during attempted venous access at the time of device implantation or revision procedures. This creates important procedural and safety implications for device implanters, particularly when unexpected resistance, atypical guidewire behavior, or abnormal fluoroscopic guidewire trajectory is encountered during vascular access. Recognition of abnormal guidewire course remains one of the most important intraoperative clues suggesting underlying central venous obstruction. Failure to identify this finding may lead to repeated traumatic venous manipulation, vascular perforation, procedural failure, unnecessary radiation exposure, prolonged operative duration, thromboembolic complications, or potentially life-threatening consequences [3]. Early recognition permits timely venographic assessment and facilitates appropriate procedural adaptation, including alternative venous access, venoplasty, or lead extraction strategies. The present report describes an incidentally discovered chronic total left subclavian vein occlusion encountered during pacemaker implantation, characterized by extensive compensatory collateral venous circulation and successful contralateral device implantation despite angiographically complex venous anatomy.

Case Presentation

A patient was referred for implantation of a single-chamber permanent pacemaker because of symptomatic bradyarrhythmia requiring permanent pacing support. Preprocedural clinical evaluation did not reveal symptoms suggestive of central venous obstruction. There was no clinically evident upper limb edema, venous congestion, cyanosis, or collateral venous prominence over the chest wall or neck. The procedure was initiated using a conventional left infraclavicular venous access approach under fluoroscopic guidance. Venous puncture was successfully achieved with free aspiration of dark non-pulsatile venous blood. However, advancement of the guidewire demonstrated an unusual tortuous superior and lateral trajectory rather than the expected smooth progression medially toward the superior vena cava and right atrium. Repeated fluoroscopic assessment confirmed persistent abnormal wire behavior despite repositioning attempts, immediately raising suspicion of occult central venous obstruction.

To further delineate the venous anatomy, contrast venography of the left upper limb was performed, Contrast drainage occurs through collateral venous channels with delayed opacification of the contralateral right brachiocephalic venous circulation, consistent with a chronic obstructive process with compensatory collateralization. After few seconds, the right subclavian vein was opacified with blind medial end tapering (Figure 1).

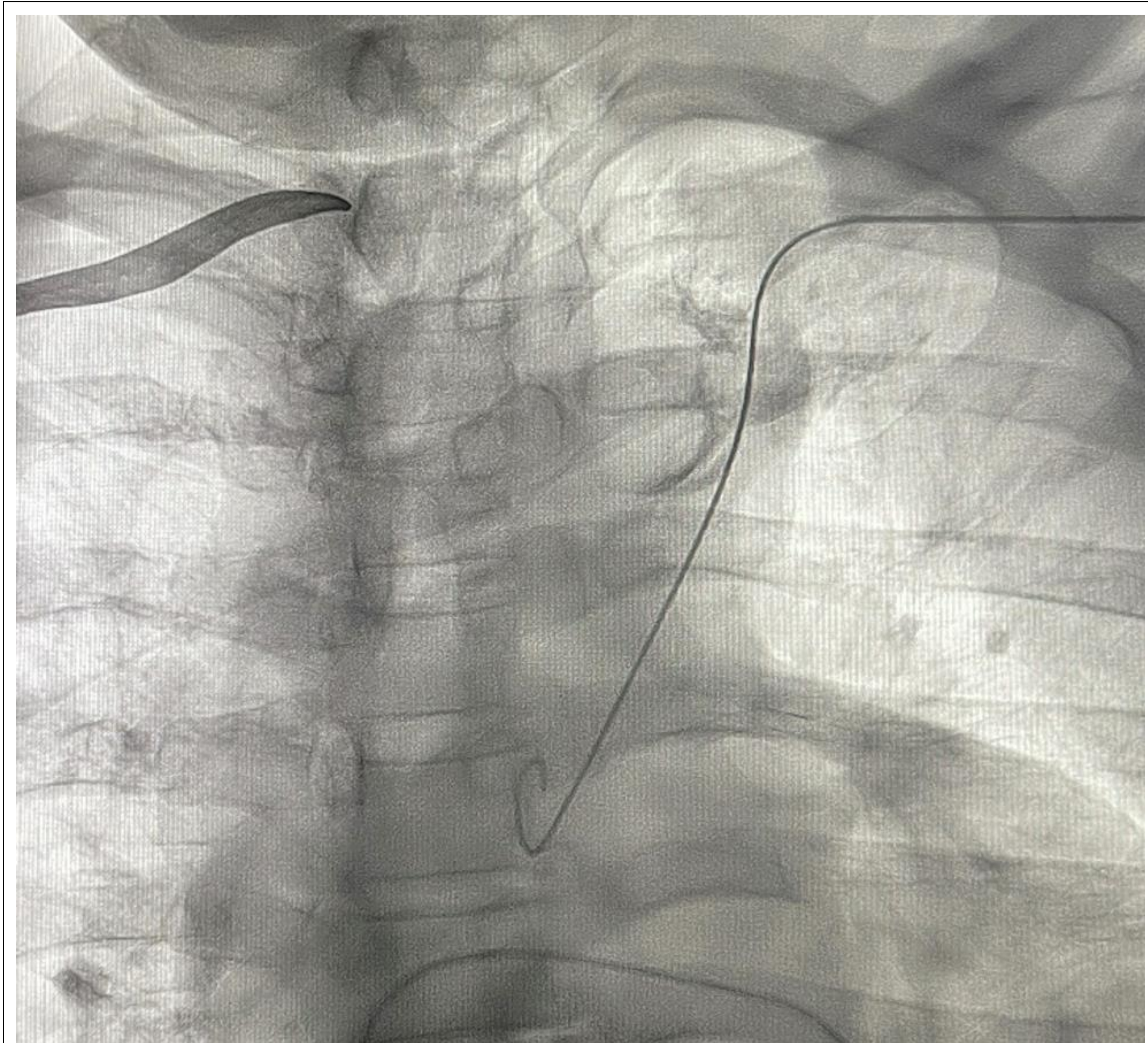

Figure 1: Left upper extremity venography demonstrating chronic total occlusion of the left subclavian vein with absence of direct opacification of the central venous lumen. The guidewire demonstrates an abnormal superior and lateral trajectory away from the expected course toward the superior vena cava, raising suspicion of central venous obstruction. Contrast drainage occurs through collateral venous channels with delayed opacification of the contralateral right brachiocephalic venous circulation, consistent with a chronic obstructive process with compensatory collateralization.

Given the complete left-sided venous occlusion, the decision was made to abandon left transvenous implantation and proceed with contralateral venous access. Right subclavian venous cannulation was subsequently attempted and achieved successfully on the first puncture. However, contrast venography revealed an unusual medially peaked and relatively narrowed appearance of the right subclavian venous segment with suboptimal visualization of a continuous central lumen (Figure 2).

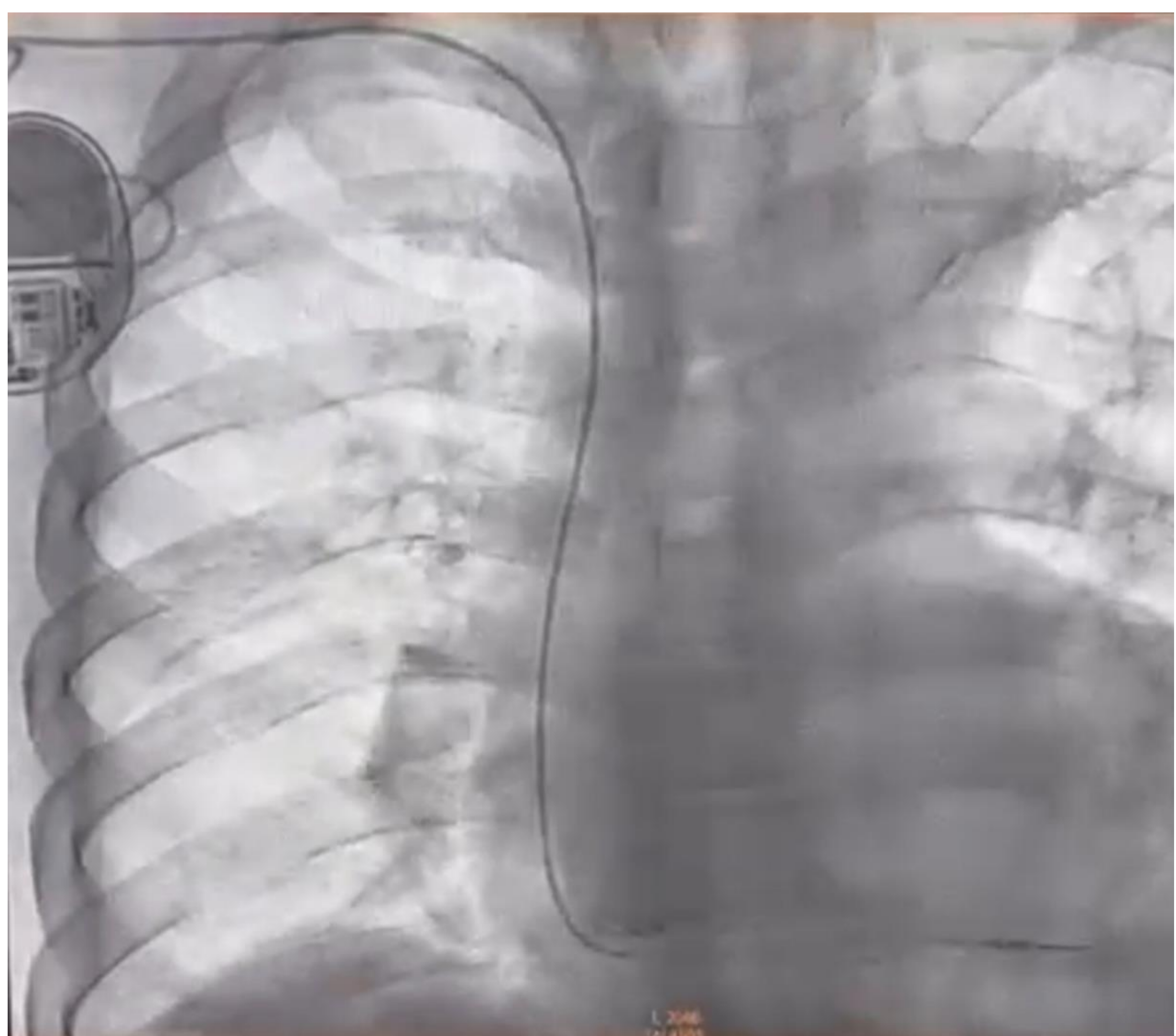

Figure 2: Right subclavian venography obtained following abandonment of the left-sided approach demonstrated a relatively narrowed venous segment with a sharp tapered medial end and limited direct visualization of the central lumen, creating initial angiographic concern for possible functional obstruction. In contrast to the complete chronic total occlusion observed on the left side in Figure 1, where the guidewire failed to progress centrally and was diverted through extensive collateral venous pathways, careful dynamic fluoroscopic assessment on the right side confirmed preserved luminal continuity. Careful meticulous guidewire manipulation allowed successful traversal into the superior vena cava and subsequent uncomplicated transvenous right ventricular lead implantation. This figure highlights the critical procedural distinction between static angiographic appearance and true functional venous patency during cardiac implantable electronic device implantation.

Despite the concerning angiographic appearance, careful fluoroscopic manipulation demonstrated successful guidewire traversal into the superior vena cava without resistance or vessel injury. Functional venous patency was therefore confirmed dynamically. A right ventricular pacing lead was subsequently advanced successfully and positioned in the right ventricle without mechanical difficulty. Electrical measurements were excellent with stable lead performance. Implant Device Parameters were as follows:

• Mode: VVI
• Lower rate limit: 65 bpm
• R-wave amplitude: 9 mV
• Capture threshold: 1.25 V
• Lead impedance: 850 Ω

The procedure was completed without immediate complications including, pneumothorax, hematoma, vascular injury, lead instability, or arrhythmia.

Discussion

Chronic thoracic venous obstruction represents one of the most important yet frequently underestimated challenges encountered during cardiac implantable electronic device (CIED) implantation and revision procedures. Although traditionally associated with prior transvenous pacing leads or central venous instrumentation, clinically silent venous occlusion may also occur in patients without obvious predisposing history, becoming apparent only during attempted venous cannulation or guidewire advancement [4,5]. The present case illustrates several critical procedural, anatomical, and pathophysiological concepts with direct relevance to modern electrophysiological practice. A particularly important observation in this patient is the complete absence of clinical manifestations despite total chronic left subclavian venous occlusion. This phenomenon reflects the remarkable adaptive capacity of the thoracic venous circulation. Progressive venous obstruction occurring over prolonged periods permits gradual recruitment and enlargement of collateral venous pathways involving the internal mammary, thoracoepigastric, vertebral, intercostal, mediastinal, azygos, hemiazygos, and cervical venous systems [6,7]. Because collateral venous development evolves progressively, venous return from the ipsilateral upper extremity may remain sufficiently preserved to prevent edema, cyanosis, venous hypertension, or visible chest wall collateralization. Consequently, severe anatomical obstruction may remain clinically occult for years. From a vascular biology perspective, chronic venous obstruction initiates a highly dynamic remodeling process characterized by endothelial activation, alterations in venous shear stress, nitric oxide pathway modulation, inflammatory cytokine signaling, extracellular matrix remodeling, and angiogenic stimulation mediated largely through vascular endothelial growth factor (VEGF) dependent pathways [8,9]. Sustained hemodynamic stress promotes collateral vessel maturation and adaptive venogenesis, ultimately generating alternative low resistance drainage circuits capable of maintaining near normal venous hemodynamics. The absence of clinical manifestations in this case strongly supports a chronic adaptive process rather than acute thrombotic occlusion. The case also highlights a critically important procedural teaching point: successful venous puncture with free aspiration of dark non-pulsatile blood does not confirm central venous patency. In patients with chronic proximal obstruction, venous access needles may cannulate collateralized distal venous segments that remain pressurized and patent despite complete central occlusion [10].

Consequently, reliance solely on blood aspiration may create false procedural reassurance. Instead, fluoroscopic guidewire behavior becomes the most sensitive early intraoperative marker of occult venous obstruction. Under normal anatomical conditions, a guidewire introduced through the subclavian or axillary venous system should progress smoothly and medially toward the superior vena cava and right atrium. In contrast, abnormal superior migration, looping, lateral diversion, cephalad angulation, resistance, or tortuous collateral tracking strongly suggests underlying obstruction or severe stenosis [11]. In the present case, immediate recognition of the abnormal guidewire trajectory prompted timely venographic assessment, thereby avoiding repeated traumatic manipulations that could have resulted in venous perforation, mediastinal injury, thrombosis, embolization, or hemothorax.

Another highly instructive aspect of this case is the discrepancy between static angiographic appearance and true functional venous patency. Right subclavian venography demonstrated a narrowed medially tapered venous segment with incomplete visualization of the central lumen, initially raising concern for additional significant obstruction. However, careful dynamic fluoroscopic guidewire manipulation demonstrated preserved luminal continuity sufficient for safe transvenous lead delivery. This distinction between anatomical irregularity and functional traversability is highly important during device implantation procedures. Static venography may occasionally overestimate the severity of venous compromise because contrast flow is influenced by injection pressure, projection angle, collateral flow competition, respiratory variation, and low-flow hemodynamics [12]. Dynamic fluoroscopic assessment during real-time guidewire advancement often provides more clinically meaningful information regarding true procedural feasibility. This principle is particularly relevant in elderly patients with chronic venous remodeling, fibrosis, calcification, or external compression where angiographic appearances may appear disproportionately severe relative to actual mechanical traversability.

The present case additionally raises important mechanobiological considerations regarding chronic venous remodeling. Long standing alterations in venous wall tension, disturbed laminar flow, turbulent hemodynamics, endothelial dysfunction, collagen deposition, intimal fibrosis, and adaptive extracellular matrix restructuring may substantially alter venographic morphology [13]. These chronic structural adaptations may create angiographic appearances suggestive of near complete obstruction while preserving sufficient residual luminal continuity for guidewire and lead passage. Such observations reinforce the importance of integrating anatomical imaging with dynamic procedural physiology rather than relying exclusively on static contrast appearance.

From an interventional electrophysiology standpoint, early procedural adaptability remains essential when occult venous obstruction is encountered unexpectedly. Available management strategies depend on operator expertise, patient characteristics, venous anatomy, device indication, and institutional resources. Potential approaches include contralateral implantation, balloon venoplasty, blunt or radiofrequency assisted recanalization, lead extraction with venous re-entry, femoral or iliac access routes, surgical epicardial lead implantation, or use of leadless pacing systems [14-17]. In the current patient, contralateral implantation represented the safest and most efficient strategy because preserved functional right-sided venous continuity was confirmed fluoroscopically.

The increasing prevalence of CIED implantation worldwide, combined with aging populations and rising rates of lead revision and system upgrade procedures, suggests that electrophysiologists will encounter venous obstruction with increasing frequency [18]. Routine awareness of abnormal guidewire behavior, early venographic assessment, and preservation of procedural flexibility are therefore essential competencies in contemporary device implantation practice, and fellowship programs.

Finally, this case reinforces the value of meticulous fluoroscopic observation during even apparently routine procedures. The principal educational value of this report lies not in the presence of venous occlusion itself, but in the dynamic fluoroscopic interpretation of guidewire behavior and the procedural distinction between angiographic morphology and true functional venous traversability. The earliest abnormality in this patient was not angiographic but kinetic, namely the atypical guidewire trajectory. In many procedural settings, fluoroscopic wire dynamics may reveal clinically important pathology before contrast venography is performed. Recognition of these subtle procedural warning signs may significantly reduce complications, operative duration, radiation exposure, and procedural failure (Figure 3).

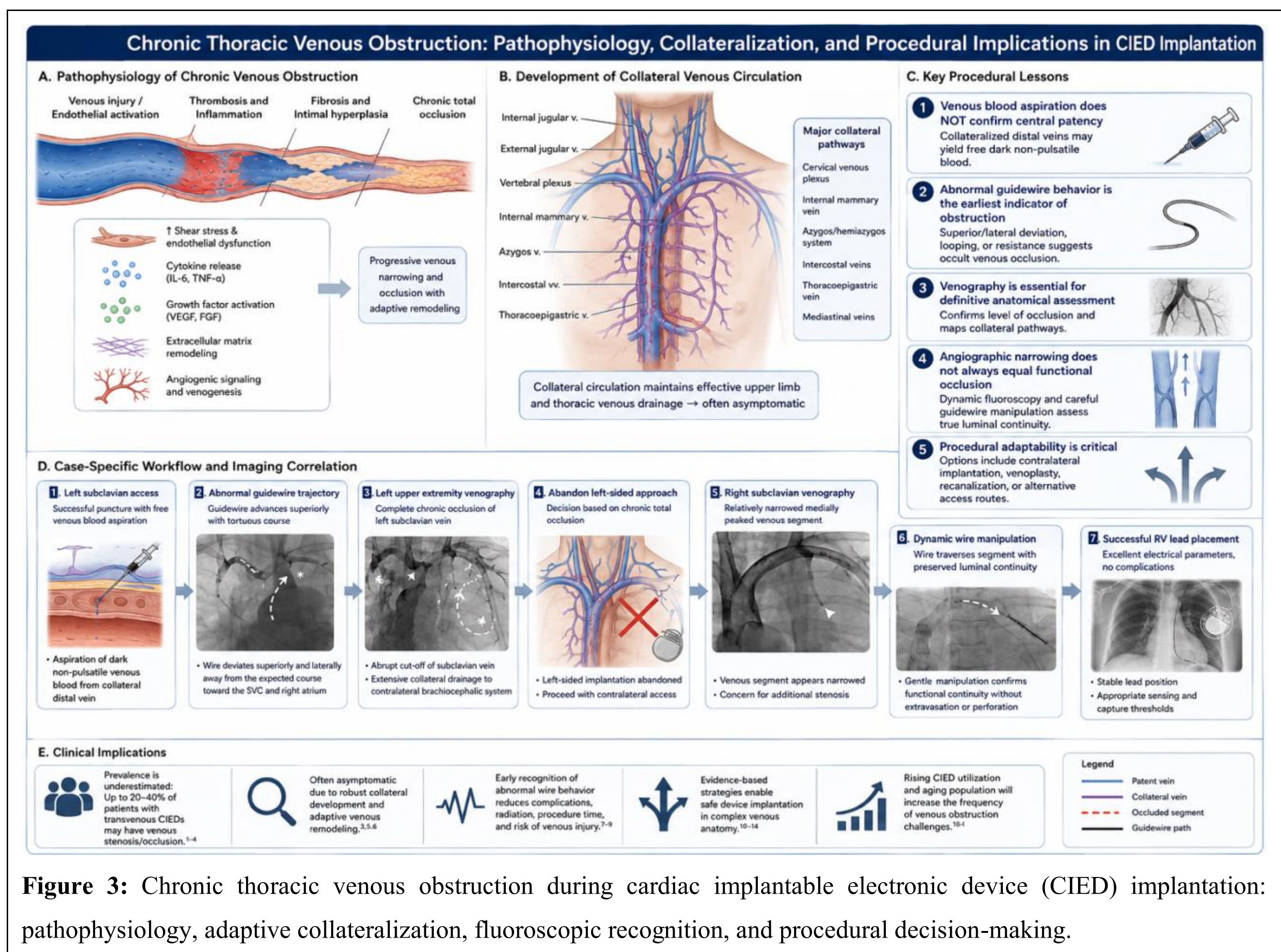

Figure 3: Chronic thoracic venous obstruction during cardiac implantable electronic device (CIED) implantation: pathophysiology, adaptive collateralization, fluoroscopic recognition, and procedural decision-making.

This integrative schematic illustrates the mechanistic and procedural continuum observed in the present case of incidentally discovered chronic left subclavian vein occlusion during permanent pacemaker implantation. Panel A demonstrates the progressive vascular biological cascade leading to chronic venous obstruction, beginning with endothelial injury, thrombosis, inflammatory activation, intimal hyperplasia, extracellular matrix remodeling, fibrosis, and eventual chronic total venous occlusion. Sustained alterations in venous shear stress and endothelial signaling stimulate angiogenic and venogenic pathways, including vascular endothelial growth factor (VEGF)-mediated collateral vessel formation. Panel B illustrates the extensive compensatory collateral venous circulation involving the internal mammary, vertebral, azygos, intercostal, thoracoepigastric, mediastinal, and cervical venous systems, explaining the absence of overt clinical manifestations despite complete anatomical obstruction. Panel C summarizes the principal procedural lessons highlighted by this case, emphasizing that free venous blood aspiration does not confirm central venous patency, abnormal guidewire trajectory represents an early fluoroscopic marker of occult obstruction, and dynamic fluoroscopic wire behavior may provide greater functional information than static venographic appearance alone. Panel D reconstructs the procedural workflow of the present case, including successful left venous puncture with abnormal guidewire deviation, diagnostic venographic confirmation of chronic left subclavian vein occlusion with extensive collateralization, abandonment of the left-sided approach, subsequent right subclavian late phase venography demonstrating a medially tapered venous segment, and eventual successful right ventricular lead implantation following dynamic confirmation of preserved luminal continuity. Panel E highlights the broader clinical implications for contemporary electrophysiology practice, including the increasing prevalence of occult venous obstruction in aging CIED populations, the critical importance of early fluoroscopic recognition, and the necessity for procedural adaptability using alternative venous access strategies when conventional transvenous pathways are compromised.

Collectively, the present case and the integrative mechanistic framework illustrated in Figure 3 emphasize that chronic thoracic venous obstruction should not be viewed merely as an anatomical obstacle, but rather as a dynamic biological and procedural continuum involving vascular remodeling, collateral adaptation, altered hemodynamics, and real-time fluoroscopic physiology. The case further demonstrates that subtle kinetic procedural observations, particularly abnormal guidewire trajectory, may provide earlier and more clinically meaningful diagnostic information than static anatomical imaging alone. As CIED implantation rates continue to rise globally, increasing recognition of these fluoroscopic warning patterns and adaptive venous phenomena will become progressively more important for reducing procedural complications, improving implantation success, and enhancing operator decision-making during complex transvenous interventions [19,20].

Learning Objectives

1. To identify abnormal fluoroscopic guidewire trajectory as an early and highly sensitive procedural marker of occult central thoracic venous obstruction during cardiac implantable electronic device (CIED) implantation.

2. To recognize that successful venous puncture with free aspiration of non-pulsatile venous blood does not necessarily confirm functional central venous patency in the presence of chronic proximal venous occlusion.

3. To understand the adaptive vascular biology and collateral venous remodeling mechanisms that may allow chronic total subclavian vein occlusion to remain entirely clinically silent for prolonged periods.

4. To differentiate between static angiographic venous narrowing and true functional luminal traversability through dynamic fluoroscopic guidewire assessment during transvenous device implantation procedures.

5. To appreciate the importance of integrating anatomical imaging, procedural fluoroscopic kinetics, and real-time physiological assessment when managing complex thoracic venous anatomy.
6. To understand the major procedural strategies available for management of unexpected thoracic venous obstruction, including contralateral implantation, venoplasty, alternative venous access routes, lead extraction techniques, and leadless pacing systems.
7. To reinforce the importance of procedural adaptability, meticulous fluoroscopic observation, and early venographic assessment in minimizing complications and improving procedural success during contemporary electrophysiology practice.

Conclusion

This case highlights the critical importance of recognizing occult thoracic venous obstruction during cardiac implantable electronic device implantation, even in patients without any clinical manifestations of venous disease. Chronic total subclavian vein occlusion may remain entirely silent because of extensive adaptive collateral venous remodeling capable of preserving effective upper extremity drainage over prolonged periods. The present report further demonstrates that successful venous puncture with free blood aspiration does not guarantee functional central venous patency, whereas abnormal fluoroscopic guidewire behavior may serve as the earliest and most sensitive intraoperative marker of occult obstruction. Importantly, static venographic appearance alone may overestimate the severity of venous compromise, and dynamic fluoroscopic assessment of guidewire traversability frequently provides more clinically relevant procedural information. Early recognition of abnormal venous anatomy, prompt venographic clarification, and procedural adaptability remain essential for minimizing complications and achieving successful device implantation in increasingly complex contemporary electrophysiology practice.

Disclosures

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REFERENCES

1. Oginosawa Y, Abe H, Nakashima Y, et al. The incidence and risk factors for venous obstruction after implantation of transvenous pacing leads. Pacing Clin Electrophysiol. 2002; 25: 1605-1611.
2. Lickfett L, Bitzen A, Arepally A, et al. Incidence of venous obstruction following insertion of an implantable cardioverter-defibrillator. J Am Coll Cardiol. 2004; 43: 113-117.
3. Worley SJ. How to use balloons to facilitate lead implant or revision in patients with central venous obstruction. Heart Rhythm. 2009; 6: 852-856.
4. Lickfett L, Bitzen A, Arepally A, et al. Incidence of venous obstruction following insertion of an implantable cardioverter-defibrillator. J Am Coll Cardiol. 2004; 43: 113-117.
5. Oginosawa Y, Abe H, Nakashima Y, et al. The incidence and risk factors for venous obstruction after implantation of transvenous pacing leads. Pacing Clin Electrophysiol. 2002; 25: 1605-1611.
6. Bracke FA, Meijer A, Van Gelder LM. Venous occlusion of the access vein in patients referred for lead extraction. Pacing Clin Electrophysiol. 2003; 26: 1649-1652.
7. Rozmus G, Daubert JP, Huang DT, et al. Venous thrombosis and stenosis after implantation of pacemakers and defibrillators. J Interv Card Electrophysiol. 2005; 13: 9-19.
8. Carmeliet P. Angiogenesis in health and disease. Nat Med. 2003; 9: 653-660.

9. Eyries M, Collins T, Khachigian LM. Modulation of growth factor gene expression in vascular remodeling. Circ Res. 2004; 95: 329-342.
10. Worley SJ. How to obtain venous access in patients with central venous obstruction undergoing cardiac device implantation. Heart Rhythm. 2009; 6: 852-857.
11. Burri H, Sunthorn H, Dorsaz PA, et al. Venous obstruction and collateral circulation after pacemaker implantation. Europace. 2007; 9: 455-459.
12. Chan AW, Bhatt DL, Wilkoff BL. Percutaneous treatment for pacemaker-associated subclavian vein stenosis. Am Heart J. 2003; 146: 260-266.
13. Lee AYY, Kamphuisen PW, Meyer G, et al. Trousseau syndrome and mechanisms of venous remodeling. Blood. 2012; 120: 4062-4067.
14. Worley SJ. Venoplasty during pacemaker or ICD implantation. Heart Rhythm. 2008; 5: 526-533.
15. Hussein AA, Wilkoff BL, Martin DO, et al. Initial experience with the leadless cardiac pacemaker. N Engl J Med. 2015; 373: 1125-1135.
16. Wilkoff BL, Love CJ, Byrd CL, et al. Transvenous lead extraction: Heart Rhythm Society expert consensus. Heart Rhythm. 2009; 6: 1085-1104.
17. Bongiorni MG, Burri H, Deharo JC, et al. 2018 EHRA expert consensus statement on lead extraction. Europace. 2018; 20: 1217.
18. Greenspon AJ, Patel JD, Lau E, et al. Trends in permanent pacemaker implantation in the United States. J Am Coll Cardiol. 2012; 60: 1540-1545.
19. Boyle TA, et al. Lead Implantation in the Setting of Venous Occlusion. JACC Clinical Electrophysiology. 2025.
20. Menemencioglu C, Canpolat U, Ates AH, et al. Venoplasty-facilitated cardiac implantable electronic device implantation and upgrade: feasibility and outcomes from a single center. J Interv Card Electrophysiol. 2026.