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Localized left brachiocephalic vein obstruction during the respiration cycle as a potential trigger of thrombotic complications of cardiac implantable electronic device and central venous catheter placement procedures

Lokalne zaburzenie drożności lewej żyły ramienno-głowowej podczas cyklu oddechowego przez potencjalny czynnik inicjacji zmian pozakrzepowych po zabiegach implantacji wszczepialnego urządzenia do elektroterapii serca czy wprowadzeniu cewnika do żyły centralnej

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Abstract

Introduction. The left brachiocephalic vein (LBCV) courses between the sternum and the aortic arch (or its main branches). Anomalous spatial positioning of the LBCV and these adjacent anatomical structures may result in LBCV compression. Such circumstances result in a narrowing or complete occlusion of this vein, which may affect the course of intravascular procedures, such as cardiac implantable electronic device (CIED) or central venous catheter (CVC) placement. This increases the risk of venous injury and/or the subsequent occlusion due to thrombus formation.

Material and methods. Out of 2,112 *de novo* CIED implantation procedures performed in the period 2014–2019, 391 cases required intraoperative venography to identify the cause of difficulties in endovascular lead advancement. Venography data from these cases were analysed and only those cases were included for further analysis where the cause of difficulties was shown to be LBCV compression, further exacerbated on expiration.

Results. Severe localized LBCV compression affecting the course of CIED implantation procedures was detected in 12 patients (8 women and 4 men, aged 80 ± 7 years). Such localized venous compression was observed mainly at the level where the brachiocephalic trunk and the left common carotid artery branch of the aortic arch.

Conclusions. The analysed data showed the phenomenon of localized, respiration-related LBCV compression, which became exacerbated on expiration, in 3% of the analysed de novo CIED implantation procedures. This phenomenon may be associated with increased long-term thrombotic venous occlusion in the vascular segments containing cardiac leads or CVCs.

Key words: left brachiocephalic vein, venography, venous compression, occlusion, stenosis, CIED, CVC

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Introduction

The number of minimally invasive intravascular procedures, such as cardiac implantable electronic device (CIED) or central venous catheter (CVC) placement has been steadily increasing [1]. The convenient anatomical position of the veins in clavipectoral triangles has led to their use for pacemaker lead insertion or angiocatheter insertion. In a left-sided approach, the vein used for lead advancement through the middle mediastinum is the left brachiocephalic vein (BCV), which connects the left subclavian vein (SV) with the superior vena cava (SVC).

A favourable spatial relationship of the LBCV with the sternum and with the aortic arch [or its branches: the brachiocephalic trunk (BCT), left common carotid artery (CCA), and left subclavian artery (SA)] facilitates uneventful CIED or CVC placement [2–4].

The position of the LBCV with respect to the adjacent anatomical structures can be visualized via targeted three-dimensional (3D) computed tomography (Figure 1). However, intraoperative visualization of LBCV position during CIED implantation procedures is still achieved largely via classic venography, whose two-dimensional views limit the scope of obtainable data regarding the shape and course of the LBCV. Nonetheless, unexpected vascular morphometry alterations detected during an intravascular procedure require that the cause be determined promptly. Severe venous obstruction may impede or prevent lead advancement through the affected vascular segment [5–8].

A local LBCV deformity due to the vessel's compression by adjacent anatomical structures, but without a complete obstruction, may be asymptomatic (example in Figure 1E, F) [3, 9]. The gradual development of collateral circulation compensates for chronic progressive blood flow obstruction within systemic veins and delays clinical manifestations. In such cases, a narrowing of the LBCV is typically an accidental finding during diagnostic computed tomography (CT), magnetic resonance imaging, or ultrasonography examinations performed in patients with mediastinal lymphadenopathy, advanced goitre, or neurological problems [10–13].

Somewhat less important scenarios — nonetheless also potentially contributing to LBCV compression and the resulting mechanical and haemodynamic obstruction — are aortic arch enlargement, the expiratory phase of a physiological respiratory cycle, supine position, and advanced age. These may be amplified by excessive proximity of the LBCV to the sternum, which can be particularly salient at those sites along the LBCV course through the mediastinum that are particularly vulnerable to compression due to their proximity to the aortic arch, left CCA, or left SA (Figure 1E, F) [3, 8, 9].

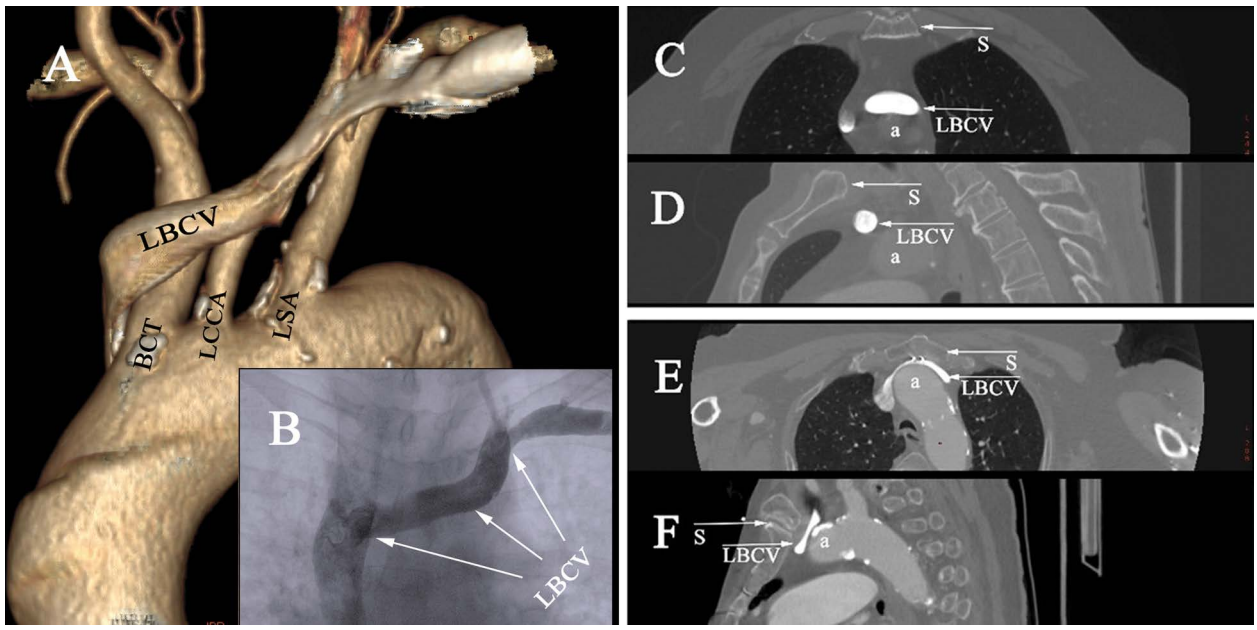

Figure 1. Radiographic images illustrating left brachiocephalic vein (LBCV) topography and morphometry: **A.** Computed tomography: a three-dimensional (3D) view of the LBCV and aortic arch branches; **B.** Intraoperative venography [posterior-anterior (PA) view]: the flow of contrast shows the LBCV lumen; **C–F.** Position of the LBCV with respect to the sternum and aortic arch; **C, D.** Normal spatial arrangement of these anatomical structures in a 66-year-old man; axial section (**C**), sagittal section (**D**); **E, F.** Venous compression by adjacent anatomical structures in a 90-year-old woman, axial section (**E**), sagittal section (**F**); a — aorta; BCT — brachiocephalic trunk; LBCV — left brachiocephalic vein; LCCA — left common carotid artery; LSA — left subclavian artery; S — *manubrium sterni*

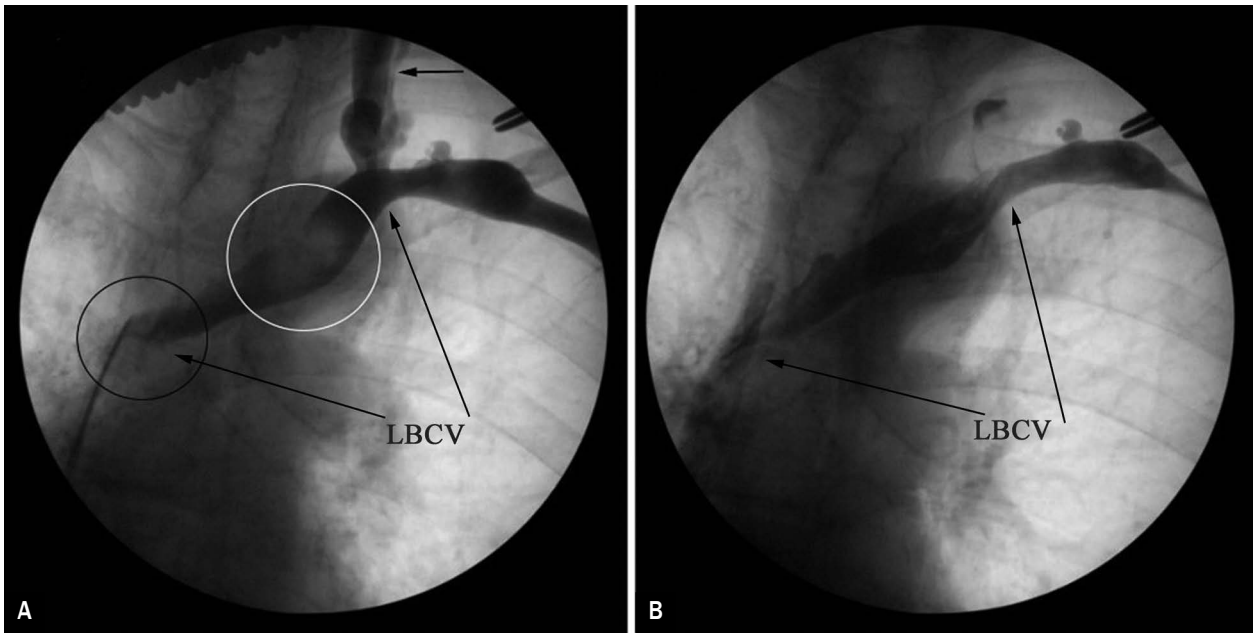

Figure 2. VVI pacemaker implantation in an 85-year-old woman: **A.** Expiratory phase: left brachiocephalic vein (BCV) compression and contrast flow obstruction in two places: at the site where the left common carotid artery branches off the aortic arch (white oval) and at the site where the LBCV drains into the superior vena cava (black oval). Retrograde flow of contrast into the left internal jugular vein (IJV) (arrow); **B.** Inspiratory phase: a normal flow of contrast through both of the sites mentioned above and no retrograde contrast flow into the left IJV

Local anatomical abnormalities that are undetected before CIED implantation or CVC procedure may lead to venous wall injury, including its perforation, caused by attempts to force the lead or catheter through the unexpected narrowing [14–17]. Damaged venous endothelium may contribute to further LBCV narrowing, and even a complete occlusion, as it promotes local inflammatory and thrombotic responses. Such consequences may also result from chronic venous wall irritation at the narrowed site by the indwelling leads or catheters [18–20].

Due to the paucity of available literature reports on LBCV occlusion by adjacent arteries that is exacerbated during the expiratory phase, the purpose of this paper was to present severe cases of this phenomenon encountered during CIED implantation procedures.

Material and methods

The authors performed a retrospective analysis of venography images obtained during de novo CIED implantation procedures performed at the centre in the period 2014–2019. They assessed the presence and character of LBCV lumen alterations detected during intraoperative venographies in cases of problematic intravenous lead advancement within the superior mediastinum.

All analysed images had been obtained during procedures conducted by the same team of operators. This factor ensured a high homogeneity of both the employed diagnostic imaging techniques and data interpretation criteria. Posteroanterior cine imaging (pulse sequences at 12 frames per second) was conducted with the patient in a supine position and the workstation set to cardiac imaging.

The contrast agent was administered either through a peripheral vein in the arm or, selectively, via cephalic vein (CV) cutdown or axillary or subclavian vein (AV/SV) puncture. One advantage of selective venography was achieving clearer images of the LBCV while using less contrast agent.

Those cases were analysed, where the intraoperatively recorded fluoroscopic images showed altered LBCV contours and/or uneven intraluminal contrast intensity. Two frames were analysed from each venography during which a complete respiratory cycle had been captured during contrast administration. These frames were selected from the continuous fluoroscopy recording to show the LBCV lumen during the inspiratory and expiratory phases.

Results

Out of all 2,112 procedures of de novo CIED implantation performed over the evaluated six-year period (2014–2019), the authors analysed 391 procedures that had required venography of the vessels used for cardiac lead advancement.

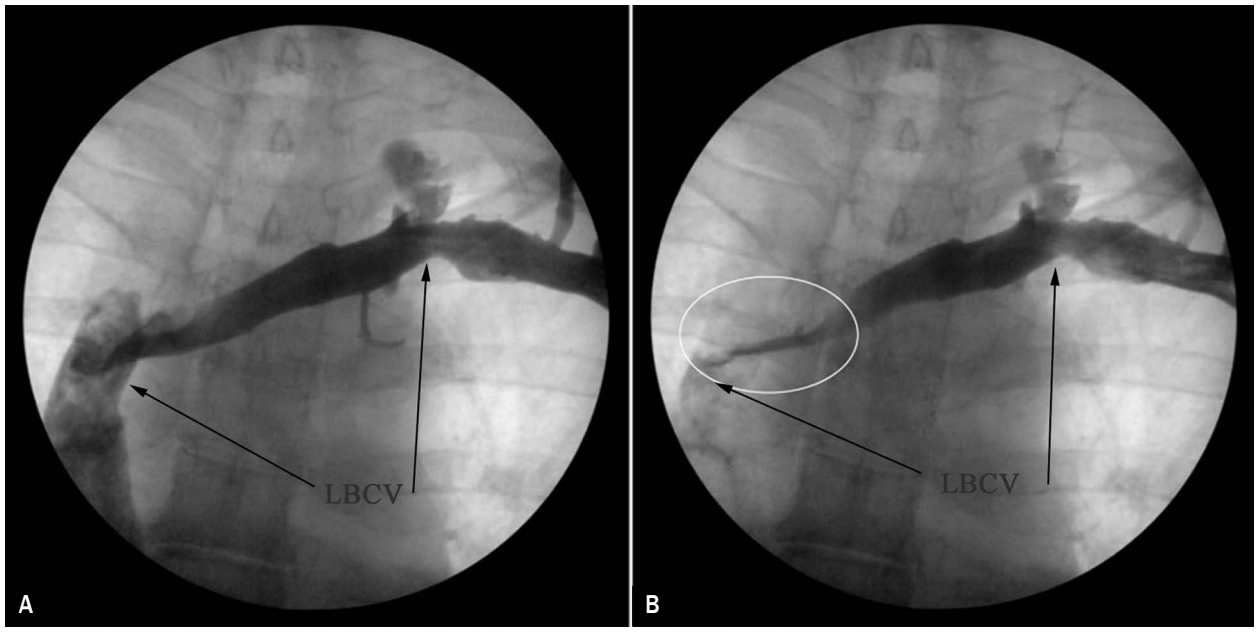

Figure 3. DDD pacemaker implantation in a 69-year-old woman; venous lumen obstruction: **A.** A slight compression of the left brachiocephalic vein by the brachiocephalic trunk; **B.** Expiratory phase shows a more pronounced reduction in the flow of contrast in this venous segment (oval)

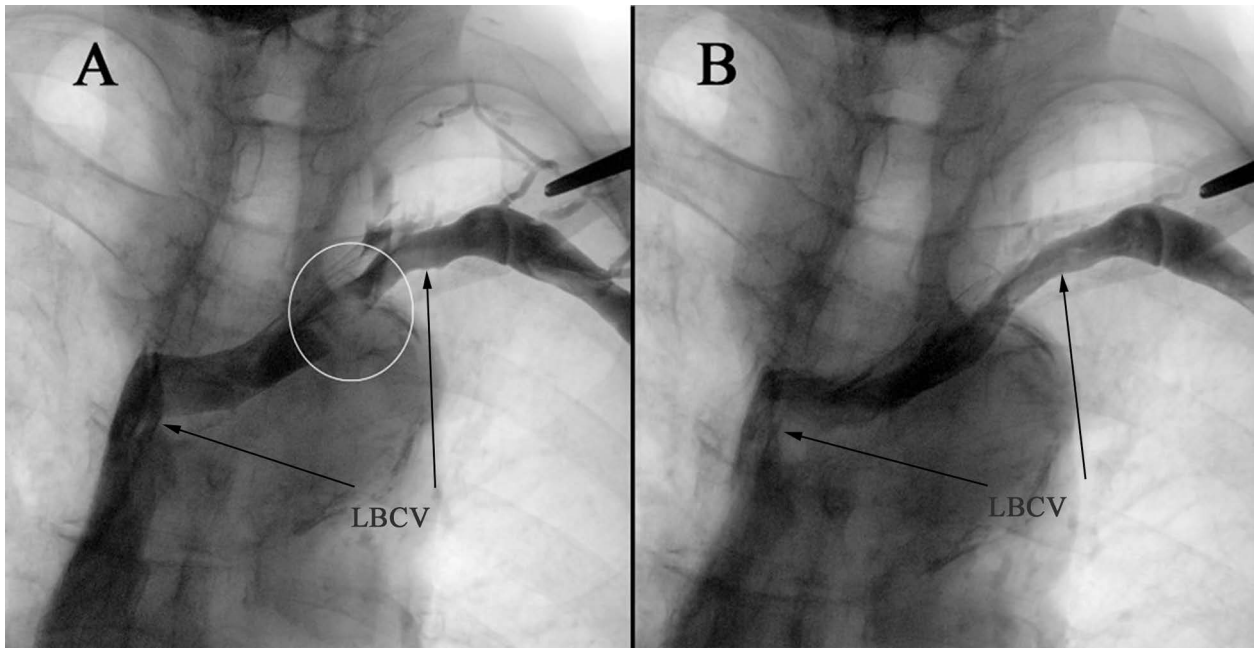

Figure 4. VVI pacemaker implantation in a 94-year-old man: **A.** Expiratory phase shows localized compression of the left brachiocephalic vein at the site where the left common carotid artery branches off the aortic arch (white oval); **B.** Uniform width of the venous lumen seen on inspiration

This subset of analysed procedures was ultimately further pared down to the images obtained from 12 patients (aged 80 ± 7 years), including 8 women and 4 men, in whom the captured venography images showed a considerable exacerbation of local LBCV compression during the expiratory phase.

This phenomenon, which was of key interest in the present analysis and was unequivocally documented by

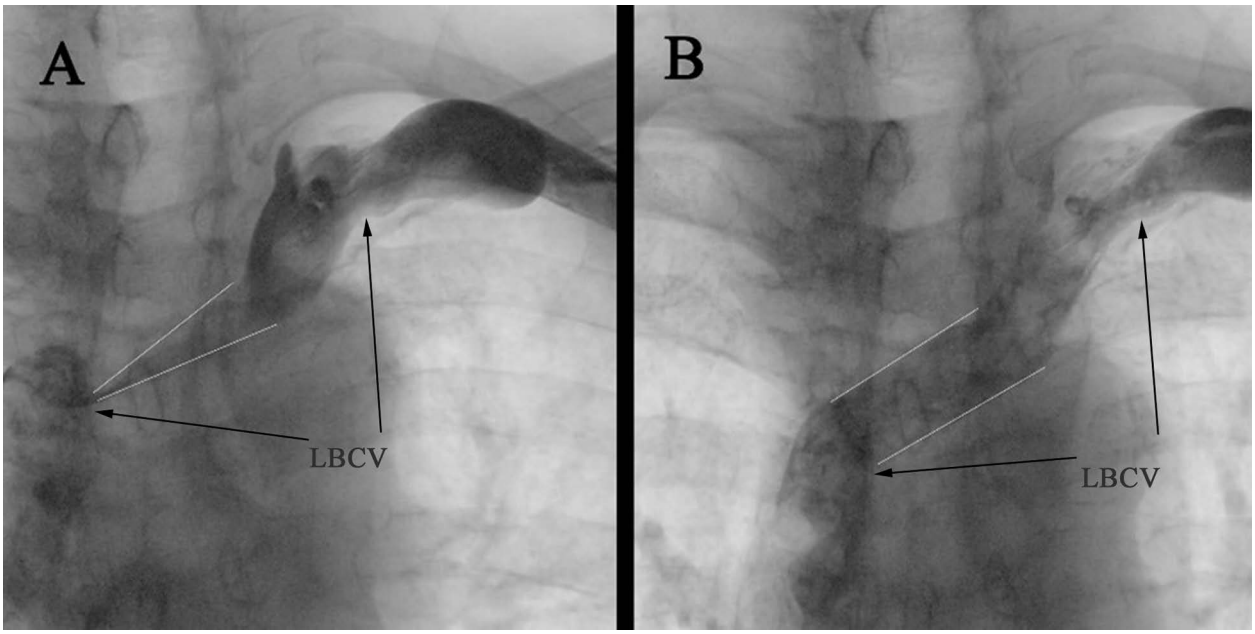

Figure 5. DDD pacemaker implantation in a 76-year-old woman: **A.** Image captured during the expiratory phase suggests a complete left brachiocephalic vein occlusion (white line) at the site where the brachiocephalic trunk branches of the aortic arch; **B.** Despite a considerable venous narrowing, the inspiratory phase shows a narrow stream of contrast at this site. Note the retrograde flow of contrast into the left internal jugular vein and thyroid veins (oval)

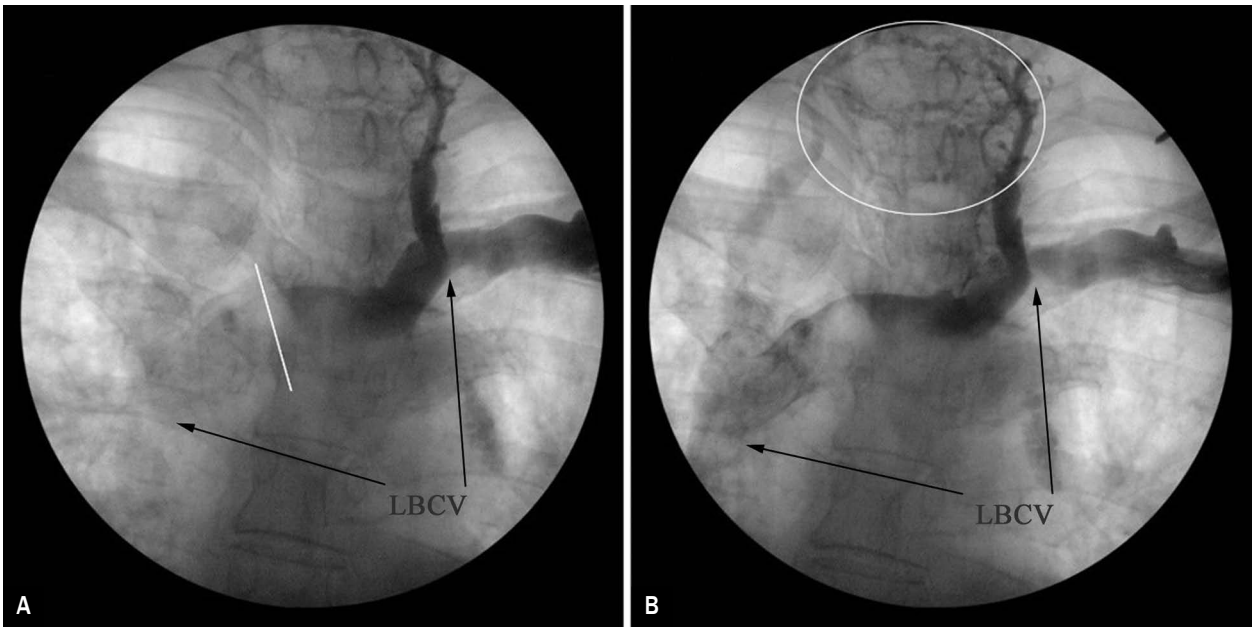

Figure 6. Single-chamber (VVI mode) pacemaker implantation in an 80-year-old man: **A.** Compression of the left brachiocephalic vein as it drains into the superior vena cava, visible at the site where the brachiocephalic trunk branches of the aortic arch, narrowing the venous lumen (thin white lines); **B.** Venous compression resolves on inspiration and the venous lumen returns to normal (thin white lines)

alterations in intraluminal contrast intensity depending on respiratory phases and exacerbated on expiration, was detected at the site where the BCT branches off the aortic arch (6 cases), the left CCA branches off the aortic arch (5 cases), and at the aortic arch itself (1 case).

These ultimately selected cases illustrated diverse situations:

— focal contrast attenuation located centrally within the lumen of the contrasted vessel (example: Figure 2);

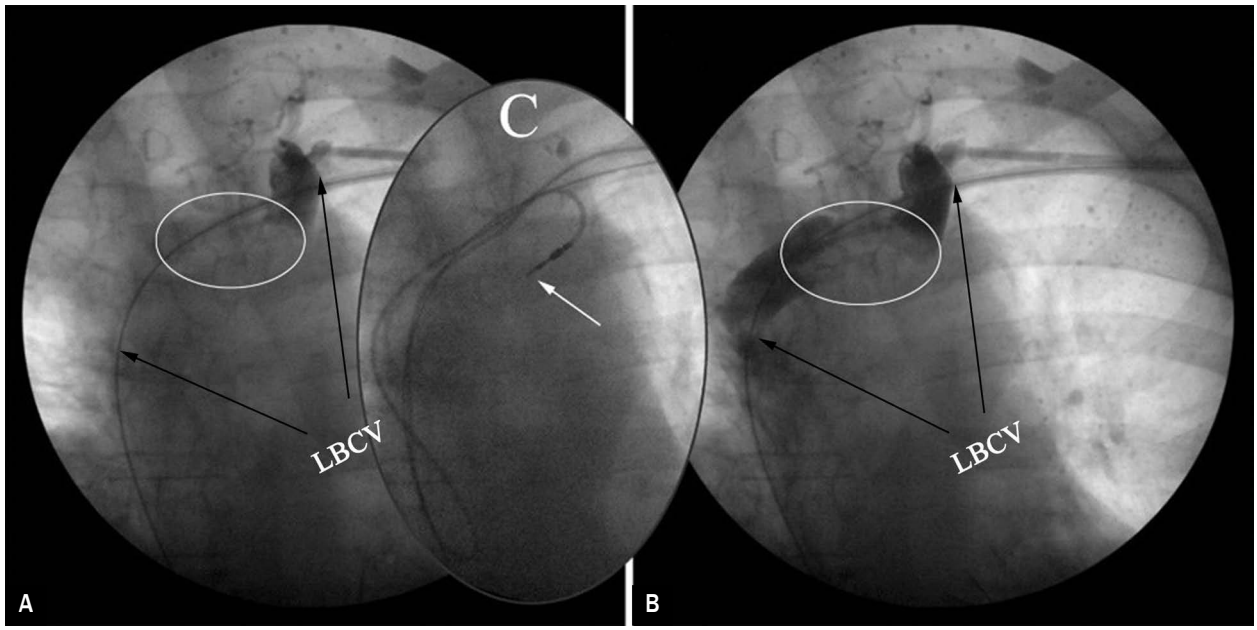

Figure 7. DDD pacemaker implantation in a 73-year-old man: **A.** Localized compression of the left brachiocephalic vein at the level where the left common carotid artery branches off the aortic arch (oval); **B.** Venous patency partially restored on inspiration (oval); **C.** Lead tip caught at the site where the venous lumen had been narrowed by compression (arrow)

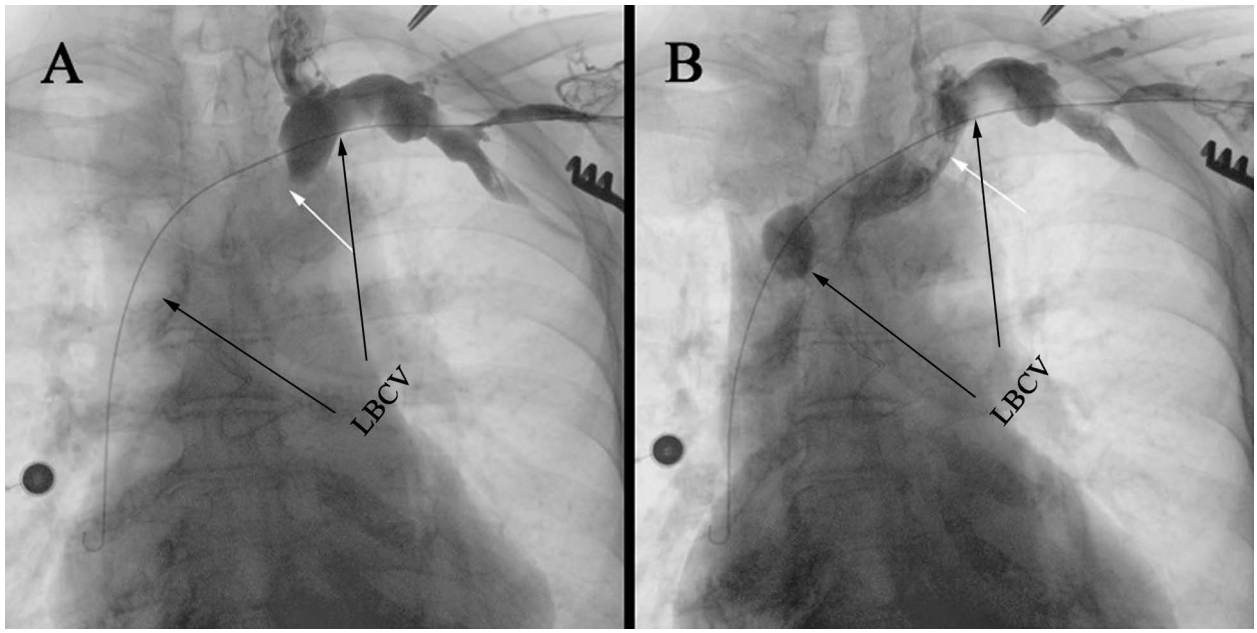

Figure 8. DDD pacemaker implantation in an 84-year-old woman: **A.** Expiratory phase image showing seemingly complete occlusion of the left brachiocephalic vein at the site of lead advancement problems where the left subclavian artery branches off the aortic arch (arrow); **B.** Inspiratory phase: local venous compression is reduced, with downstream segments of the lumen visualized by contrast.

— one-sided narrowing at the margin of the venous lumen (examples: Figures 3–5);
— complete compression of the vascular lumen (examples: Figures 6–8);

In one case, venous compression was detected in two separate sites along the LBCV (Figure 2), with the remaining images showing a single site of venous narrowing.

Despite difficulties with lead advancement, ultimately, implantation of the indicated CIED was successfully achieved in all the cases presented here.

Discussion

During normal human embryonic development, the two brachiocephalic veins form at approximately 8 weeks of gestation via an anastomosis between the left and right anterior cardinal veins. Due to asymmetry in their formation, the LBCV is ultimately nearly three times longer than the right brachiocephalic vein. The LBCV is typically positioned above the aortic arch and in front of its branches (BCT, left CCA, and left SA). Having crossed over to the right side of the superior mediastinum, the LBCV joins the right brachiocephalic vein forming the SVC [21].

The predominant approach for cardiac lead insertion during de novo CIED implantation procedures currently involves the use of one of the veins found in the left clavipectoral triangle; this approach was used during 97% of the procedures in this study. Irrespective of the specific point of access (CV cutdown or axillary/subclavian puncture), cardiac leads are subsequently advanced via the LBCV.

Along the LBCV's transverse path behind the sternum, the posterior wall of the vessel is directly adjacent to the arteries branching off the aortic arch. The physiological stability of the LBCV lumen and the vessel's haemodynamic parameters are determined by an appropriate spatial arrangement of mediastinal structures [9].

During the inspiratory phase of respiration, the space separating individual anatomical structures along with the sagittal axis increases. The reverse occurs on expiration, leading to compression and partial or complete venous lumen occlusion, as visualized in the cases presented here.

If the space separating the LBCV and sternum in a given individual is particularly narrow, the LBCV may become compressed by the adjacent arteries, particularly at the sites of direct contact between their walls [4, 9]. In such situations, localized LBCV compression may occur both during physiological respiratory movements of the chest and during active movements associated with everyday activities.

In the data evaluated as part of the present study, the phenomenon of LBCV compression was observed in patients of advanced age, with severe LBCV compression detected at the sites of the vessel's contact with the BCT and/or left CCA. A retrospective analysis of 100 CT images by Mitsuoka et al. demonstrated analogous locations of the points of direct contact between the LBCV and the adjacent arteries (the BCT in 97 cases, the left CCA in 90 cases, and the left SA several times less commonly) [3].

By altering the venous lumen, LBCV compression may increase the risk of venous wall injury during pacemaker lead insertion or angiocatheter insertion [7]. Endothelial injury due to intravenous lead manoeuvring may produce localized inflammatory and thrombotic reactions, which may contribute to subsequent venous occlusion [18, 19, 22].

Local blood flow obstruction caused by severe LBCV narrowing due to the vessel's compression may eventually lead to the formation of collateral circulation (Figure 5) [10, 11]. Haemodynamic parameters may be worsened further by the expiratory phase of respiration (which is physiologically longer than the inspiratory phase), recumbent position, and the asymptomatic nature of this phenomenon (which may remain undetected for many years). The cases presented in this paper had also been clinically asymptomatic.

Most of the studies addressing problems in cardiac lead advancement during CIED implantation procedures have focused on thrombosis-related venous occlusion [20, 23]. Decidedly less often, the cause of problems has been identified as a segmental vasoconstrictive response of the CV, AV, or SV [24, 25], and other causes of a narrowed LBCV lumen have only been reported sporadically [6, 13].

It is believed that detection of a narrowed LBCV lumen requires monitoring for any evidence of local thrombus formation and a re-evaluation of the vein's patency in case of any subsequent intravascular procedures requiring the use of this vein [4].

This paper is one of the very few that discuss the changes in radiographic parameters of the LBCV during the expiratory phase in patients with clinically asymptomatic severe LBCV compression by aortic arch branches.

Limitations

The rates of LBCV compression reported in this paper have been observed during de novo CIED implantation procedures with the use of intraoperative venography, which may not reflect the actual prevalence of this phenomenon in the general population.

Conclusions

The phenomenon of LBCV compression aggravated by expiration was observed in 3% of the group of patients undergoing de novo CIED implantation and venography and was mainly observed at the sites of the LBCV contact with the BCT and left CCA.

This phenomenon may be associated with increased long-term thrombotic venous occlusion in the vascular segments containing cardiac leads or CVCs. If the need to perform another CIED implantation or CVC placement procedure in these patients arises in the future, a pre-procedure assessment of LBCV patency seems advisable.

Streszczenie

Wstęp. Lewa żyła ramienno-głowowa (LBCV) topograficznie przebiega między mostkiem a łukiem aorty oraz poprzecznie w stosunku do odchodzących od niego tętnic. Zmiana prawidłowych przestrzennych relacji między LBCV a przyległymi do niej strukturami anatomicznymi może skutkować między innymi jej kompresją. Natomiast zniekształcenie prawidłowej morfometrii światła żyły prowadzące do jej zwężenia lub zamknięcia może wpłynąć na przebieg inwazyjnych procedur, takich jak implantacja wszczepialnego urządzenia do elektroterapii serca (CIED) czy wprowadzenie cewnika do żyły centralnej (CVC), sprzyjając doraźnej traumatyzacji żyły i/lub następowemu pozakrzepowemu dalszemu zaburzeniu jej drożności.

Materiał i metody. Spośród zrealizowanych 2112 procedur implantacji CIED *de novo* (2014–2019) ocenie poddano wenografie 391 osób wykonane w celu wyjaśnienia mechanizmu utrudnionego przemieszczania elektrod układem żylnym. Do analizy włączono tylko przypadki wizualizujące jako przyczynę supresję światła LBCV ulegającą dalszemu pogłębieniu w fazie wydechu cyklu oddechowego.

Wyniki. W badanym materiale zaawansowana miejscowa kompresja światła LBCV wpływająca na przebieg CIED ujawniła się u 12 osób (80 ± 7 lat) — 8 kobiet i 4 mężczyzn. Występowanie lokalnej supresji żyły o powyższym charakterze obserwowano głównie w miejscach odejścia od łuku aorty pnia ramienno-głowowego i lewej tętnicy szyjnej wspólnej.

Wnioski. W badanym materiale zjawisko lokalnej supresji oddechowej światła LBCV pogłębiającej się w fazie wydechu ujawniono w 3% procedur implantacji CIED *de novo*. Powyższe sytuacje w perspektywie odległej mogą sprzyjać pozakrzepowej niedrożności żyły w miejscach przebiegu CIED lub CVC.

Słowa kluczowe: lewa żyła ramienno-głowowa, wenografia, kompresja żylna, okluzja, zwężenie, CIED, CVC

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