

Axillary vein spasm during cardiac implantable electronic device implantation

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[Received: 29 February 2016; Accepted: 5 April 2016]

Background: The technique of axillary vein (AV) or subclavian vein (SV) puncture has become an important alternative to cephalic vein (CV) cutdown as an approach allowing cardiac lead introduction into the venous system during cardiac implantable electronic device (CIED) implantation procedures. Irrespective of the technique used, the injury associated with lead insertion may induce a reflex venous spasm that can even cause total venous obstruction. In order to assess the incidence of AV spasm during AV puncture, we analysed a total of 735 (382 in females and 353 in males; mean age 75 ± 11 years) de novo CIED implantation procedures involving transvenous lead insertion conducted between January 2014 and December 2015.

Materials and methods: In 337 patients the leads were introduced via AV puncture only, in 66 patients AV puncture was used in combination with CV cutdown, together yielding a total of 403 procedures (55% of all de novo CIED implantation procedures; mean patient age 72 ± 14 years), out of which we observed 12 cases (mean patient age 57 ± 25 years) of AV spasm (3%).

Results: We evaluated only the procedures with unambiguous fluoroscopy images recorded during AV puncture: complete blockage of contrast medium flow through the AV, with preserved flow through the CV or collateral vessels, followed by eventually resumed flow of contrast via the AV. The contrast-enhanced movements of AV walls showed the spasm propagating both proximally and distally along the vessel, while the subsequent vessel wall relaxation occurred along the entire spasm-affected venous segment simultaneously.

Conclusions: An AV spasm induced by AV puncture during CIED implantation is a rare phenomenon; however, if severe, it may significantly affect the course of the procedure. (Folia Morphol 2016; 75, 4: 543–549)

Key words: venous spasm, axillary vein, venipuncture, venography, cardiac implantable electronic device

INTRODUCTION

Expanded indications for permanent cardiac pacing therapy have increased the number of cardiac implantable electronic device (CIED) placement procedures [3]. A vast majority of those are proce-

dures involving transvenous cardiac lead insertion. Currently, there are two main techniques for lead insertion: cephalic vein (CV) cutdown and axillary vein or subclavian vein puncture (AV/SV puncture) [2, 5, 15].

The AV/SV puncture technique, with modifications as to the specifics of its execution, has become a valuable complement to the CV cutdown technique [11, 12, 14]. The associated injury to the surrounding tissues as well as to the vein itself may induce reflex vascular spasm. In extreme situations this may lead to a complete occlusion of the venous lumen, which directly affects the course of the procedure, which may indirectly lead to later complications [9, 13].

The nature of this stimulus-induced reaction is associated with the tunica media, the smooth muscle cell layer of the vascular wall [1, 6]. The factors that may play a role in inducing a reflex spasm include both direct mechanical vascular injury (venous puncture, lead insertion) and compression by the surrounding tissues, temperature changes, chemical factors (drugs, contrast agents), etc. [4, 9]. Currently, intraoperative venography is still the only assessment allowing immediate and objective record of the development and course of the medical phenomenon of venous reflex spasm.

In light of scarce literature reports on vascular spasm during CIED implantation procedures, we attempted to analyse the phenomenon based on venographic images collected over a 2-year period.

MATERIALS AND METHODS

We analysed images from 735 *de novo* CIED transvenous implantation procedures conducted in 382 females and 353 males (mean age 75 ± 11 years) over the period from January 2014 to December 2015. We evaluated the rates of reflex AV spasm during CIED procedures during the use of the AV puncture technique for cardiac lead insertion within the clavipectoral triangle. The default implantation site was in the left infraclavicular region, with the right site used only in selected cases or according to the patient's wishes (in 3% of cases).

The reflex venous spasm may occur during transvenous CIED implantation procedures, including *de novo*, replacement, or up-grade procedures. In order to avoid diagnostic uncertainties due to potential overlapping organic vascular morphometric alterations resulting from earlier CIED implantation procedures, our analysis included *de novo* procedures only.

Only unambiguous images obtained during intraoperative (AV/SV puncture) venography were included in our analysis. They showed a complete contrast medium flow obstruction via AV/SV with preserved CV

flow or collateral flow, and the subsequently resumed AV/SV flow. Any questionable cases showing only the waning flow were excluded.

Venography was performed only when indicated by the need to visualise vascular layout for the use of the AV puncture technique. The default protocol involved AV puncture conducted based on normal anatomy of that vessel (Fig. 1A). After unsuccessful attempts at lead insertion and uncertainty as to the course of the AV, the puncture was conducted under the guidance of contrast-enhanced fluoroscopy (Fig. 1B).

Venograms were conducted according to the routine protocol, with the contrast medium administered into the veins of the ipsilateral forearm. Contrast flow was recorded in anterior-posterior view with the use of the GE OEC 9900 Elite C-arm system. We used pulsed fluoroscopy at 8 pulses per second (capturing individual frames, such as shown in the figures presented in this article).

The contrast agents used were low-osmolality non-ionic monomers: triiodo-isophthalamide derivatives (Iomeron 300). Intravascular flow was visualised following contrast administration at 15–30 mL (mean 20 mL), depending on interindividual variability of the venous bed at the site of administration.

Our statistical analysis used numerical variables in the form of means values.

RESULTS

Over the analysed period of time, 518 out of 735 *de novo* transvenous CIED implantation procedures involved pacemakers and 217 involved implantable cardioverter-defibrillators. CIED implantation with the use of only the CV cutdown technique was conducted in 332 (45%) patients. Lead insertion via the AV/SV puncture technique only was used in 337 patients; while the remaining 66 cases required the use of both techniques (mean age 72 ± 13 years). Out of these 403 CIED procedures, 12 patients (3%) (5 women and 7 men) developed AV spasm (age range at the time of the procedure was from 18 to 90 years, mean age 57 ± 25 years).

The reflex AV spasm phenomenon was assessed in cases of AV puncture technique for cardiac lead insertion with a special kit (Fig. 1A, B). In our material the reflex spasm was induced by venous wall puncture with a needle (Fig. 2, 7), as well as needle manoeuvres in the tissues surrounding the vessel (Fig. 3–6). The contrast-enhanced images of AV spasm showed the spasm to propagate both proximally (Fig. 6, 8) and distally along the vessel (Fig. 7).

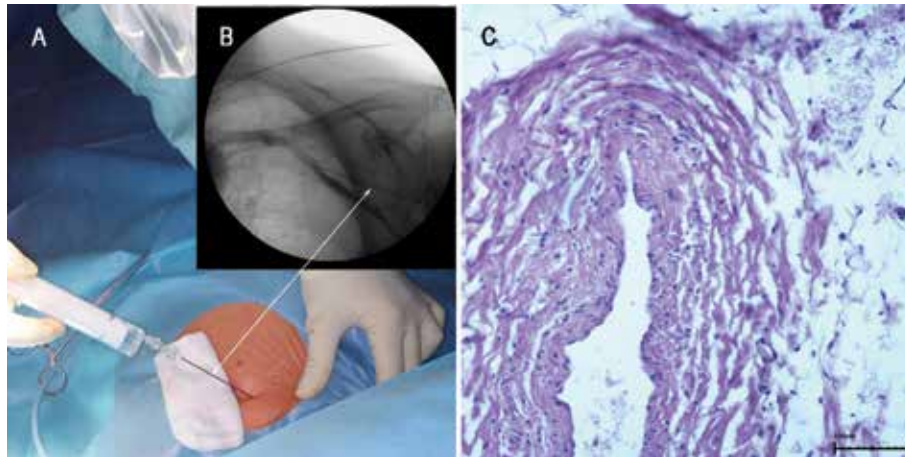


Figure 1. **A, B.** Axillary vein (AV) puncture with the use of an introducer needle attached to a syringe (white two-way arrow); **A.** Based on vessel topography; **B.** Under contrast-enhanced fluoroscopy of the clavicopectoral triangle; **C.** A cross-section of AV segment corresponding to the lead insertion site, with the smooth muscle tunica media visible. A histopathological microscopy image, haematoxylin-eosin staining (200-fold magnification). A postmortem histological sample from the Department of Pathology. Institutional Review Board's approval.

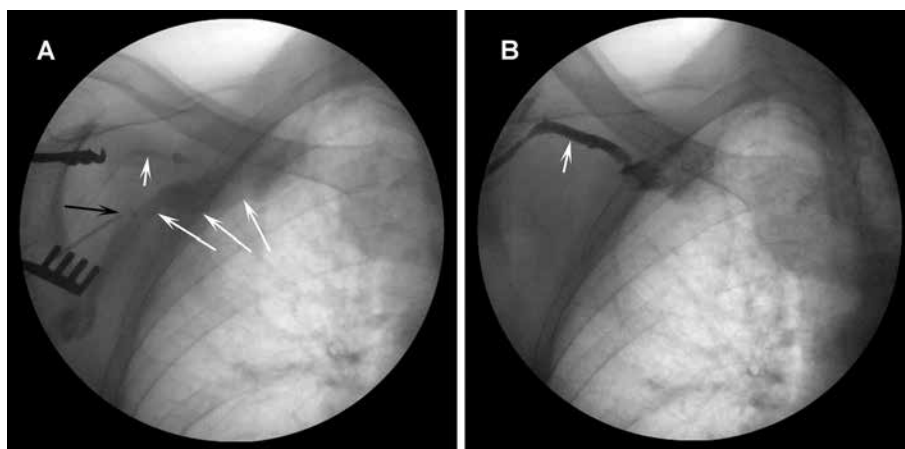


Figure 2. **A.** Venography during axillary vein (AV) puncture (black arrow) showing the contrast-enhanced AV, subclavian vein, and cephalic vein (CV) during an AV puncture attempt; **B.** Repeated venography following a failed lead insertion attempt shows only the contrast-enhanced CV with no contrast in the spasm-affected AV, distal to the CV. Short white arrow — CV, long white arrows — AV.

Irrespective of the direction of venous spasm propagation, the subsequent vascular wall relaxation occurred simultaneously in the entire spasm-affected venous segment (Fig. 3)

In cases of failed AV puncture, we achieved good effects with SV puncture. We observed no recordable imaging evidence of venous spasm within this vascular segment.

DISCUSSION

Morphometric, anatomical, and topographic characteristics of the CV, AV, and SV are decisive factors in the effectiveness of the first stage of a CIED implanta-

tion procedure, and involving cardiac lead introduction into the cardiovascular system. Both techniques, AV/SV puncture and CV cutdown, involve the vascular segments found within the clavicopectoral triangle [2, 15].

Anatomically, AV and SV belong to deep veins (coursing alongside arteries) of the upper extremity. Histologically, they are classified as medium veins. The tunica media contains circularly arranged smooth muscle fibres, which are responsible for vascular wall contraction (vasoconstriction) or relaxation (vasodilatation) (Fig. 1C). The nature and extent of this phenomenon is also regulated by fibres of the local autonomic nervous system and by local metabolic factors.

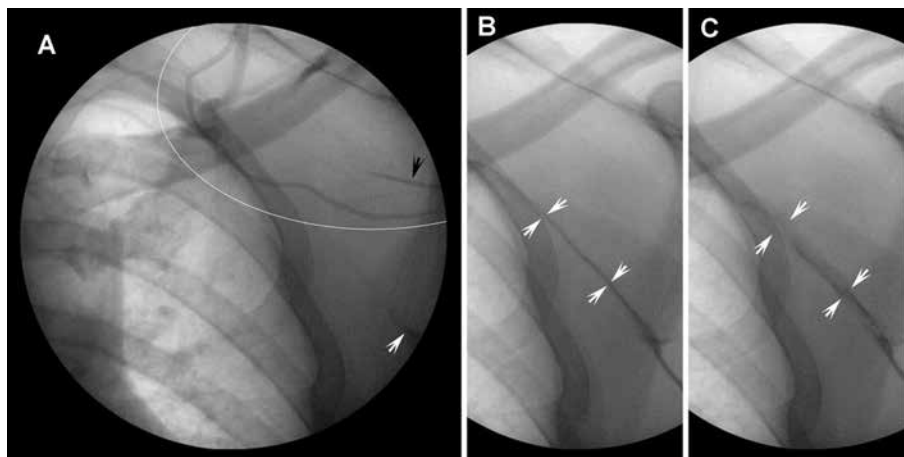


Figure 3. Venography following a failed attempt at obtaining access via axillary vein (AV) puncture (black arrow: introducer needle); **A.** A lack of contrast flow via the AV (white arrow), with simultaneous contrast flow via collateral circulation (white oval); **B, C.** A repeated assessment shows gradual return of contrast flow with AV lumen dilation (white arrow) and flow cessation in the collateral circulation.

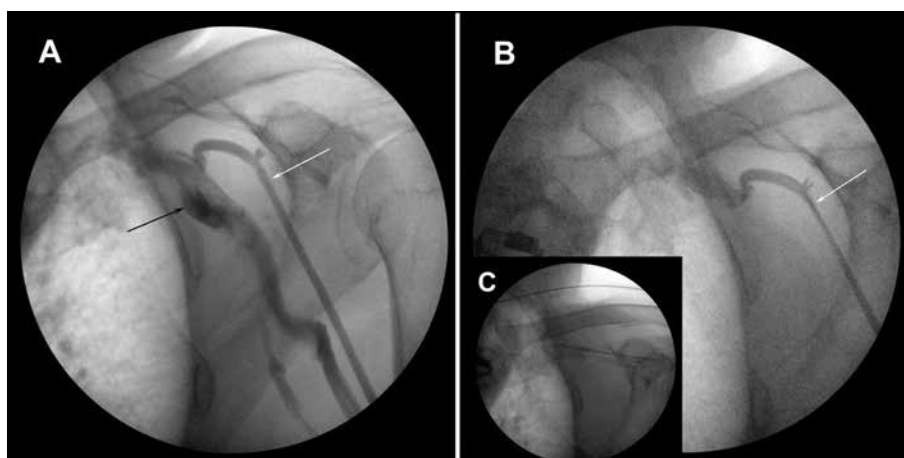


Figure 4. **A.** Venography conducted to select the cardiac lead insertion technique showed a peripherally reflected cephalic vein (CV: white arrow) drainage into the axillary vein (AV: black arrow) (precluding both lead insertion with the CV cutdown technique and any further proximal lead advancement), which was a decisive factor in choosing the AV puncture technique; **B.** Contrast re-administration to explain AV puncture failure showed no contrast flow via the AV up to the level of CV drainage; **C.** A DDD pacemaker was implanted with the use of subclavian vein puncture for lead insertion.

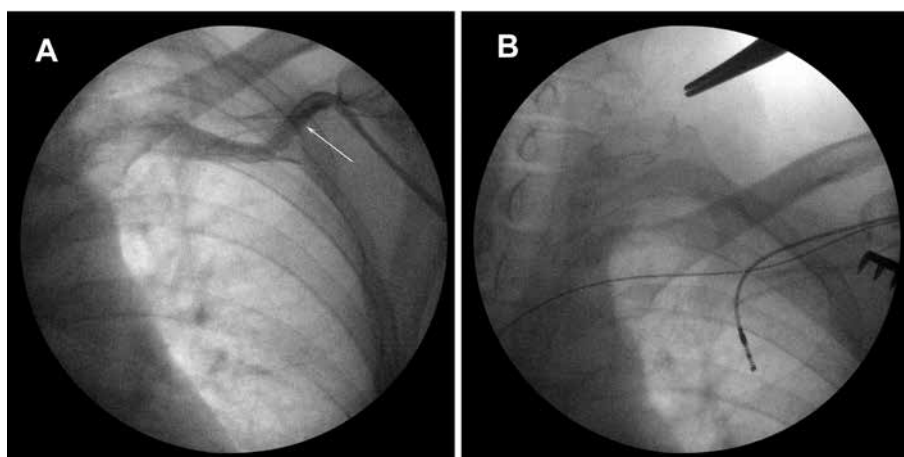


Figure 5. **A.** Failed axillary vein (AV) puncture attempts were an indication for venography, which visualised the lack AV contrast enhancement with preserved contrast flow via the cephalic vein (white arrow); **B.** The attempt at lead insertion via cephalic vein cutdown failed due to lead tip advancement peripherally within the AV; however, fluoroscopy confirmed the presence of this vessel. A DDD pacemaker was implanted using subclavian vein puncture for lead insertion.

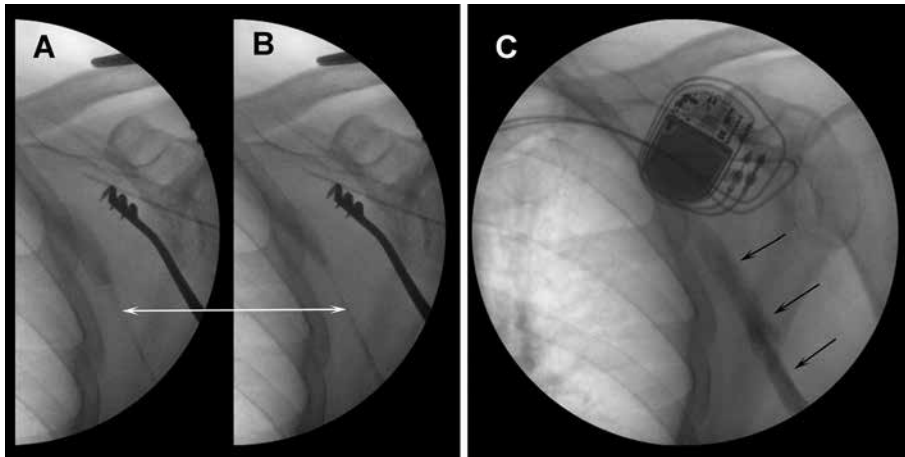


Figure 6. Cephalic vein was not detected and several failed attempts at axillary vein (AV) puncture were an indication to visualise vascular layout; **A, B.** CINE-mode fluoroscopy with contrast medium administration showed both the occurrence and proximal propagation of segmental AV spasm (white arrow); **C.** Post-procedure venography showed re-established patency of the vascular segment previously affected by the spasm (black arrows).

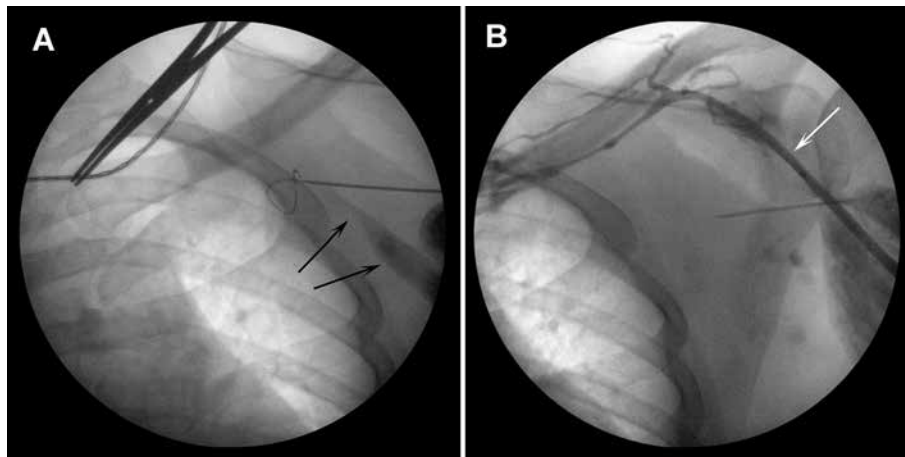


Figure 7. **A.** A failed attempt at introducer needle insertion via axillary vein puncture induced venous spasm, with spasm propagation peripherally along the vessel shown in the figure — the contrasted lumen tapered medially (black arrows); **B.** A repeated venography preceding another attempt at venous puncture showed complete axillary vein occlusion with preserved contrast flow only via the cephalic vein (white arrow).

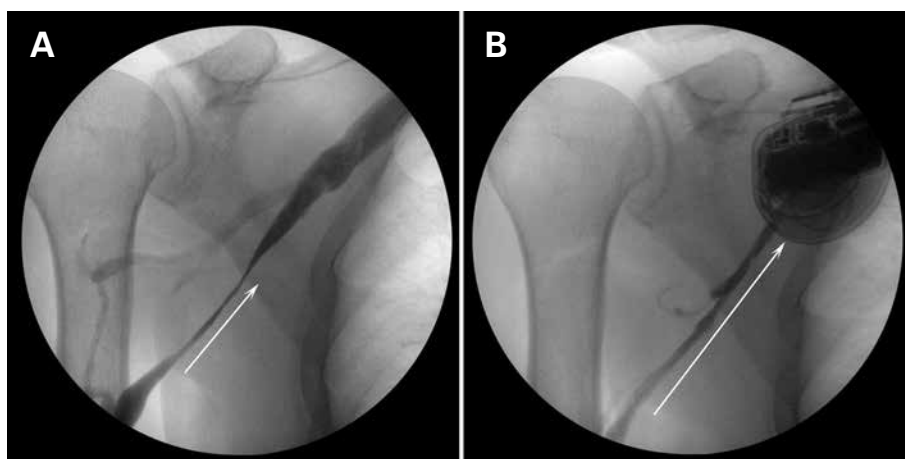


Figure 8. Difficulties in finding the cephalic vein coursing deep within tissues outside of the clavipectoral groove prompted lead insertion via axillary vein puncture. **A.** A repeated venography was performed to specify the location for another puncture site and revealed a venous spasm; **B.** Further intraoperative assessment revealed the spasm's propagation medially (white arrow).

The most common cause of blood flow obstruction and/or cardiac lead advancement during CIED implantation procedures are anatomical abnormalities at the level of the SV. This is a result of the vessel's location between the clavicle and the first rib, which offers the mechanical conditions for thrombus formation [1]. As a result, cardiac lead placement via SV puncture may facilitate its mechanical damage over time [13].

Blood vessel occlusion may be also functional. Vascular spasm may lead to a transient, complete blockage of blood flow. The signs and symptoms depend on the length and location of the occluded segment, lumen diameter, hypoperfusion duration, etc. Typically, the phenomenon subsides spontaneously without additional interventions or sequelae. However, a severe AV spasm occurring during a CIED implantation procedure may considerably affect its course. The situation may require cardiac lead insertion via SV puncture or completing the procedure on the other side of the chest [8].

A mechanical injury, such as puncture, induces a vascular defence response, and facilitates a 'spastic' reaction of vessels, including veins. Due to a larger proportion of muscle fibres in the tunica media and elastic fibres in the tunica intima, the histological wall structure of medium veins predisposes to contraction more than to relaxation. The contributing stimulating factors include increased catecholamine levels induced by injury to the vessel and surrounding tissues.

Venous spasm seems to be more common in people susceptible to stress and situations inducing elevated blood catecholamine levels, such as CIED implantation procedures. Our findings confirmed the association of this medical phenomenon with advanced age (> 70 years). However, venous spasm was also observed in young patients [9].

A loss of AV visibility during venography typically calls for SV puncture being performed instead. Our material showed no cases of reduced SV patency via venous spasm following SV puncture. This may be due to the fact that the patency of SV lumen is supported by two adjacent bony structures.

As of now, there is no single and specific preventive measure against or immediate treatment for venous spasm. There have been reports of preventive effect of 200 μg nitroglycerine administered 3 min prior to AV puncture [10]. The drug has been reported to be both effective following intravenous administration at 50 μg on the side of the spasm and

ineffective despite expectations [8]. Administration of 1% procaine, suggested by some, due to its analgesic and vasodilatory properties may have a beneficial vasorelaxant effect. On the other hand, sedatives (diazepam) might have some preventive effect or tend to shorten the duration of the venous spasm. In the cases presented here we observed a gradual, spontaneous resolution of the spasm, which is consistent with reports by other authors [7].

Venipuncture is one of the most common procedures, and vascular spasm is one of the associated phenomena. Despite this, there have been few reports on the occurrence and course of AV spasm during CIED-related procedures with the use of AV puncture, and the ones to be found rarely include larger study groups.

Limitations of the study

Our study included only a group of AV spasm cases detected *ad hoc*, without reference to a control group. CIED implantation procedures at our centre are conducted in the left infraclavicular region, which narrowed the scope of evaluating the studied phenomenon.

CONCLUSIONS

Venous spasm involving the AV and associated with the AV puncture technique during CIED implantation procedures is a rare phenomenon; however, irrespective of its extent, it may considerably affect the course of the procedure.

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