


An unyielding valve leading to venous spasm during pacemaker implantation: a case report

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Background

Subclavian venous spasm is an uncommon complication during permanent pacemaker implantation. The exact aetiology of subclavian venous spasm is not clear but has been suggested to be due to either mechanical irritation of the vein during needle puncture or due to chemical irritation from contrast injection. Here, we report a case of an unyielding subclavian vein valve that impeded guidewire advancement and the repeated guidewire manipulation led to venous spasm.

Case summary

A 45-year-old woman with a history of surgical repair of Tetralogy of Fallot in childhood presented with symptomatic bifascicular block and underwent a permanent pacemaker implantation. A subclavian venogram done prior to the procedure showed a prominent valve in the distal portion of the vein. Following venous puncture, guidewire advancement was impeded by the prominent valve. The resulting guidewire manipulation led to subclavian venous spasm necessitating a medial subclavian venous puncture and access.

Discussion

Prolonged mechanical irritation of the vein during pacemaker implantation may lead to venous spasm impeding pacemaker implantation. Early identification of an impeding valve and obtaining access medial to the valve may help prevent this uncommon complication.

Keywords

Subclavian venous spasm • Venous valve • Pacemaker • Case report

Learning points

- Subclavian venous valves can impede guidewire advancement during pacemaker implantation. Accessing the vein medial to the valve may help overcome this obstacle.
- Subclavian venous spasm is occasionally encountered during venous access. It is more likely to occur with multiple needle punctures and guidewire manipulations. Intravenous nitroglycerine maybe useful to prevent/relieve venous spasm but is associated with risk of hypotension.

Introduction

The subclavian vein—extrathoracic and intrathoracic, remain the preferred venous access routes for implantation of pacemaker and defibrillator leads. While associated with a high success rate, complications are occasionally seen and primarily include hemothorax (<1% of cases) and pneumothorax (seen in 0.6–5% of patients), which is seen less frequently with the adoption of extrathoracic subclavian vein cannulation.^{1–3} Subclavian venous spasm is a less commonly described complication and may necessitate a change in implantation site. The exact aetiology for this complication has not

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been defined but has been hypothesized to be due to mechanical or chemical irritation of the vein.⁴ Subclavian vein valves sometimes may serve as an impediment requiring increased guidewire manipulation and increasing mechanical irritation of the vein leading to venous spasm. Here, we describe a case of subclavian venous spasm, possibly related to mechanical irritation.

Timeline

- | | |
|---------|---|
| Case | <ul style="list-style-type: none"> • A 45-year-old woman presented for pacemaker implantation for symptomatic bifascicular block and intermittent 2:1 atrioventricular block. |
| Day 1 | <ul style="list-style-type: none"> • A subclavian venogram showed a patent left subclavian vein with a venous valve medial to the first rib. • Repeated needle puncture resulted in good flashback of blood, however, the guidewire could not be advanced across the valve. • Repeated guidewire manipulation resulted in venous spasm was evident on a repeat venogram. • Eventually, access was obtained medial to the site of the venous valve with subsequent implantation of a dual-chamber pacemaker. |
| Day 7 | <ul style="list-style-type: none"> • First follow-up post-implantation showed stable pacing and sensing parameters with a healthy incision. |
| Month 8 | <ul style="list-style-type: none"> • Stable pacing parameters with no recurrence of symptoms. |

Case presentation

A 45-year-old woman, with a history of hypothyroidism (on thyroid replacement therapy), scoliosis requiring surgical correction, major depressive disorder, and complete surgical repair of Tetralogy of Fallot 40 years ago, presented with symptoms of fatigue and exertional presyncope/syncope. Physical examination showed a heart rate of 50 b.p.m., blood pressure of 110/60 mmHg, with a normal first and second heart sounds, and an ejection systolic murmur in the pulmonary area. The jugular veins were not distended and there was no ankle oedema. Electrocardiogram showed a bifascicular block. Two-dimensional echocardiogram showed mild to moderate dilatation of the right atrium and ventricle with normal left ventricular size and function with moderate pulmonary regurgitation and no tricuspid regurgitation. Telemetry monitoring showed intermittent 2:1 atrioventricular (AV) block. Her current medications included levothyroxine 50 µg o.d., amitriptyline 10 mg o.d., and aripiprazole 3.75 mg o.d. In view of bifascicular block on the electrocardiogram, episodes of 2:1 AV block and symptoms of presyncope and syncope with no other aetiology identified, she was considered for a pacemaker implantation. The left extrathoracic subclavian vein was selected for venous

access. A venogram was obtained which showed an adequate sized patent subclavian vein with no evidence of stenosis (Figure 1A). Venous access was obtained with a 21G micropuncture needle (Arrow Simplicity[®]) with good flashback, however, attempts at advancing the guidewire beyond the medial head of clavicle were unsuccessful after multiple attempts. On careful inspection of the venogram, a valve was seen in the subclavian vein just before its union with the internal jugular vein. The tip of the guidewire during all attempts was seen to 'hook on' to the valve of the subclavian vein impeding further advancement beyond the valve. Three subsequent attempts were made with good flashback at all times but failure in advancing the wire. A subsequent attempt was made to gain access to the vein further laterally. However, venous access was unsuccessful with no blood flashback. Attempts at increasing the venous pressure with leg raising (Trendelenberg position) and intravenous fluid loading with 500 mL of normal saline were done but did not lead to successful venous access. A repeat venogram was obtained which showed non-visualization of a segment of the subclavian vein, with retrograde flow occurring to the medial portion of the vein through collateral vessels via the internal jugular vein and the left innominate vein (Figure 1B) (a total of 30 mL of contrast was used for both venograms). The possible aetiologies considered included Subclavian venous spasm, compression of the vein segment by a haematoma and subclavian vein thrombosis. Administration of nitroglycerine was contemplated, to relieve possible spasm, but in view of a systolic blood pressure of 90 mmHg it was not done. We contemplated switching over to the right side, but since the pulse generator pocket was already created we decided to attempt a more medial access to the intrathoracic subclavian vein which was successful. A dilator was passed over the guidewire and an additional guidewire was passed through the dilator to provide vascular access for two pacemaker leads, one placed in the right atrial appendage and the other in the right ventricular apical septum, with subsequent dual-chamber pacemaker implantation (the total procedural fluoroscopy time was 25 min) (Figure 2). A Doppler venous ultrasound was obtained the next day which showed a patent left subclavian vein with a normal flow pattern with no evidence of thrombosis or haematoma. The patient has completed 8 months of follow-up with no recurrence of symptoms.

Discussion

The extrathoracic subclavian vein has fast become the preferred access site for pacemaker and defibrillator implantation owing to the lower risk of pneumothorax and subclavian crush syndrome. Extrathoracic subclavian vein access has a success rate ranging from 95% to 98%.^{5,6}

Valves are commonly seen in the subclavian and internal jugular veins. While these valves usually can be traversed and do not impede lead placement, prominent valves may occasionally serve as an impediment as was seen in our patient. In most cases even such impeding valves can usually be overcome by prolapsing the body of the guidewire across the valve with subsequent dislodgement of the wire tip from the valve. However, this approach was not successful in our

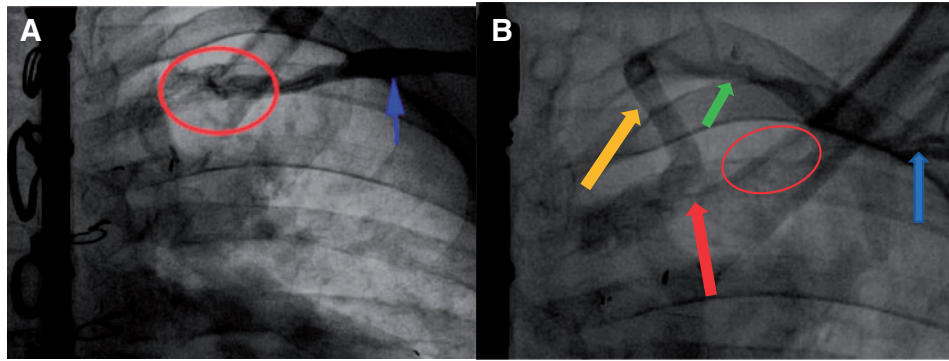


Figure 1 Subclavian venograms obtained by injecting contrast through a vein in the left forearm. (A) Left subclavian venogram obtained prior to subclavian vein puncture. The red oval indicates a prominent subclavian vein valve. Blue arrow indicates opacified subclavian vein. (B) Left subclavian venogram obtained after initial attempts at venous access. The red circle indicates non-opacified segment of subclavian vein due to venous spasm with opacification of vein laterally (indicated by the blue arrow). Opacification of the vein medially is through venous collaterals [green arrow; this collateral is seen arising from the lateral segment of the vessel and draining into the left jugular vein (yellow arrow) which drains into the subclavian vein to form the left innominate vein; opacification of the medial segment of the subclavian vein is retrograde]. Red arrow represents final site of venous access.

case. Alternatively, hydrophilic guidewires can be used in an attempt to negotiate the valve. We did not attempt this in our case.

Spasm of the subclavian vein during pacemaker implantation is a rarely described entity and is usually seen in elderly patients.^{4,7-9} However, Duan *et al.* reported on 74 patients undergoing extrathoracic subclavian vein puncture for pacemaker implantation. They performed a venogram in all cases both before venepuncture and after puncture either successful or unsuccessful. Venous spasm was seen in 28 patients (37.8%) with 22 patients having mild spasm and 6 (8.1%) patients having severe venous spasm. Access failure was seen in three patients with all patients having severe venous spasm.⁶ It is therefore likely that subclavian venous spasm is more common but underappreciated due to failure to get a second venogram especially during difficult or failed venepuncture.

The aetiology of venous spasm is unclear. It is postulated to be either due to the mechanical effect of needle puncture and guidewire manipulation or due to chemical irritation caused by the contrast. In the index case, given the multiple attempts at passing the guidewire across the valve, mechanical irritation was likely the offending mechanism.⁴

Duan *et al.*¹⁰ studied the role of intravenous nitroglycerine for the prevention of venous spasm and found lower rates of severe venous spasm in patients receiving nitroglycerine prior to puncture but resulted in a lower blood pressure compared to control patients. Nitroglycerine can also be used therapeutically following venous spasm and may be useful to help relieve venous spasm. In the index case, we decided against the use of nitroglycerine due to the lower baseline blood pressure.

A medial subclavian venous access was the preferred approach for pacemaker implantation previously but has now been replaced by access of the extrathoracic subclavian vein (axillary vein) due to the lower risk of pneumothorax and subclavian crush syndrome resulting in lead fracture.¹¹ In our case, we decided to proceed with a medial

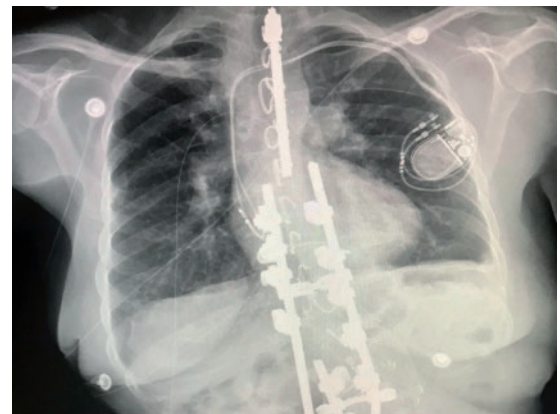


Figure 2 Chest X-ray PA view showing final device and lead position.

subclavian vein access (which was medial to the valve seen) so as to avoid crossing over to the right side. This approach was successful with no complications.

Conclusion

Valves in the subclavian and internal jugular veins may rarely impede pacemaker lead implantation. Prolonged manipulation of the guidewire within the vein may lead to subclavian venous spasm necessitating a cross over to the opposite side. If the medial subclavian vein is patent, a medial approach may be used though associated with a higher risk for pneumothorax.

Lead author biography



Darshan Krishnappa MD, DM, is a Cardiac Electrophysiology Fellow at the University of Minnesota. His areas of interest include catheter ablation of cardiac arrhythmias and cardiovascular manifestations of autonomic dysfunction.

Supplementary material

[Supplementary material](#) is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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