High-output heart failure due to subclavian vessel fistula () CrossMark as a late complication following implantation of a biventricular pacemaker



Björn Müller-Edenborn, MD,^{*} Jürgen Allgeier, MD,[†] Ralph Hartmann, MD,[‡] Thomas Zeller, MD,[§] Thomas Arentz, MD[†]

From the ^{*}Department of Cardiology, University Heart Center Freiburg - Bad Krozingen, Bad Krozingen, Germany, [†]Department of Electrophysiology, University Heart Center Freiburg - Bad Krozingen, Bad Krozingen, Germany, [‡]Department of Radiology, University Heart Center Freiburg - Bad Krozingen, Bad Krozingen, Germany, and [§]Department of Angiology, University Heart Center Freiburg - Bad Krozingen, Bad Krozingen, Germany.

Introduction

Biventricular pacing for cardiac resynchronization therapy (CRT) is a well-established treatment option for patients with inter- and/or intraventricular conduction delay with QRS width of more than 120 ms, systolic heart failure with ejection fraction (EF) <35%, and persistent symptoms of NYHA class II and higher despite optimal medical treatment. While some patients experience a large benefit from CRT, others (so-called "nonresponders") do not. Factors that favor a beneficial response to CRT include QRS width of >150 ms and left bundle branch block morphology, resulting in a class IA recommendation for CRT in these patients according to current guidelines.¹

We present the case of a patient who met all the above criteria for a class IA indication to CRT but, following an initial improvement of NYHA class, deteriorated owing to an uncommon late complication of CRT implantation.

Case report

We report the case of a 74-year-old patient with ischemic cardiomyopathy due to extended 3-vessel disease with repetitive coronary interventions. Left ventricular function remained moderately impaired with EF 30%-35% despite optimal heart failure medication. As electrocardiography demonstrated left bundle branch block with a QRS width of 180 ms, the decision for implantation of a biventricular pacemaker/defibrillator device was taken in 2011. However, implantation of the coronary sinus lead via the left

KEYWORDS Arrhyhtmias; Catheter ablation and implantable cardioverterdefibrillator; Hemodynamics; Cardiomyopathy; Heart failure; Pacemaker; Stent; Vascular disease(Heart Rhythm Case Reports 2016;2:425-427)

subclavian vein initially failed, leaving the patient with right-sided atrial and ventricular leads only. Because daily symptoms remained severe with exertional dyspnea NYHA class III, a second attempt for coronary sinus lead implantation was conducted in 2013 and succeeded without apparent complications. Two months later, both exercise tolerance and left ventricular function had significantly improved to NYHA II and EF 44%, respectively.

In 2015, the patient reported a drastic decrease in exercise tolerance within a week, with dyspnea after a few steps of walking. Pacemaker control at that time revealed 99% biventricular pacing. Echocardiography demonstrated a diffuse deterioration of left ventricular function with an EF of 26%, and physical examination revealed a new left-sided continuous bruit, as well as a thrill in the middle subclavian region. Computed tomography demonstrated a large fistula between the left subclavian artery and the subclavian vein with a connecting orifice of 6 mm (Video 1, available online, and Figure 1). The shunt volume was estimated by duplex sonography to approximately 2 L/min (Figure 2). We hypothesized that the deterioration of left ventricular function and exercise tolerance was attributable to the volume overload associated with the shunt. Implantation of a covered stent graft (Be-Graft 8/27 mm) via the femoral artery was performed (Video 2, available online, and Figure 3). Both postinterventional angiography and duplex sonography demonstrated no residual shunting. The patient experienced a significant improvement of subjective functional capacity and was able to climb a flight of stairs within a few days of the intervention.

A regular follow-up for reevaluation of left ventricular function was scheduled at 3 months. Unfortunately, the patient experienced non-ST-segment elevation myocardial infarction requiring stenting of the right coronary artery 2 weeks before scheduled follow-up. Echocardiography was

Address reprint requests and correspondence: Dr Björn Müller-Edenborn, University Heart Center Freiburg - Bad Krozingen, Südring 15, 79189 Bad Krozingen, Germany. E-mail address: bjoern.mueller-edenborn@ universitaets-herzzentrum.de.

KEY TEACHING POINTS

- Procedure-related complications can lead to deterioration of otherwise stable heart failure in cardiac resynchronization therapy patients and should be actively sought for.
- High-flow arteriovenous fistulas can present as a late complication several years following pacemaker implantation.
- A thorough physical examination including auscultation of the pacemaker-sided periclavicular area can help to diagnose arteriovenous fistulas.

performed following coronary intervention and demonstrated an EF of 30%.

Discussion

Arteriovenous fistulas (AVF) are infrequent complications in patients requiring cardiac pacemakers and are reported mostly following pacemaker lead extraction, rather than implantation. In this setting, Cronin et al² reported AVFs in only 8 out of 2471 patients (0.3%) that underwent pacemaker lead extraction. In only 1 of these cases, the AVF was located at the subclavian vessels. Surprisingly, most cases of AVF in this study were recognized not during the procedure, but only up to 587 days later. As in our case, the presence of a continuous bruit over the anterior chest was a consistent finding.

The etiology of AVF development following lead extraction or, as in our case, lead implantation remains poorly understood. Direct trauma to the artery owing to inadvertent puncture and subsequent local arteriovenous shunting

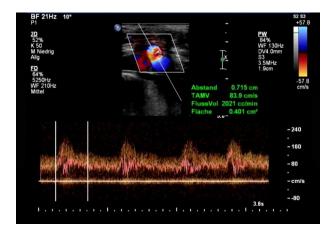


Figure 2 Doppler/duplex sonography with the pulsed-wave doppler window within the arteriovenous fistula demonstrates a calculated flow volume of 2021 cc/min.

preventing closure of the puncture site might play an important role in the development of AVF early following the intervention. Because Cronin et al reported a delayed diagnosis of AVF following lead explantation in most cases, we presume that AVF size increases over time until the shunt volume is large enough to produce a significant bruit or hemodynamic symptoms. Following lead implantation, local compression to the vessel wall might contribute to late *de novo* formation of AVF, but clinical data to support this hypothesis are unavailable.

The hemodynamic consequences of AVF are mostly studied in the context of AVF that were placed for hemodialysis. In these patients, central upper arm AVF were associated with almost 2-fold higher flow rates compared with peripheral forearm fistulas.³ As a consequence, invasive hemodynamic measurement revealed increased cardiac output in patients with upper arm fistulas.³

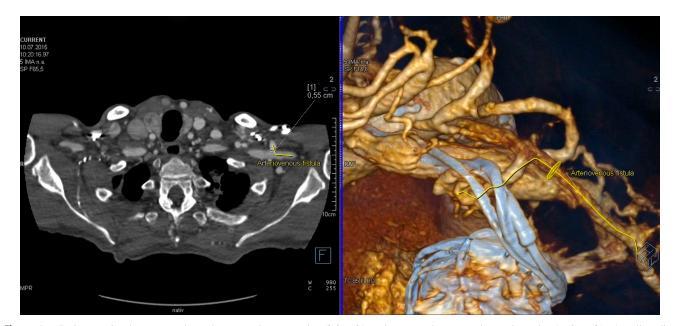


Figure 1 Preinterventional contrast-enhanced computed tomography (*left side*) and computed tomography angiography (*right side*). A yellow line demonstrates the course of the arteriovenous fistula.

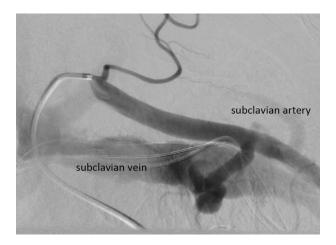


Figure 3 Preinterventional angiography via the femoral artery with the tip of the catheter located in the proximal subclavian artery shows large amounts of contrast media flowing through the fistula into the subclavian vein.

Although the general concept of high-output heart failure owing to increased AVF shunt volumes is well recognized, large controlled studies on this topic are scarce. Basile et al⁴ found that a cutoff shunt volume of 2 L/min was predictive for the development of high-output heart failure. Although their calculations were based on a very limited number of patients in a subgroup with NYHA class III symptoms, it was considered a landmark study when published in 2008. More recently, an independent group also reported a higher risk for heart failure with shunt volumes of 2 L/min or more.⁵ As a consequence, a shunt volume of 2 L/min or more is recommended for diagnosis of high-output heart failure.⁶ Of note, these recommendations refer to patients without a history of heart failure for other reasons. Our patient presented with a calculated shunt flow of 2.1 L/min. Taking into account his underlying heart disease and severe symptoms despite otherwise optimal medical treatment, the decision was taken for an interventional approach to close the AVF by covered stenting. His functional status improved significantly over the following days, demonstrating that closure of high-flow AVF should be evaluated in this patient subset.

Appendix

Supplementary data

Supplementary data are available in the online version of this article at http://dx.doi.org/10.1016/j.hrcr.2016.06.005

References

- Brignole M, Auricchio A, Baron-Esquivias G, et al. 2013 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy: the Task Force on cardiac pacing and resynchronization therapy of the European Society of Cardiology (ESC). Developed in collaboration with the European Heart Rhythm Association (EHRA). Eur Heart J 2013:2281–2329.
- Cronin EM, Brunner MP, Tan CD, Rene Rodriguez E, Rickard J, Martin DO, Wazni OM, Tarakji KG, Wilkoff BL, Baranowski BJ. Incidence, management, and outcomes of the arteriovenous fistula complicating transvenous lead extraction. Heart Rhythm 2014;11:404–411.
- Wijnen E, Keuter XH, Planken NR, van der Sande FM, Tordoir JH, Leunissen KM, Kooman JP. The relation between vascular access flow and different types of vascular access with systemic hemodynamics in hemodialysis patients. Artif Organs 2005;29:960–964.
- Basile C, Lomonte C, Vernaglione L, Casucci F, Antonelli M, Losurdo N. The relationship between the flow of arteriovenous fistula and cardiac output in haemodialysis patients. Nephrol Dial Transplant 2008;23:282–287.
- Ye W-L, Fang L-G, Ma J, Li X-M. Long-term effects of arteriovenous fistula on cardiac structure and function in non-diabetic hemodialysis patients. Zhongguo Yi Xue Ke Xue Yuan Xue Bao 2013;35:95–101.
- Stern AB, Klemmer PJ. High-output heart failure secondary to arteriovenous fistula. Hemodial Int 2011;15:104–107.