First-in-Man Coronary Sinus Lead Stabilization Using a Bioresorbable Vascular Scaffold System

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Cardiac resynchronization therapy has become an integral part of treatment in patients presenting with reduced ventricular function (left ventricular ejection fraction <35%), clinically symptomatic dyspnea (New York Heart Association II–IV), and complete left bundle branch block. Currently, the standard approach of left ventricular lead placement is transvenously via the coronary sinus (CS). Although a wide range of CS leads, sheaths, and subselectors are available, peri- or postinterventional lead dislodgement is still a cause for placement failure. Interventional stabilization by metallic stents of the CS lead has been described, but there are concerns on mid- and long-term effects because of possible mechanical irritation. Here we describe the first case in which a bioresorbable vascular scaffold was used to stabilize a CS lead in a lateral side branch against the vessel wall.

Case Report

A 74-year-old man with dilated cardiomyopathy, New York Heart Association Class III, had an implantable cardioverter defibrillator placed in 2008 for primary prophylaxis of sudden cardiac death. In January 2015, the patient presented with worsening heart failure and a markedly decreased ejection fraction (left ventricular ejection fraction 20%). Bradyarrhythmia (<40/min) resulted in dyssynchronous right ventricular apical pacing ≥295%.

Decision to upgrade the patient’s system to a cardiac resynchronization therapy system (St Jude Medical, PROMOTE CD3211-36, St Paul, MN) was made in line with current guidelines.1 The venogramm revealed a huge CS with only one suitable lateral side branch with a 110° angle and a kinking at 1.5 cm followed by a distribution into 2 smaller branches (Figure 1). A quadripolar lead (St Jude Medical Quartet, St Paul, MN) became immediately dislodged. After changing the lead to a bipolar lead (St Jude Medical Quickflex μ 1258T, St Paul, MN), the proximal portion of the target branch was reached with good sensing and pacing values and no phrenic nerve capture, but dislodgement occurred again. However, despite the use of a stiff wire and an additional buddy wire (Galeo Pro; Biotronik, Berlin, Germany), the lead could not be further advanced to establish a stable position. We decided to introduce a 3.00x12 mm bioresorbable scaffold (Abbott, Absorb, North Chicago, IL) via a second simultaneous CS catheter (St Jude Medical CPS 115, St Paul, MN) into the proximal portion of the side branch in an attempt to stabilize the lead against the side of the vessel and prevent its dislodgement. The scaffold was positioned directly proximally to the second electrode. The balloon was inflated for 60 s at 14 atm (Figure 2). Afterward, the final position showed a capture threshold of 1.0 V, pulse width of 0.5 ms, impedance of 890 Ω, and R-wave sensing of 7.7 mV (Figure 3). A 30-day follow-up showed stable measurements with improved heart failure symptoms.

Discussion

Currently, the most widely used method of left ventricular lead placement involves transvenous placement via the CS into a preferentially lateral or posterolateral side branch.2 Despite interventional advances, peri- or postinterventional lead dislodgement in 6% to 14% of all cases is a common cause for redo procedures and results not infrequently in epicardial positioning of the left ventricular lead.3 Interventional stabilization of the CS lead by metallic stents has been described before showing reasonable outcomes.4 However, a major concern has always been raised regarding the extractability of a stented CS lead, for example, in case of infective endocarditis.

The utilization of a bioresorbable vascular scaffold, which disappears after ≈2 years, offers potential benefits, such as restoration of physiological vasomotion. In addition to the stability acquired by the scaffold, we expect a common thrombotic and fibrotic fixation of the lead after a few months. Once the scaffold is absorbed, a lower risk of future lead fracture and easier extractability because of nonpermanent trapping can be expected. Yet, late CS lead dislodgement is conceivable because of a loss of support after full resorption of the bioresorbable vascular scaffold, despite increased endovascular stability over time because of formation of an intimal tissue layer with tube-like configuration around the lead. In conclusion, this is the first report of bioresorbable vascular scaffold placement in the venous system in general and particularly for CS lead stabilization. It remains to be seen how the scaffold’s resorption characteristics affect the lead’s long-term integrity, future extractability, and position stability.
Disclosures
Drs Schmitt and Erkapic have received speaker’s honoraria, congress and institutional research grant from St Jude Medical. Dr Nef has received speaker’s honoraria and institutional research grant from St Jude Medical and Abbott Vascular. The other authors report no conflicts.

References

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Figure 1. Coronary sinus (CS) angiography showing the lateral target vein (LAO 40°).

Figure 2. Delivery of the bioresorbable vascular scaffold (BVS) proximal to the second electrode pole of the coronary sinus (CS) lead via a second CS sheath (SJRM, CPS 115, and subselector 90°). LAO 28°.

Figure 3. Final position of the bioresorbable vascular scaffold (BVS)–stabilized lead after removal of the sheaths. LAO 40°.
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