Unusual Placement of a Coronary Sinus Lead for Resynchronization Therapy Resulting in Late Lead Fracture

Sergio F. Cossú, MD, FACC, FHRS

The Arrhythmia Center at the Charlotte Heart and Vascular Institute, Port Charlotte, FL

Abstract. An 88-year-old female underwent upgrade of a dual-chamber cardioverter-defibrillator to an atrial synchronous biventricular defibrillator with adequate sensing and pacing thresholds from the coronary sinus lead. The patient initially had significant hemodynamic improvement in her heart failure symptoms; however, after 5 years she deteriorated once again to her pre-implant status. This was associated with loss of capture of the coronary sinus lead and a significant increase in pacing lead impedance suggesting lead fracture. Intraoperative evaluation demonstrated that the lead had most likely inadvertently perforated the right ventricular wall at implant and was within the pericardial space thus pacing the left ventricle from an epicardial location. Persistent myocardial contraction around this lead eventually led to its fracture. This is an extremely rare case where the patient actually did have 5 years of apparent resynchronization therapy with significant hemodynamic improvement as a result of inadvertent epicardial placement of a left ventricular lead.

Keywords. cardiac perforation, cardiac resynchronization therapy devices, coronary sinus lead implantation, epicardial pacing, pacemaker lead fracture.

Introduction

Cardiac resynchronization therapy for patients with congestive heart failure and ventricular dyssynchrony has become an effective and established method of treatment. In situations where the coronary sinus cannot be cannulated for lead placement or adequate locations for pacing cannot be achieved, an epicardial left ventricular lead is implanted via a thoracotomy approach. Lead fractures may occur chronically in patients and are usually due to continuous friction or traction on the lead. The most common location for lead fracture is subclavicular, due to continuous compression, or “crush” on the lead. Perforation of the right atria or right ventricle during placement of pacing leads has been described and is a rare and known complication. Perforation during placement of a coronary sinus lead usually involves dissection of the coronary sinus. We present here an interesting and rare situation where the coronary sinus lead was intended and thought to be within the coronary sinus and indeed demonstrated evidence of left ventricular capture; however, the lead had perforated through the right ventricle and paced the left ventricle from within the pericardium. The continuous myocardial contraction around the lead, from where it exited the right ventricle, resulted in significant strain and tension and eventually led to its fracture at this location.

Case summary

The patient is an 88-year-old female with a history of a non-ischemic cardiomyopathy and ejection fraction of 15%. The patient underwent initial placement of a dual-chamber implantable cardioverter-defibrillator in 2005, which was upgraded in 2006 at another institution to a biventricular device because of a significant deterioration...
in the patient’s functional classification. The operative report from this procedure stated that the patient’s heart was extremely rotated, making coronary sinus access extremely difficult; however, a wire was successfully advanced into a mid-lateral branch of the coronary sinus, where a lead was positioned. The operative report does not mention if coronary sinus venography had been performed. The implanted coronary sinus lead was a Boston Scientific (Boston, MA) model 4518 (5.4 French). The measured R-waves in this location were 25 mV, with a pacing threshold of 1.0 V at 1.5 ms and an impedance of 970 ohms. The patient returned to our institution, and follow-up clearly documented evidence of both right and left ventricular capture. Intracardiac electrograms are shown in Figure 1 clearly demonstrating both right and left ventricular capture. Over the subsequent 5 years, the patient responded well to her resynchronization therapy with significant improvement in her congestive heart failure symptoms and New York Heart Association classification. In 2011 it was noted that the left ventricular lead impedance was gradually increasing until finally there was evidence of loss of left ventricular capture and an impedance of greater than 2,000 ohms. This was associated with a significant decline in the patient’s clinical status. A chest X-ray was performed which demonstrated a strange discontinuity approximately 7 cm from the distal end of the left ventricular lead, a portion of which should still be well within the body of the coronary sinus (Figure 2). The patient was subsequently taken to the electrophysiology laboratory with the intent to extract this lead and place a new lead in the coronary sinus. Initial fluoroscopy clearly demonstrated that there was evidence of a fracture in the same region as seen on the chest X-ray, and there appeared to be a hinge point at this location, a finding which would be extremely rare if the lead was indeed within the coronary sinus. After the coronary sinus was engaged and cineangiography was performed it became evident that the left ventricular lead was not in the coronary sinus but rather had perforated through the right ventricular wall and was within the pericardial space (Figure 3). The patient underwent extraction of this lead in a cardiothoracic surgical suite with transesophageal echocardiographic visualization. A laser locking stylet was passed through the lead; however, it could not be advanced beyond the area of the lead fracture. Attempts at withdrawing the lead with gentle traction were unsuccessful as well. Subsequently, a 12 French excimer laser was advanced over the lead into the right ventricle. The lead separated completely at the exit point from the right ventricle and the entire portion of the lead which was within the right ventricle was able to be extracted along with a considerable amount of insulation accompanying the lead. Fluoroscopically, the pericardial portion of the lead remained undisturbed in place. Transesophageal visualization did not demonstrate any significant increase in pericardial effusion. Figure 4 demonstrates the distal tip of the old lead, which could not be recovered and remained within the pericardial space, as well as the appropriate placement of the new laterally placed coronary sinus lead. After eventually having successful placement of a coronary sinus lead in a mid-lateral branch, the patient achieved resynchronization and clinical improvement.

**Discussion**

The placement of pacing devices with non-thoracotomy lead systems has become routine and is usually quite safe with an extremely low procedural complication rate (8%). The incidence of lead perforation is low (0.1–0.8% for pacemaker leads and 0.6–5.2% for defibrillator leads). This occurs more commonly in the right atrium than in the right ventricle because of the variation in wall thickness (2 mm versus 4–5 mm), and can occur with both passive and active fixation leads. Predictors for perforation include the concomitant use of a temporary transvenous pacing lead, steroid use, older age, body mass index <20, and the use of a helical screw lead. Although perforation may occur acutely and result in cardiac tamponade, recent studies reported the occurrence of delayed perforation, which may be a result of an asymptomatic acute perforation or a true late perforation. Perforation of the heart with a pacing lead usually does not result in significant hemodynamic compromise,
Figure 2: Posterior-anterior chest X-ray demonstrating a “kink” in the left ventricular lead within the coronary sinus (arrow).
most likely due to the low-pressure system associated with the right heart and fibrosis occurring at the lead–myocardium interface, causing the perforation site to seal up. Hirschl et al recently reported the incidence of asymptomatic lead perforation diagnosed by computed tomography to be quite high and usually unrecognized (15%). Interestingly, measured follow-up data from the pacing device, i.e., sensing and pacing thresholds and lead impedances, are usually not affected and thus makes the recognition of the perforation more difficult.

The incidence of pacing lead fractures is about 1–2.5% but has been reported as high as 7.2%11 and even higher (12%) in implantable cardioverter-defibrillator leads.12 The most common location for lead fracture is in the infraclavicular region. This occurs because of the lead becoming entrapped within the subclavius muscle or costoclavicular ligament or due to compression of the lead between the clavicle and the first rib (subclavian crush syndrome). Distal lead fractures are extremely rare and have only been reported in isolated case studies.13 A fracture in a distal location as presented here would also be extremely rare; however, a similar mechanism to lead binding, or entrapment within the muscle, could be invoked. It is interesting to note that the fracture did not occur sooner and most likely speaks of the durability of the lead insulation. The lead had scarred into its position in the pericardium, most likely at the point where it entered the epicardium from the endocardium. This is evident by the inability to effectively remove the distal portion of the lead that was within the pericardial space at the time of the lead extraction. The persistent myocardial contraction occurring around the lead over 5 years, created a hinge point in the lead, most likely at the point where it exited the epicardium, which eventually led to fracture of the lead. This mechanism has been thoroughly described previously.14 A lead which is designed as a helix is able to bend back and forth without causing any stress at the site of flexure. However, if part of the lead is entrapped within muscle, i.e., the right ventricular wall, the wire may fracture usually at the junction between the bound and the unbound portions of the helix.

Complications related to placement of a left ventricular lead in resynchronization devices are usually related to dissection of the coronary sinus, with an incidence of approximately 6.8%.15,16 We report here for the first time an unusual complication where the coronary sinus lead had most likely acutely perforated through the right ventricle at the time of the implant and was able to pace epically. It is unlikely that the lead itself actually caused the perforation, but rather that the coronary sinus guiding catheter entered the right ventricle while attempting to engage the coronary sinus os, subsequently perforating through the right ventricular wall, and the lead then advanced into the pericardial space. Interestingly the patient did not suffer any hemodynamic compromise at the time of the initial implant once the guiding catheter (8 French in diameter) was removed, obviously leaving a significant space around the 5.4 French lead now in the pericardial space. The second and more fascinating aspect of this case is the fact that the lead was actually successfully placed in an epicardial position with adequate sensing and pacing thresholds. Indeed, epicardial leads are sometimes placed in patients in whom the coronary sinus anatomy does not allow for placement of the lead transvenously.

**Figure 3:** Fluoroscopy with left anterior oblique and right anterior oblique views during coronary sinus angiography demonstrating the location of the left ventricular lead well outside of the coronary sinus. A discontinuity of the lead is visualized which corresponds to the site of perforation through the right ventricular wall.
and are usually placed via a thoracotomy approach. In this case, the “epicardial” lead was placed via an endocardial approach. The third interesting feature of this case is that even though the lead, as visualized by fluoroscopy, was in a septal location within the epicardium, the patient actually enjoyed 5 years of improvement in heart failure symptoms as a result of resynchronization therapy. Further, this reverted back to the pre-implant scenario once the left ventricular lead failed to capture. Although a placebo effect could be invoked here, the patient did have significant objective clinical evidence of worsening New York Association Classification status after the lead fractured and failed to capture. Following placement of the new coronary sinus lead in a lateral position as shown in Figure 4, the patient regained her quality of life and had a significant improvement in cardiac function.

In conclusion, we report here an extremely rare complication of placement of a coronary sinus lead never documented previously in the literature. This initially resulted in a clinical benefit to the patient, but ultimately gave way to a serious detriment requiring further surgical intervention.

References


Figure 4: Left anterior oblique fluoroscopic view of the new left ventricular lead in a proper location within a lateral branch of the coronary sinus lead. The remnant of the previous lead is seen within the pericardial space.