Late resolution of pacemaker lead–related severe tricuspid regurgitation and right ventricular dysfunction after percutaneous lead extraction: A case report and review of the literature

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Introduction

Endocardial lead–related tricuspid regurgitation (ELTR) is an increasingly recognized complication of cardiac device implantation that can result in right ventricular (RV) dysfunction and right heart failure.[1] Several mechanisms of ELTR have been proposed, including lead impingement on tricuspid valve (TV) leaflets; leaflet perforation; entanglement within the valve apparatus; adherence to TV leaflets, chordae, or papillary muscles; and altered RV activation or geometry owing to RV pacing.[1,2] Irrespective of the mechanism(s) involved, it has been postulated that the mechanical and hemodynamic consequences of ELTR on the TV and RV should manifest by 6 months post–device implantation, delineating the time period during which compatible signs and symptoms should most raise suspicion for this condition and suggesting that early intervention may be important.[1]

Though both surgical and percutaneous treatment options for ELTR have been reported, surgery has been advocated as the default treatment by some, in part because of a perceived high risk of procedural complications with percutaneous options, including damage to the TV.[3] However, case series of patients undergoing TV surgery for isolated severe tricuspid regurgitation (TR), including cases of ELTR, have reported considerable operative morbidity and short-term mortality as high as 19%.[4–6] In contrast, major complications occur in <0.8% of patients undergoing contemporary percutaneous lead removal at experienced centers[7] and in as little as 0% of cases involving leads less than 1 year old.[8] Nevertheless, the role of percutaneous interventions for ELTR remains unclear, with 3 reports suggesting unpredictable improvements in TR and RV function with this

approach.[9–11] We describe a patient with severe, symptomatic ELTR associated with RV dysfunction and tricuspid annular dilatation that resolved between 10 and 12 months after percutaneous ventricular lead extraction.

Case report

An 84-year-old woman with paroxysmal atrial fibrillation (AF), hypertension, mild cognitive impairment, and remote upper gastrointestinal bleed presented to hospital with palpitations. She was noted to have prolonged postconversion sinus pauses on telemetry with presyncope and therefore underwent an uncomplicated dual-chamber permanent pacemaker (PPM) implantation (Sensia; Medtronic, Minneapolis, MN). A transthoracic echocardiogram (TTE) performed prior to device implantation documented normal biventricular size and systolic function, as well as mild mitral regurgitation and mild TR.

Within several weeks she began to complain of "fluid retention," requiring readmission to hospital approximately 6 months post–PPM implantation because of progressive dyspnea, fatigue, marked peripheral edema, and gross ascites. TTE showed new severe TR (Figure) and moderate RV dysfunction, as well as mild left ventricular (LV) dysfunction with an LV ejection fraction (LVEF) of 45%– 50% (see Supplementary Table 1 and Video, available online, for details of all echocardiographic studies). Interrogation of her PPM revealed sustained AF and minimal RV pacing. A 3-dimensional echocardiogram (3DE) demonstrated that the ventricular lead ran in the TV posteroseptal commissure with evidence of resultant restriction predominantly of septal leaflet excursion (Figure). Clinically, the patient continued to deteriorate despite aggressive diuresis.

Following discussions with the patient and her family, it was decided to percutaneously remove the RV lead. This was performed using a lead locking device and gentle traction without complication 10 months after device implantation. A ventricular lead was not reimplanted in an alternate

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KEY TEACHING POINTS

- Endocardial leads from cardiac devices have the potential to interfere with tricuspid valve function. This process should be considered in patients who present with signs or symptoms compatible with tricuspid valve dysfunction after device implantation.

- Surgical management of endocardial lead–related tricuspid regurgitation (ELTR) is generally favored in the literature; however, there are limited data to guide treatment.

- Our case suggests that percutaneous lead removal may have a role even in advanced cases of ELTR and that subsequent recovery of right ventricular function can be delayed.

position (ie, coronary sinus) because the patient had remained in persistent AF.

When seen in clinic 3 months afterwards, she continued to have marked right heart failure. A repeat TTE showed persistent severe TR with severe RV dysfunction and moderate

RV dilatation but normalized LVEF. At 5 months post–lead extraction, she was readmitted to hospital for diuresis and paracentesis, and to explore the option of TV surgery. However, given her frailty, advanced age, and high procedural risk of surgical options, it was decided to continue conservative management. After a prolonged stay in hospital, Palliative Care was consulted and she was discharged home with a guarded prognosis, briefly requiring a stay in a palliative care hospital unit for delirium attributed to aggressive diuresis. A repeat TTE the following month was unchanged.

However, over the subsequent few months her symptomatic status progressively improved and a TTE performed 12 months post–lead extraction revealed only mild TR with normal biventricular size and function. She was once again living in her retirement home, was walking on a daily basis, and had not required any further hospital admissions.

Discussion

Clinically significant ELTR is thought to be relatively rare, but its incidence, prevalence, and clinical impact are unknown and likely underappreciated.[1,3] In addition to the potential morbidity associated with the condition, it likely
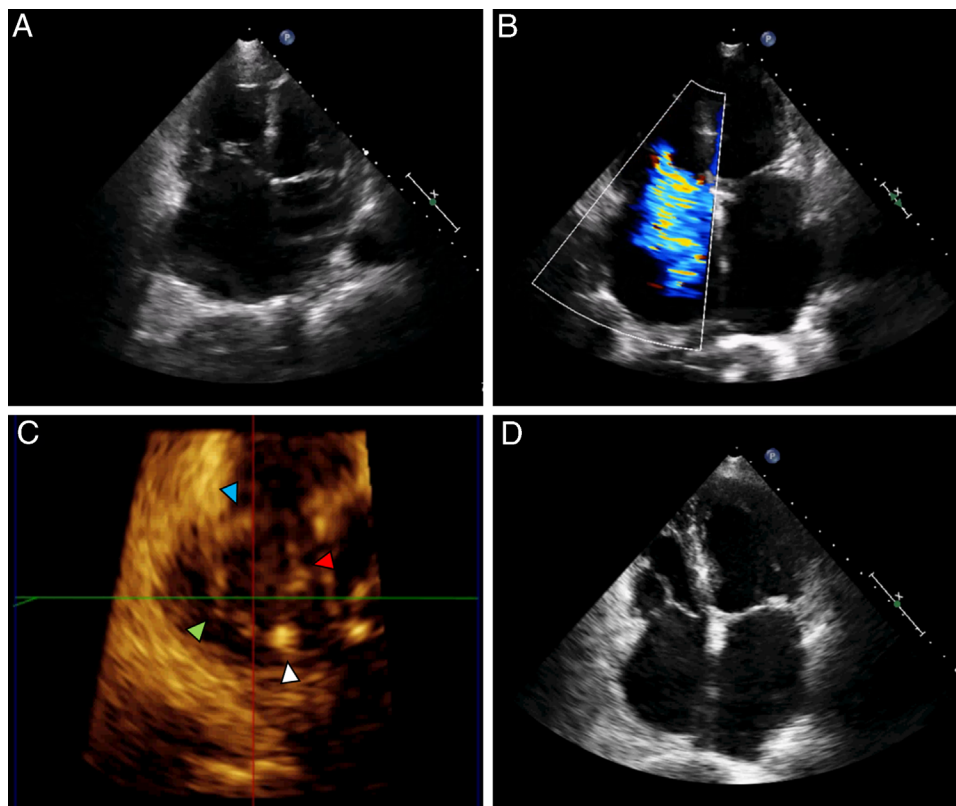
Figure Echocardiographic images. **A:** Apical 4-chamber view demonstrating poor coaptation of tricuspid valve (TV) leaflets, biatrial enlargement, and right ventricular (RV) dilatation 6 months after pacemaker implantation. **B:** Doppler signal confirmed severe tricuspid regurgitation (TR), but its etiology could not be determined. **C:** Three-dimensional echocardiogram showing the pacemaker lead (*white arrowhead*) lying in the commissure between the posterior (*green*) and septal (*red*) TV leaflets. Restriction of septal leaflet systolic excursion was observed and attributed to the lead. The anterior leaflet (*blue*) is also visible. **D:** Apical 4-chamber view demonstrating improved TV leaflet coaptation and normalization of RV size 12 months after percutaneous lead extraction. Mild TR was noted (*not shown*).

impacts patient survival, as moderate or greater TR has been associated with increased mortality irrespective of pulmonary artery pressure and LVEF.[12] However, the optimal management of the condition remains unclear.

The strongest evidence for surgical management for ELTR is from a case series by Lin et al[2] of 41 patients undergoing TV surgery for this indication over an 11-year period at a single center. The authors reported 1 perioperative death with an additional 4 deaths in follow-up, as well as clinical improvement in all patients with follow-up data available. However, only 19 of the cases underwent isolated TV surgery. Furthermore, follow-up data were not available for over one fifth of surviving patients, outcomes in the subset of isolated TV surgeries were not described separately, and details regarding symptomatic improvement were limited. Additional evidence in support of surgical intervention for ELTR stems from case reports and small case series, mostly of patients who underwent lead removal and TV repair or replacement. Nearly all of these cases describe some degree of symptomatic or echocardiographic improvement, with no deaths reported either in the perioperative period or at follow-up (Supplementary Table 2, available online).

Evidence for percutaneous interventions for ELTR similarly consists of observational data. Nazmul et al[9] retrospectively reviewed all cardiac device procedures performed at a single center over an 11-year period and identified only 4 patients who underwent percutaneous RV lead extraction for ELTR with ventricular lead reimplantation in the coronary sinus. One patient died 8 days post–lead extraction and only 1 patient was noted to have modest echocardiographic improvement in TR severity following the procedure, despite all extractions occurring within 1 year of lead implantation. It was proposed that tricuspid annular dilatation, which was present in all cases, may be both a marker and a mechanism for irreversible ELTR and therefore that surgical options may be required once it occurs. In contrast, Polewczyk et al[10] described improvement in TV function in 15 of 24 patients with lead-related TV dysfunction (20 with severe ELTR, 4 with coexisting tricuspid stenosis) treated with percutaneous lead extraction despite reimplantation of an RV lead in the majority of cases. "Marked clinical improvement" was noted in 75% of patients at a mean follow-up of 1.5 years, including improved exercise tolerance and peripheral edema. However, many cases of ELTR in their series were attributed to atrial or LV leads, which have not been described by others as causes of the condition, and details regarding RV function, time course of recovery, or frequency of TV damage during lead extraction were not provided. Wardell et al[11] recently described 6 patients with presumed ELTR who underwent percutaneous RV lead removal and reimplantation. Four had prompt clinical improvement; however, 1 patient did not, and died soon afterward, while another required surgical repair for iatrogenic TV damage (Supplementary Table 2).

Wardell et al[11] proposed that ventricular septal flattening on echocardiography in the absence of an alternate explanation for RV dysfunction may be of diagnostic value for ELTR and that it may predict whether percutaneous RV lead reimplantation could be beneficial. Though LV septal flattening during end-diastole on echocardiography (resulting in a "D-shaped" cavity or an elevated "eccentricity index") is suggestive of RV volume overload,[13] in cases of suspected ELTR cautious interpretation of this finding is required, as it may not distinguish between lead-related and lead-unrelated etiologies or elucidate the mechanism of ELTR, and therefore it may not be sufficient to determine the safety of either percutaneous or surgical RV lead removal (or reimplantation)—determinations that are needed to predict either intervention's net benefit. Indeed, the limitations of 2-dimensional echocardiography and the unique and emerging potential for 3DE in diagnosing and characterizing ELTR are illustrated in our case and have previously been reported by others.[1,6,14] Three-dimensional echocardiography is able to simultaneously visualize all TV leaflets, their coaptation, and the precise course of the lead, which more clearly establishes the etiology and mechanism of TR.[1] Mediratta et al[14] reported that leads in commissural positions as identified by 3DE (as in our patient's case) were associated with less severe TR; however, their study included relatively few subjects with severe TR, the measure of TR severity used (vena contracta width) has not been defined for nonsevere TR, and details regarding patient symptoms or the indications for 3DE studies were not provided.

The findings in our case are consistent with severe TR due to entrapment of a TV leaflet rather than direct structural damage to the TV. However, indirectly structural changes to the TV apparatus occurred, as the hemodynamic consequences of the regurgitant volume likely resulted in right atrial and RV enlargement with accompanying tricuspid annular dilatation. Once this process reaches a critical stage, RV lead removal alone may not suffice, as significant TR may persist. Indeed, this mechanism has been put forth by others to explain a lack of echocardiographic or symptomatic improvement following percutaneous lead removal alone in certain cases.[9] The risk of developing adhesions between the lead and TV apparatus may further support early intervention, particularly if a percutaneous strategy is being considered, because once such changes occur, surgery may be the only option. However, though the benefits of surgically addressing functional TR and/or tricuspid annular dilatation at the time of mitral valve surgery is increasingly recognized, the role of surgery for isolated TR is not well established and the procedure is rarely performed.[3] Furthermore, though significant TR is often described as a progressive condition, data comparing surgical and nonsurgical management of TR are scarce,[3] and the natural history of residual TR after RV lead removal for ELTR, in particular, has not been studied.

Furthermore, contrary to the body of literature on LV reverse remodeling, there is comparatively little data on the time course of this process in the RV. Studies of surgical TV correction for TR have reported improved RV function within 6 months.[4] In our case, significant echocardiographic and symptomatic improvements occurred despite severe symptoms, RV dysfunction, and tricuspid annular dilatation,

but not until 10–12 months after lead extraction. This degree of improvement and delay have not been reported with percutaneous lead extraction alone and highlight the poorly understood pathophysiology and poorly defined optimal treatment of ELTR and of TR and RV dysfunction in general.

Of note, reduced RV filling pressures from aggressive diuresis likely contributed to our patient's improved TR. Additionally, AF has been hypothesized to be causally linked to mitral regurgitation and TR and though the clinical significance of this purported phenomenon is debated, it is possible that it played a role, as our patient was noted to be in an intermittently atrial-paced rhythm during her last echocardiogram.[15] The degree of TR resolution and echocardiographic features of the TV suggest that these were not primary contributors, however.

Conclusion

Percutaneous lead removal represents a minimally invasive alternative to surgery that should not be discounted as an option for ELTR. Given the low procedural risk associated with percutaneous options relative to surgery, it should be particularly considered in individuals with isolated ELTR early after device implantation, for poor surgical candidates, and in patients with relatively preserved TV structure. Clinicians should be aware that it may take >6 months for RV function to recover.

Appendix
Supplementary material

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.hrcr.2016.03.007.

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