HEART FAILURE

COMPLEX CASE STUDY

Pacemaker-associated Right Heart Failure: An Under-recognized Problem

ALEC VISHNEVSKY, MD, PRAVEEN MEHROTRA, MD, REGINALD HO, MD, MATTHEW V. DECARO, MD and DAVID L. FISCHMAN, MD

Division of Cardiology, Department of Medicine, Sidney Kimmel Medical College at Thomas Jefferson University, Philadelphia, PA

ABSTRACT. Patients presenting with right-sided heart failure represent a clinical challenge with numerous potential etiologies and limited treatment options. This report presents a challenging case involving right-sided heart failure due to persistent tricuspid regurgitation (TR) exacerbated by the placement of a right ventricular (RV) pacing lead after mitral valve repair. Ultimately, lead extraction resulted in decreased TR severity, improvement in RV function, and resolution of heart failure symptoms, highlighting the importance of prompt recognition of lead-associated TR.

KEYWORDS. pacemaker, right heart failure, tricuspid regurgitation.

Introduction

Patients presenting with right-sided heart failure represent a clinical challenge with numerous potential etiologies and limited treatment options. In particular, right-sided heart failure due to tricuspid regurgitation (TR) related to permanent pacemaker (PPM) or implantable cardioverter-defibrillator (ICD) leads is under-recognized.1–4 We present a patient with severe TR and right-sided heart failure occurring after implantation of a PPM following surgery for degenerative mitral valve regurgitation.

Case description

A 75-year-old female with a history of hypertension, rheumatic fever, and lung carcinoma secondary to a remote smoking history status post lung resection and radiation therapy presented with a chief complaint of progressive shortness of breath for 1 month. Her physical examination was remarkable for a holosystolic murmur at the apex. An electrocardiogram (ECG) revealed normal sinus rhythm with an incomplete right bundle branch block. An initial transthoracic echocardiogram revealed severe mitral regurgitation, mild TR, and normal left ventricular (LV) and right ventricular (RV) function. A transesophageal echocardiogram (TEE) confirmed the severity of mitral regurgitation and demonstrated diffuse mitral valve thickening and focal calcification. These changes were felt to be due to history of rheumatic fever and/or radiation-induced.

Given the clinical presentation and echocardiographic findings, she was referred for mitral valve repair. Her postoperative course was complicated by hypercarbic respiratory failure requiring bilevel positive airway pressure (biPAP) ventilation following extubation. She developed rapid atrial fibrillation treated with intravenous amiodarone. The patient subsequently developed bradycardia with a rate of 30 bpm. Because of prolonged conversion pauses and evidence of atrioventricular (AV) nodal disease, a dual-chamber pacemaker was implanted on postoperative day 7. Owing to persistent hypercarbia, she was discharged to a rehabilitation facility on oxygen and nocturnal biPAP. One month later, she was readmitted with urosepsis and hypercarbic respiratory failure, treated, and again discharged to a rehabilitation facility. Two months postoperatively, she was found to be in typical atrial flutter, with symptoms and signs suggestive of right-sided heart failure. A repeat transthoracic echocardiogram revealed moderate to severe TR with severe RV enlargement and decreased RV function. There was severe pulmonary hypertension and no significant
mitral regurgitation (Figure 1). The patient underwent successful atrial flutter ablation and was discharged home on amiodarone and anticoagulation. The patient was readmitted 1 month later with worsening right-sided heart failure and recurrent atrial flutter. Cardioversion initially restored sinus rhythm, but the flutter recurred 8 days later. A TEE was performed that showed malcoaptation of the tricuspid valve leaflets, although the exact course of the RV lead could not be ascertained. Device interrogation revealed that the patient was V-paced less than 1% of the time. Given persistent right-sided heart failure symptoms in the setting of severe TR and resolution of high-grade AV block, the patient underwent removal of the RV pacemaker lead in the event that this was contributing to the TR and RV dysfunction. Her device was adjusted to AAIR mode following the RV lead extraction, and she required atrial pacing when in sinus rhythm. She returned 1 month later with persistent right-sided heart failure. Following a prolonged hospital course, which included treatment for urinary retention, hypercarbic respiratory failure, and spontaneous bacterial peritonitis, she was discharged to hospice care. She remained on amiodarone, lasix, and metoprolol. Eighteen months later, she was discharged from hospice care due to a dramatic and unexpected improvement in her functional status. At follow-up, she reported good exercise capacity and exhibited no signs or symptoms of right-sided heart failure. An ECG revealed normal sinus rhythm with a heart rate of 80 bpm. A repeat echocardiogram revealed a dramatic improvement in her RV function and TR (Figure 2).

Discussion

TR is a common echocardiographic finding, with approximately 1.6 million people in the United States affected by moderate to severe TR.1,2 TR is often functional or “secondary” to tricuspid annular dilation, resulting from left heart failure, RV volume or pressure overload, or any other process causing dilation of cardiac chambers.3 PPM or ICD lead-induced TR and right heart volume overload is an under-recognized but clinically important etiology of right heart failure.4 This association has become increasingly important over the past few decades as the implantation of cardiac implantable electronic devices has increased due to expanding indications and increased life expectancy. The overall prevalence of TR in patients with PPM is between 25% and 29%. Numerous studies have found worsening of pre-existing TR by 1–2 grades in up to 25% of patients with newly implanted devices.2 This is more common with implantable cardiac defibrillators than permanent pacemakers due to differences in lead size.3

**Figure 1:** Transthoracic echocardiogram post-pacemaker implantation demonstrating a dilated right ventricle with systolic malcoaptation of the tricuspid valve (arrow) in the presence of a right ventricular lead (arrow head) (A), and severe tricuspid regurgitation (B).

**Figure 2:** Transthoracic echocardiogram following removal of the right ventricular (RV) pacemaker lead demonstrating normal RV size with normal systolic coaptation of the tricuspid valve leaflets (A), and minimal tricuspid regurgitation (B).
A recent retrospective cohort study by Lee et al. demonstrated that right atrial area, RV systolic pressure, and the presence of moderate or greater mitral regurgitation (MR) were all correlated with an increase in TR of 2 grades or more. Although cardiac resynchronization therapy (CRT) with biventricular pacing has been shown to improve TR due to dyssynchrony, lack of response to CRT can be attributed to worsening of the TR, possibly due to the lead implant itself. Moreover, new-onset TR or worsening of existing TR can occur up to 7 years following device implantation, highlighting the importance of maintaining a high index of suspicion in patients who have had devices for many years.

Although current literature regarding lead-related TR following PPM or ICD implantation is limited, surgical and autopsy series have elucidated potential mechanisms. Lin et al. examined the characteristics of 41 patients with autopsy confirmed, lead-related severe TR, and observed that lead impingement and lead adherence were the most common causes. Frank leaflet perforation and lead entanglement with the valve or chords occurred less commonly. Other studies have attributed worsening TR to active RV pacing that causes valve dysfunction either through delayed RV activation or alterations in RV geometry. Seo et al. observed that lead associated mild to moderate TR occurs often when the lead is placed between the posterior and septal leaflets, but more severe TR is seen when the leads impinge on the tips of the tricuspid valve leaflets.

Diagnosing lead-induced TR can be challenging. Transthoracic echocardiography has a low sensitivity for detecting TV pathology due to lead positioning, while two-dimensional transesophageal echocardiography only improves the sensitivity to approximately 45%. Appreciating the relationship between the tricuspid valve and the RV lead may be difficult due to limited visualization of the posterior leaflet and acoustic shadowing due to leads. Real-time three-dimensional (3D) echocardiography, however, can provide unique en face views of the tricuspid valve that allows better assessment of TV anatomy and leaflet function as it relates to device leads. Seo et al. demonstrated that 3D echocardiography can identify the lead position at the valve apparatus in up to 94% of patients. Additionally, Mediratta et al. demonstrated by 3D echocardiography that a commissural or central position of the pacemaker lead is associated with significantly less TR. Despite a diagnosis of lead-induced severe TR, extraction is not routinely recommended due to procedural risks, especially if the leads are adherent to the valve leaflets.

Current valvular guidelines recommend concomitant TV repair in the setting of mild to severe TR when there is evidence of tricuspid annular dilation or prior evidence of right-sided heart failure. This recommendation is based on the observation that in approximately 25% of patients even mild to moderate TR can progress following repair of left-sided valvular lesions, especially in the setting of atrial fibrillation, pulmonary hypertension, or right-sided PPM or ICD leads. Furthermore, the presence of TR in the postoperative period following mitral valve surgery may be more common in those with repaired functional or ischemic mitral regurgitation compared to those with degenerative (primary) mitral valve regurgitation.

We hypothesize that in the context of existing pulmonary hypertension related to lung disease and significant mitral regurgitation, the addition of an RV lead contributed to the incompetence of the TV leading to worsening regurgitation, persistent RV dysfunction, and right-sided heart failure. Once the lead was removed, the presence of atrial flutter with associated tachycardia may have delayed RV recovery until she converted into a paced atrial rhythm while in hospice. Possible mechanisms for spontaneous flutter conversion include the presence of a partial flutter line from the prior ablation, continued use of amiodarone, and a decrease in size of the right atrium as her TR severity decreased and right heart failure improved. With time and appropriate medical management, her RV recovered and right-sided heart failure resolved.

**Conclusion**

This report presents a challenging case involving multifactorial right-sided heart failure due to persistent TR exacerbated by the placement of a RV pacing lead after mitral valve repair. Ultimately, lead extraction resulted in decreased TR severity, improvement in RV function, and resolution of heart failure symptoms. Our case highlights the importance not only of prompt recognition of lead-associated severe TR which is under-recognized, but also of maintaining a broad differential to optimize outcomes in patients with right-sided heart failure. Lastly, the advent of subcutaneous ICDs and leadless pacemakers provides a new technology that will enable therapy without interference with the tricuspid valve apparatus. This will play an important role in similar patients with significant tricuspid regurgitation who require device therapy.

**References**


