Permanent Pacemaker Lead Induced Severe Tricuspid Regurgitation in Patient Undergoing Multiple Valve Surgery

Jung Hee Lee, M.D., Tae Ho Kim, M.D., Wook Sung Kim, M.D., Ph.D.

Severe and permanent tricuspid regurgitation induced by pacemaker leads is rarely reported in the literature. The mechanism of pacemaker-induced tricuspid regurgitation has been identified, but its management has not been well established. Furthermore, debate still exists regarding the proper surgical approach. We present the case of a patient with severe tricuspid regurgitation induced by a pacemaker lead, accompanied by tricuspid valve disease. The patient underwent double valve replacement and tricuspid valve repair without removal of the pre-existing pacemaker lead. The operation was successful and the surgical procedure is discussed in detail.

CASE REPORT

A 69-year-old female was referred to Samsung Medical Center for progressive dyspnea and chest discomfort. A year before her referral, she had developed exertional dyspnea and general weakness. She had been diagnosed with severe aortic stenosis with regurgitation, moderate mitral regurgitation, and severe tricuspid regurgitation (TR) before presenting at Samsung Medical Center. Her medical history revealed that a single chamber transvenous permanent pacemaker (VVI mode) had been implanted 14 years prior due to sick sinus syndrome. Repeat echocardiography confirmed severe aortic stenosis, moderate aortic regurgitation, moderate mitral regurgitation, and severe TR. On her aortic valve leaflet, thickening, calcification, and retraction with motion limitation were present. The aortic valvular area was 0.87 m² and the mean pressure gradient was 41 mmHg. In addition, the posterior mitral valve leaflet showed limited motion accompanied by moderate mitral regurgitation. These findings suggested a rheumatic etiology of the dysfunction in the two valves. However, the severity of this patient’s TR was not fully understood, since only annular dilatation secondary to left-sided lesions was observed (Fig. 1). A pacemaker lead around the tricuspid valve was found on echocardiography, but we were not able to determine its significance. Her severe TR seemed to be related to coaptation failure in the tricuspid valve, but the coaptation failure was not serious enough to explain the severity of the TR. Further surgical analysis of her tricuspid valve was necessary. Nevertheless, echocardiography results suggested triple valve disease, for which surgical cor-
Fig. 1. Preoperative echocardiography. Severe tricuspid regurgitation is demonstrated. (A) Apical 2 chamber view and (B) with color doppler are showing pacemaker lead and severe tricuspid regurgitation. (C) Subcostal 2 chamber view and (D) apical 4 chamber view are showing regurgitant jet toward atrial septum suggesting organic origin of tricuspid regurgitation. RA, right atrium; RV, right ventricle.

Fig. 2. Tricuspid valve analysis before repair. TR, tricuspid regurgitation; PM, pace maker; IVC, inferior vena cava.

rection was indicated.

There were no abnormal values in her preoperative blood tests, but electrocardiography showed atrial fibrillation with a slow ventricular response and an intermittent ventricular pacing rhythm. Her native heart rate was 30 to 60 beats per minute. Cardiomegaly was apparent on a chest X-ray, and a coronary computed tomography angiogram showed no signs of coronary artery disease.

The operation was carried out through a median sternotomy with cardiopulmonary bypass support. After the induction of cardioplegic arrest, tricuspid valve analysis was performed. The pacemaker lead was found inside the right atrium, stretching from the tricuspid annulus toward the right ventricular cavity. The mechanism of severe TR resulted from
Tricuspid Valve Repair in Patient with Pacemaker Lead Induced Severe TR

three processes: impingement of the pacemaker lead, entrapment, and tricuspid annular dilatation (Fig. 2). Tricuspid annular dilatation was noted together with the impingement of the pacemaker lead into the right third of the septal leaflet. As well, lead entrapment was noted on the anterior papillary muscle, limiting the motion of the septal and anterior leaflets. Two decisions had to be made at this point. First, we had to decide whether to leave the pacemaker lead inside the heart chamber or implant new epicardial pacemaker leads. Second, we had to decide whether to repair her tricuspid valve or replace it. Fundamentally, this patient needed to maintain a permanent pacemaker due to her intractable sick sinus syndrome. With this in mind, it seemed significantly preferable to retain her transvenous pacemaker, because epicardial pacemakers have a relatively high stimulation threshold and require frequent battery changes. Moreover, her tricuspid valve morphology seemed repairable; as such, repairing her tricuspid valve was an obvious choice in light of the morbidity and mortality involved in triple valve replacement. Therefore, we decided to leave the pacemaker lead in place and to repair the tricuspid valve.

First, we had to release the pacemaker lead from the valve and subvalvar apparatus. The pacemaker lead that impinged on the septal leaflet was detached by leaflet slicing without injuring the leaflet. The pacemaker lead attachment on the anterior papillary muscle was also released (Fig. 3A). Subsequently, the pacemaker lead was moved to the posteroseptal annulus and placed between two Lembert-type

Fig. 3. Tricuspid valve repair. (A) Septal leaflet and papillary muscle were detached from pacemaker lead. (B) Lead inclusion into posterior septal annulus with horizontal mattress sutures. (C) Anchoring pacemaker lead at anterior papillary muscle. (D) Tricuspid annuloplasty with Duran AnCore Annuloplasty band (Medtronic Inc., Minneapolis, MN, USA) 29 mm. IVC, inferior vena cava.
pledge-buttressed interrupted horizontal mattress sutures, in order to ensure that it was located inside the posteroseptal annulus (Fig. 3B). Tethering of the leaflet did not take place after this procedure; instead, the size of the annulus was reduced. However, the pacemaker lead was still not firmly in place and wriggled from the tricuspid opening toward the right atrial cavity. Therefore, we pushed the pacemaker lead inside the ventricle and anchored it loosely to the anterior papillary muscle, using a 5-0 polypropylene simple interrupted suture (Fig. 3C). A TR test using saline confirmed that the motion of the leaflet was not limited, so we proceeded to place an annuloplasty band using a 29-mm Duran AnCore Annuloplasty Band (Medtronic Inc., Minneapolis, MN, USA) (Fig. 3D). Another TR test using saline confirmed the absence of TR. In turn, mitral valve replacement and aortic valve replacement were performed and the patient was weaned off cardiopulmonary bypass. After terminating the cardiopulmonary bypass, intraoperative transesophageal echography confirmed that no TR was present. The operation was completed and she was transferred to the intensive care unit. The day after surgery, she was extubated, and she was moved to the general ward on the second postoperative day. Postoperative echocardiography was performed five days after surgery, finding minimal TR and good functional recovery. She was discharged nine days after surgery after warfarinization was completed. Currently, she remains asymptomatic in outpatient follow-up examinations.

**DISCUSSION**

Pacemaker-induced TR is not a rare disease. The prevalence of TR in patients with permanent pacemakers has been reported as 25% to 29%. New TR or aggravated pre-existing TR can develop within seven years after the implantation of the transvenous devices [1]. Few case reports or case series have been published, but most previously published reports present cases of mild to moderate TR. Severe TR induced by pacemaker leads is uncommon [2].

Multiple mechanisms have been reported for the pathogenesis of TR induced by pacemaker leads. It may result from mechanical causes that impair closure, such as scar formation or thrombus on the leads, although the perforation or laceration of the valve leaflets is another cause of TR. Another mechanism is asynchrony, occurring when a pacemaker causes abnormal right ventricle activation [3,4]. Lin et al. [5] found that the mechanism of TR after pacemaker implantation in 41 patients was lead impingement in 39%, lead adherence in 34%, lead perforation in 17%, and lead entanglement in 39%.

The management of TR induced by pacemaker leads is not well-established. Nonetheless, it can be managed according to general guidelines. Surgical correction is clearly more necessary in such cases than in cases of moderate TR associated with left heart lesions. As in the case described above, an important decision must be made in such circumstances: whether the pacemaker leads should be left in situ or removed and replaced with newer epicardial pacemaker leads.

Lin et al. [5] reported no reoperations due to recurrent TR during a mean 8.2-year follow-up period after TR repair without lead extraction. In addition, no residual TR or recurrent TR occurred during the follow-up period. However, they did not provide a detailed description of the surgical approach used in TR repair without lead extraction. In contrast, McCarthy et al. [6] found that the presence of a pacemaker lead has been shown to be a significant risk factor for late recurrent TR after tricuspid valve repair. They concluded that trans-tricuspid pacing leads should be explanted and replaced with epicardial leads during tricuspid valve repair surgery. However, the details of the specific tricuspid valve pathology were not recorded along with the surgical procedures, making the results somewhat challenging to interpret.

In patients who require tricuspid valve replacement, removal of the pacemaker lead with the placement of an epicardial lead is routinely recommended [7]. However, epicardial pacemakers have a relatively high stimulation threshold and require frequent battery changes. Another option is to place the pre-existing pacemaker lead between the sewing ring and the native annulus [8] or to place it inside the posteroseptal annulus with Lembert-type sutures as described above. However, such techniques make lead removal difficult in the future, if removing the lead is indicated due to an infection caused by the pacemaker lead.

In conclusion, functional TR caused by left heart disease can be repaired, even if it is caused by a pacemaker lead.
Tricuspid valve repair without extraction of the pacemaker lead resulted a good immediate postoperative outcome and is a viable surgical option to treat pacemaker-induced TR. However, a careful estimation should be made of the risks and benefits of leaving the pacemaker lead in situ, and subsequent studies should be performed to assess the long-term outcomes of this procedure.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

REFERENCES


