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Three-dimensional Echocardiographic Evaluation of Severe Tricuspid Regurgitation due to Leaflet Damage by Endocardial Pacing Lead

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LEE ET AL.: Three-dimensional Echocardiographic Evaluation of Severe Tricuspid Regurgitation due to Leaflet Damage by Endocardial Pacing Lead. A 76-year-old woman developed congestive heart failure within a year following permanent pacemaker implantation. She was found to have moderate to severe functional mitral regurgitation and severe tricuspid regurgitation. However, two-dimensional echocardiography was unable to delineate the impact of pacing lead on tricuspid regurgitation. Subsequent three-dimensional echocardiography visualized that the pacing lead had passed through the tricuspid septal leaflet causing severe regurgitation. This finding was confirmed during successful mitral and tricuspid repair. (J HK Coll Cardiol 2014;22:5-8)

Echocardiography, Pacing lead, Tricuspid regurgitation, Valve injury, Valve repair

Case report

A 76-year-old woman with background history of atrial fibrillation had repeated episodes of non-sustained ventricular tachycardia and associated syncope, which even led to head injury with radiological evidence of subdural and subarachnoid hemorrhage. Electrophysiological investigation demonstrated inducible ventricular tachycardia and long pause (>4 seconds). Thus a single-chamber VVIR permanent pacemaker (St Jude Medical, St Paul, MN, USA), with single transvenous right ventricular pacing lead, was implanted for her. She had no previous history of heart failure and her echocardiogram before pacemaker implantation showed well-preserved left ventricular function, without significant valvular problem.

Within one year after the pacemaker insertion, she gradually developed congestive heart failure. Transthoracic echocardiography (TTE) showed impaired left ventricular function (left ventricular ejection fraction = 38%), moderate to severe functional mitral regurgitation, and severe tricuspid regurgitation (TR). However, the mechanism of pacemaker lead causing severe TR was not directly visualized on two-dimensional (2D) TTE imaging (Figure 1). Subsequent three-dimensional (3D) TTE revealed the pacing lead was "stuck" to the septal leaflet of the tricuspid valve, raising the suspicion of pacing lead damage of the valve.
as the cause of severe TR (Figure 2). Her preoperative coronary angiogram confirmed normal findings.

Mitral and tricuspid valves repair was then performed through standard median sternotomy, with the application of cardiopulmonary bypass. Following anesthetic induction, 3D transesophageal echocardiography demonstrated clearly the pacing lead passed through the body of the tricuspid septal leaflet (Figure 3), hindering its excursion and causing organic regurgitation. Surgical inspection through right atriotomy confirmed the perforation of the tricuspid septal leaflet by the pacing lead (Figure 4). The lead was surgically freed from the tricuspid valve and the septal leaflet perforation was repaired with Gore-Tex sutures. The tricuspid valve was stabilized by an annuloplasty using a Carpentier-Edwards MC₃ ring

**Figure 1. Preoperative two-dimensional transthoracic echocardiography imaging.**

**Figure 2. Preoperative three-dimensional transthoracic echocardiography imaging.**
The endocardial pacing lead was not removed as the intraoperative test confirmed satisfactory pacing function. The mitral valve was also repaired with a "down-size" annuloplasty using a Rigid Saddle ring (St Jude Medical, St Paul, MN, USA). Postoperative 3D transesophageal echocardiography confirmed competent mitral and tricuspid valve closure (Figure 5). The patient had an uneventful recovery. Her follow-up echocardiography 3 months after the operation showed trivial TR only, with much improved bi-ventricular function.

Discussion

Endocardial pacing lead-induced TR has not been widely documented, either clinically or echocardiographically. However, this complication is expected to become increasingly important owing to the worldwide aging trend and the expanding capabilities of pacing devices or the implantable cardioverter-defibrillators. In severe cases such as the present one, it can result in congestive heart failure and tricuspid valve surgery would be unavoidable. Although the underlying mechanisms and the time course of the development of TR remain largely unclear, significant lead-induced TR was observed in 38% of patients 1-1.5 years following lead placement. More importantly, such type of TR was independently associated with much worsened long-term survival. Previously it was believed by many that a blunt-tipped pacing lead can hardly perforate valve leaflet edge particularly due to the mobility of the leaflet. Hence, it was even proposed that the pacing lead may pass through "a natural hole" on the leaflet, instead of truly penetrates it. Nevertheless, our intra-operative finding does not support such skepticism. Moreover, in a recent report, the pacing lead-induced leaflet damage was identical as in the
current case. A high index of suspicion for direct lead-induced valvular injury is essential to early diagnose this specific pathological condition and to limit its long-term consequences.

It has been recognized that the mechanism and the severity of endocardial lead-induced TR may not be well evaluated by 2D echocardiography. Real-time 3D echocardiography appears to be a promising technique to appraise the mechanism of TR and may allow the early detection of patients who will develop severe lead-induced TR. Our current case illustrated how 3D echocardiographic imaging was useful to clearly delineate the location of the pacing lead and its impact on the tricuspid valve. Even though the worsened heart failure in this particular case may not be solely attributed to pacing lead-induced damage, severe TR was definitely the most important contributor to her deteriorated cardiac function and symptoms. For determining surgical indication and to plan the appropriate intervention, it would be extremely helpful to appreciate this rare etiology preoperatively. Obviously, a better understanding of the mechanism of lead-induced TR will also be essential to the future development of preventive strategies.

Declaration of Interest

All authors have no conflict of interest.

References