Diagnosis of lead-induced tricuspid regurgitation

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Introduction

Clinically important lead-induced tricuspid regurgitation (LITR) is an uncommon complication following cardiac device implantation. Surgical removal and replacement of a lead may improve heart failure caused by LITR.[1] Percutaneous lead extraction and reimplantation may also improve or correct LITR.[2] However, whether severe tricuspid regurgitation (TR) is caused by a lead is often unclear, especially in patients with left ventricular (LV) dysfunction; worsening heart failure in such patients is most often attributed to progression of LV dysfunction.[1–3]

Echocardiography may clarify whether worsening heart failure is due to LITR. It is only occasionally obvious that a lead is the culprit.[1,3] However, other findings may still implicate the lead. Right heart failure disproportionate to that expected for the degree of LV dysfunction should suggest possible LITR. In left heart failure, LV end-diastolic pressure (LVEDP) is expected to be greater than right ventricular (RV) end-diastolic pressure (RVEDP): the positive transseptal pressure gradient (LVEDP − RVEDP) would cause the ventricular septum to be concave toward the LV.[4,5] If the ventricular septum is flattened, the transseptal pressure gradient is close to zero or negative; that is, RVEDP is greater than or equal to LVEDP. This should suggest other possible causes of RV failure, including LITR.

Our experience suggests that the reasoning described above can help determine whether the lead is responsible for, or contributes to, severe TR and that percutaneous lead reimplantation may be beneficial.

Methods

Percutaneous lead replacement for presumed LITR was performed in 6 patients between July 2001 and July 2012. Echocardiograms were reviewed to assess biventricular function, lead relationship to the tricuspid valve (TV), end-diastolic ventricular septal flattening (VSF), and estimated peak systolic pulmonary artery pressure.

Results

Table 1 lists the clinical characteristics of the 6 patients and the devices used. Table 2 summarizes the outcomes. LITR was verified in 4 patients (cases 1, 2, 4, and 6) by substantial clinical improvement. Case 5 needed an open TV repair because a leaflet was torn during lead extraction. In case 3, the cause of heart failure remained unclear. VSF was present in the 5 available echocardiograms.

Case 1

A 68-year-old male patient with moderate LV dysfunction and prior coronary artery bypass surgery had a pacemaker implanted in 2006, which was upgraded to a cardiac resynchronization therapy with defibrillator device in 2009. He subsequently developed heart failure resistant to medical therapy.

Echocardiography revealed severe TR, but the effect of the lead was unclear. The estimated peak RV systolic pressure was 33 mm Hg; there was mild RV dysfunction.

Initially, the implantable cardioverter-defibrillator (ICD) lead was removed percutaneously and a defibrillator coil was placed in the azygous vein. The abandoned pace/sense lead was not removed. TR and heart failure remained severe, and 3 months later, the pace/sense lead was replaced percutaneously. TR was improved, his heart failure was easily controlled without diuretics, and he returned to active living. Follow-up echocardiography showed normal ventricular septal shape.

Case 2

A 72-year-old male patient (LV ejection fraction 12%) with prior coronary artery bypass surgery and a pacemaker presented 6 months later with severe right heart failure. There was severe TR with clear lead impingement, an estimated peak RV systolic pressure of 51 mm Hg, mild-to-moderate RV dysfunction, and VSF.

The lead was replaced percutaneously 11 months following the original implantation. He lost 5.6 kg within 4 days. He remains stable 6 years later without diuretics. An

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KEY TEACHING POINTS

- Lead-induced tricuspid regurgitation is underrecognized, and increased vigilance is needed by health care professionals managing patients with cardiac rhythm management devices.
- Echocardiography may be a valuable tool for establishing lead-induced tricuspid regurgitation.
- Contrary to traditional teaching, patients with lead-induced tricuspid regurgitation, even with leads that are a few years old, may still benefit from removal of the lead.

echocardiogram recorded 2 years postrevision showed moderate TR and normal ventricular septal shape.

Case 3

A 79-year-old male patient with mitral regurgitation and a pacemaker presented 6 years later with severe heart failure and TR, most likely a ruptured TV chord, moderate-to-severe mitral regurgitation, normal LV systolic function, an estimated peak RV systolic pressure of 54 mm Hg, and mild RV dysfunction. There was VSF.

He underwent percutaneous lead replacement; intraoperative transesophageal echocardiography did not clarify the cause of TR. Subsequently, TR and VSF appeared unchanged, and the patient died several months later. The cause of the persistent heart failure was not clarified.

Case 4

An 82-year-old male patient with a previous mitral valve repair had an ICD implanted after a cardiac arrest. The device was later upgraded to a dual-chamber ICD, and the RV lead was replaced. He developed progressive heart and renal failure requiring frequent hospitalizations. A furosemide infusion (8 mg/h) caused a loss of 24 kg, but severe TR persisted.

LV function was moderately reduced; there was mild-to-moderate mitral regurgitation, an estimated peak RV systolic pressure of 56 mm Hg, moderate RV dysfunction, and VSF.

After percutaneous lead replacement, TR was mild to moderate with normalization of septal shape. A furosemide infusion (2 mg/h) caused loss of another 5.9 kg following the procedure. The serum creatinine level improved from 350 to 140 μmol/L. Subsequently, he required furosemide 20 mg twice daily.

Case 5

A 70-year-old female patient had a pacemaker implanted and a new RV lead was placed 4 years later for lead failure; the old lead was abandoned. She developed severe right heart failure 10 years later.

Echocardiography showed moderate-to-severe aortic and mitral regurgitation with normal LV size and systolic function. There was severe TR, with the lead impinging on the posterior tricuspid leaflet, an estimated peak RV systolic pressure of 70 mm Hg, and normal RV systolic function. Images were not available for review, and septal shape was not described in the report.

After percutaneous lead replacement, TR was mild to moderate and there was spontaneous diuresis in hospital with no alteration in diuretic dose. The follow-up echocardiogram was not available to assess septal shape. She remained asymptomatic 5 years later.

Case 6

A 24-year-old female patient had a pacemaker implanted at the age of 5 years. Eleven years later, the lead failed and she had a passive fixation VDD pacemaker implanted. Severe TR was diagnosed 7 years later. There was no history of congenital or acquired TV disease; there was minimal TR before lead insertion.

LV size and function were normal; there was severe TR, RV dilation (RV basal diameter 6.1 cm) with normal RV function, and estimated pulmonary artery pressure. There was VSF. A lead impinged on the septal leaflet. The chest radiograph suggested that the 2 leads were at opposite sides of the tricuspid annulus, possibly "spreading" the leaflets apart (Figure 1).

She underwent lead reimplantation of a new DDDR system. However, a leaflet of the valve became flail. She remained well, not requiring any diuretics, but developed

Table 1 Patient characteristics and devices used

Case no.	Age (y)	Sex	Clinical diagnosis	Device	No. of leads	Lead type	Years from insertion to removal
1	68	M	Ischemic CMP; CHB	CRT-D	2	5076 AF 7122 AF	6.2 4.3
2	72	M	A. fib; bradycardia	VVI PPM	1	5076 AF	0.9
3	79	M	A. fib; bradycardia	VVI PPM	1	4088 AF	6.8
4	82	M	Mitral valve repair; VF	DDD ICD	1	0181 AF	4.2
5	70	F	SSS	VVI PPM	2	4082 PF 430-10 PF	10.2 6.4
6	24	F	CHB	VDD PPM	2	5038 PF U	8.4 19

AF = active fixation; A. fib = atrial fibrillation; CHB = complete heart block; CMP = cardiomyopathy; CRT-D = cardiac resynchronization therapy with defibrillator; F = female; ICD = implantable cardioverter-defibrillator; M = male; PF = passive fixation; PPM = permanent pacemaker; SSS = sick sinus syndrome; U = unknown lead type; VF = ventricular fibrillation.