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CASE REPORT
Other

Pacing lead extraction in the management of tricuspid regurgitation: a case report

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Background Patients with a cardiac implantable electronic device (CIED)-induced tricuspid regurgitation (TR) have an increased mortality and morbidity. However, the impact of CIED-lead extraction and its indications are not well-defined.

Case summary A 69-year-old woman presented with recurrent hospital admissions for right heart failure refractory to medical therapy, on the background of a single-chamber permanent pacemaker (Biotronik) implanted 6 years ago for tachycardia–bradycardia syndrome. Transoesophageal echocardiography identified severe TR which was predominantly CIED-induced from a lead impingement of the posterior tricuspid valve (TV) leaflet preventing adequate leaflet coaptation. This had progressed to cause a degree of secondary functional TR. The patient underwent pacing lead extraction followed by epicardial lead placement via minithoracotomy, with significant symptomatic and echographic improvement of TR.

Discussion CIED-induced TR from a lead impingement of TV leaflets carries the highest risk of TR and its consequences. This case illustrates the significance of the relationship between CIED-leads and the TV, which impacts management strategy. We recommend a mechanistic approach and incorporating CIED-lead interaction with the TV apparatus as the underlying principle in developing future management guidelines for CIED-induced TR.

Keywords Tricuspid regurgitation • Cardiac implantable electronic devices • Pacing lead extraction • Lead impingement • Case report

ESC Curriculum 4.5 Tricuspid regurgitation • 5.9 Pacemakers • 6.7 Right heart dysfunction • 2.2 Echocardiography

Learning points

- Cardiac implantable electronic device (CIED)-lead impingement could lead to mechanical obstruction to tricuspid valve leaflets causing moderate to severe tricuspid regurgitation (TR).
- Severe TR from CIED-lead impingement is unlikely able to be adequately managed with medical therapy alone, in the absence of lead extraction.
- Given the high-risk nature of CIED-lead extraction, an intricate understanding of the relationship between CIED-lead and tricuspid valve is paramount in management strategy.

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Introduction

It is well established that patients with cardiac implantable electronic devices (CIEDs) are more than twice as likely to have moderate to severe tricuspid regurgitation (TR) in comparison with patients without CIEDs.[1] They have an increased risk of mortality and heart failure hospitalizations.[2] This is broadly divided into primary lead-related or CIED-induced TR, whereby the CIED-lead interferes with tricuspid valve (TV) leaflet mobility or coaptation causing TR; and secondary functional or CIED-associated TR, whereby TR begets TR with annular and right ventricular (RV) dilatation.[3]

CIED-induced TR can be due to iatrogenic damage of the TV apparatus, mechanical interference with TV leaflet mobility and coaptation, as well as pacing-related RV dyssynchrony.[4] Due to a paucity of prospective studies in CIED-induced and CIED-associated TR, current Heart Rhythm Society (HRS) guidelines do not weigh in on lead extraction in these situations.[5]

We hereby report a case of successful transvenous lead extraction in severe symptomatic TR secondary to CIED-induced TR. The patient experienced significant improvement of symptoms, echocardiographic parameters, and quality of life.

Timeline

Date	Progress
1984	Mechanical mitral valve replacement (Starr–Edwards) for rheumatic mitral stenosis
October 2014	Single-chamber transvenous permanent pacemaker (Biotronik) implanted for tachycardia-bradycardia syndrome with underlying chronic atrial fibrillation
September 2020	Recurrent decompensated right heart failure refractory to medical therapy
October 2020	Transoesophageal echocardiogram demonstrates normal mechanical mitral valve function, and CIED-lead impingement of TV posterior leaflet as predominant cause of TR
November 2020	CIED-lead extraction with epicardial lead placement via minithoracotomy
January 2021	Sustained symptomatic and echographic improvement of TR

Case presentation

A 69-year-old woman presented with New York Heart Association (NYHA) Class III dyspnoea, and recurrent hospital admissions for decompensated right heart failure refractory to medical therapy. Her comorbidities included a mechanical mitral valve replacement 36 years ago (Starr–Edwards valve) for rheumatic mitral stenosis, transvenous single-chamber permanent pacemaker (Biotronik) 6 years prior for tachycardia–bradycardia syndrome, permanent atrial fibrillation, and well-controlled asthma. Physical examination revealed predominantly signs of right heart failure, in particular jugular venous pressure up to the angle of her jaw with prominent v-waves, pansystolic murmur with a pulsatile liver and peripheral pitting oedema, consistent with severe TR. Her medications were Furosemide 80 mg twice daily, Spironolactone 25 mg daily, Acetazolamide 125 mg twice daily, Digoxin 125 µg daily, Atorvastatin 20 mg daily, Warfarin, and asthma inhalers. Blood test results including liver function tests were normal.

Transthoracic echocardiogram (TTE) confirmed severe TR with inferior vena cava plethora, and systolic flow reversal in the hepatic vein. The RV was severely dilated up to 5.2 cm basal diameter with reduced systolic function (*Figure 1*). The mechanical mitral valve was well seated with no regurgitation. There was diastolic septal flattening from RV volume overload, but the left ventricular systolic function was preserved. Biatria were severely dilated. Transoesophageal echocardiogram (TOE) demonstrated severe TR predominantly due to transvenous pacing lead impingement of the posterior leaflet causing inadequate leaflet coaptation, with an element of secondary functional TR from annular dilatation (*Figures 2–4*; see Supplementary material online, *Videos S1 and S2*).

Coronary angiogram showed minor coronary artery disease. Right heart catheterization showed mild post-capillary pulmonary venous hypertension (mean pulmonary artery pressure 29 mmHg), with raised pulmonary capillary wedge pressure (21 mmHg) and normal transpulmonary gradient (8 mmHg). Prominent v-waves were evident on right atrial (RA) pressure waveform tracings, with RA pressure higher than RV pressure (RA pressure 15 mmHg, RV pressure 12 mmHg), consistent with severe TR. Her cardiac output was preserved at 4.41 L/min (cardiac index 3.14 L/min) by indirect Fick's method. Pacemaker interrogation revealed ventricular-pacing 60–65% of the time in underlying chronic atrial fibrillation.

Based on TOE, the Structural Heart Team hypothesized that the predominant cause of TR was lead impingement preventing adequate coaptation of the TV leaflets. Therefore, decision was for her to undergo lead extraction to reduce TR severity, and to prevent progressive TV annular dilatation. Her EuroSCORE II surgical mortality risk was estimated at 6.2%.

She underwent successful lead extraction with the aid of a 12-French laser along the TV and lead insertion point into the myocardium with no postoperative complications. Intraoperative TOE showed immediate significant reduction in tricuspid regurgitant volume (by approximately 50%). A new epicardial lead was tunnelled up via left minithoracotomy.

The patient experienced significant symptomatic improvement sustained months after lead extraction. While her diuretic therapy remained unchanged, she transitioned from NYHA Class III to Class I, with resolution of early satiety from hepatic congestion. Her jugular venous pressure and precordium examination remained consistent with moderate TR; however, she no longer experienced peripheral oedema. Three-month follow-up TTE showed residual moderate functional TR, and reduction of RV base diameter from 5.2 to 4.3 cm (*Figure 5*; see Supplementary material online, *Video S3*).

Discussion

This case reinforces the hypothesis that CIED-induced TR from direct lead impingement of TV leaflets preventing adequate coaptation leads to TR that is most significant. This is followed by a CIED-lead

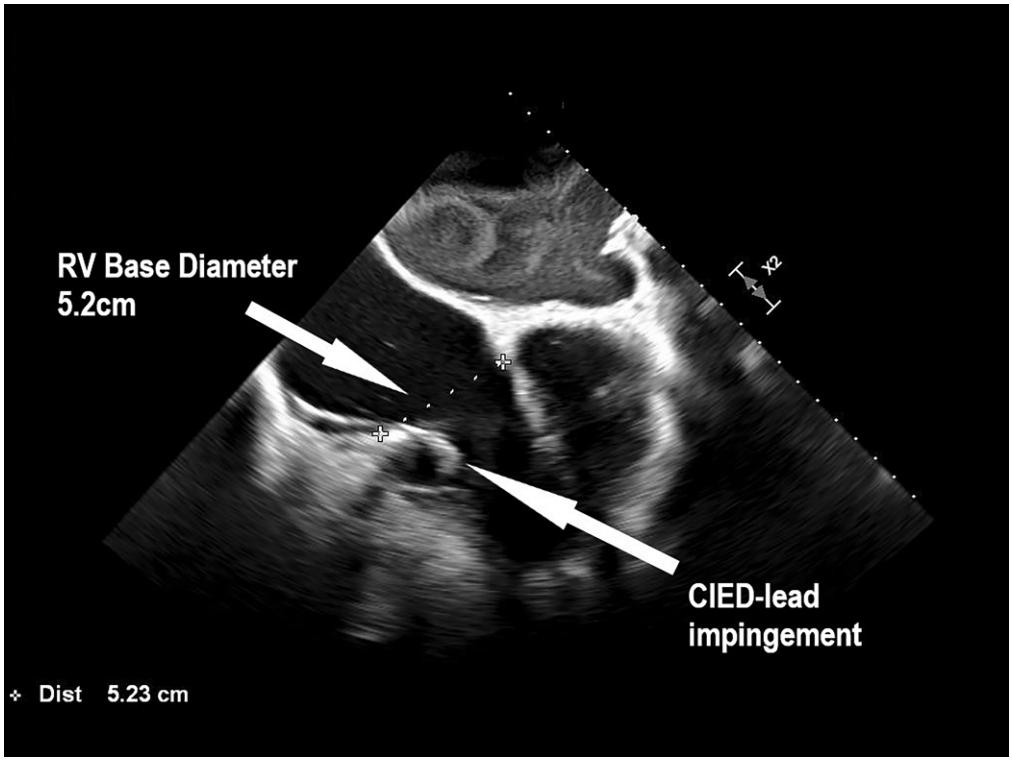

Figure 1 Transoesophageal echocardiogram showing a dilated right ventricle.

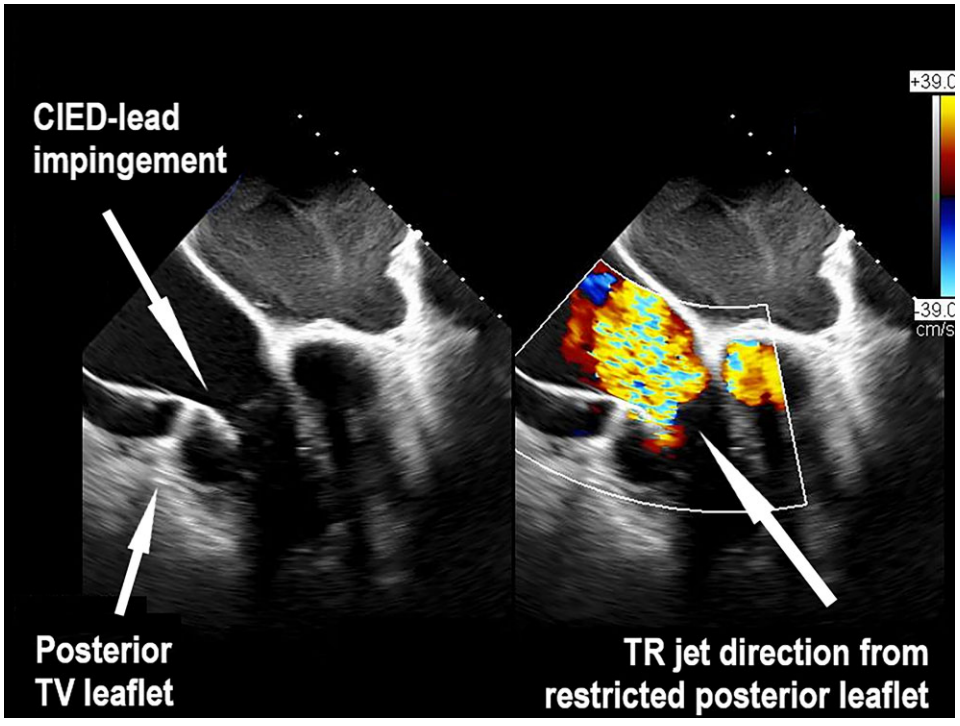

Figure 2 Transoesophageal echocardiogram showing tricuspid regurgitant jet direction going away from the posterior leaflet restriction by cardiac implantable electronic device-lead impingement.

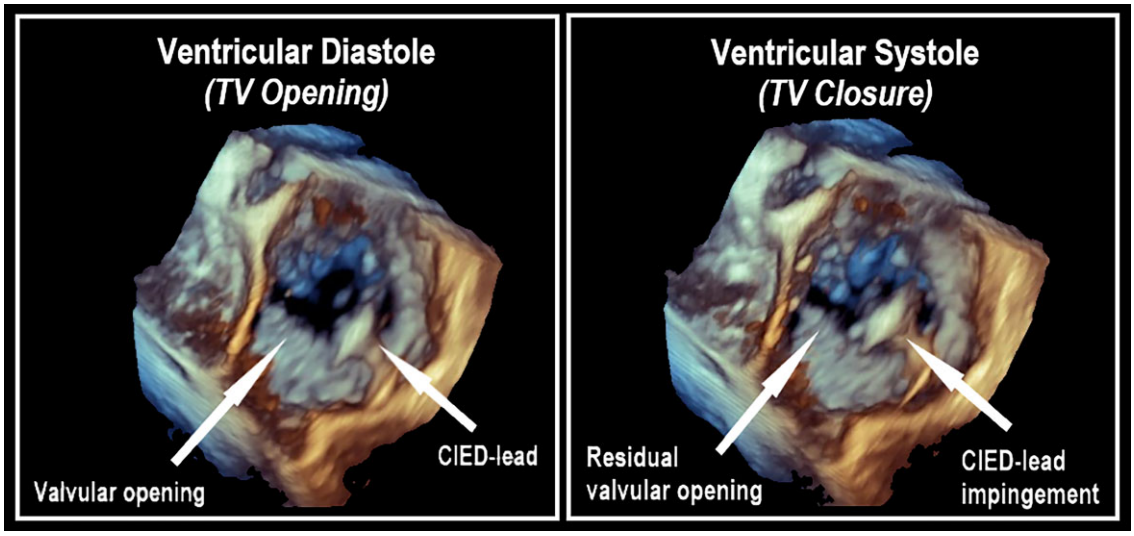

Figure 3 Three-dimensional transoesophageal echocardiogram showing residual valvular opening during ventricular systole due to lead impingement on tricuspid valve posterior leaflet causing inadequate leaflet coaptation.

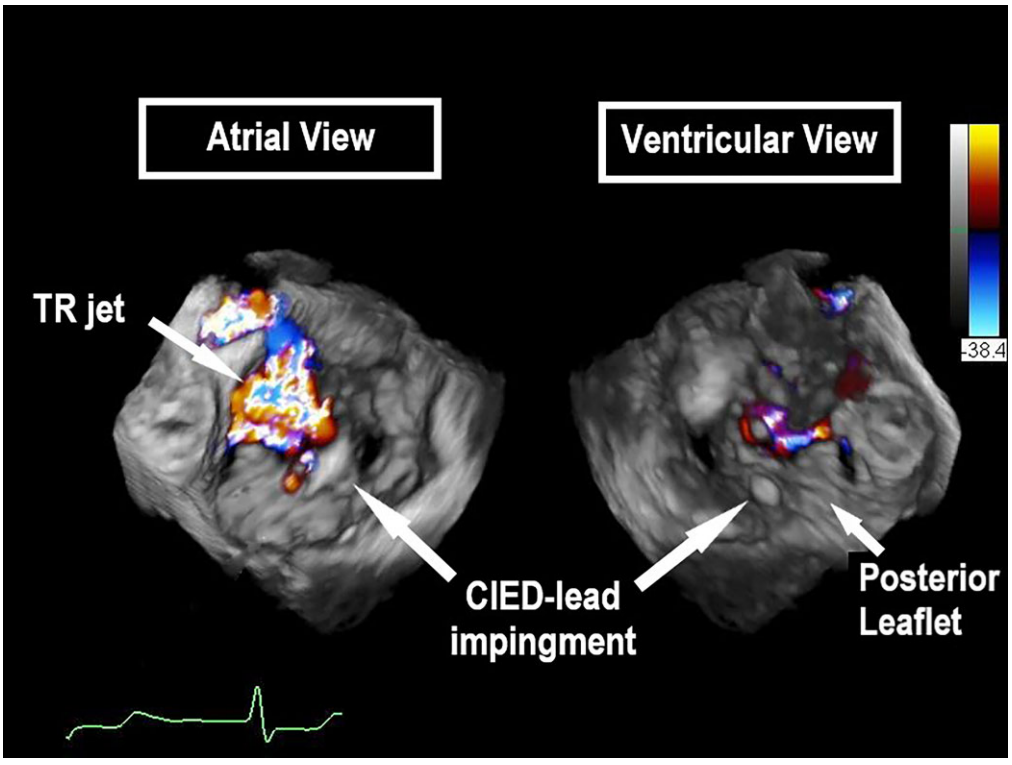

Figure 4 Three-dimensional transoesophageal echocardiogram showing cardiac implantable electronic device-lead impingement of tricuspid valve posterior leaflet.

that is adherent to the TV apparatus but still moves along with it.[6] The least TR-inducing lead positions are either commissural or in the center of the valve.[7] Our patient underwent epicardial lead replacement due to local expertise, however, another potential consideration would have been a leadless pacemaker, both of which avoid traversing the TV.

The 2017 HRS consensus statement broadly categorizes indications for CIED-lead extraction into infectious and non-infectious. Unlike

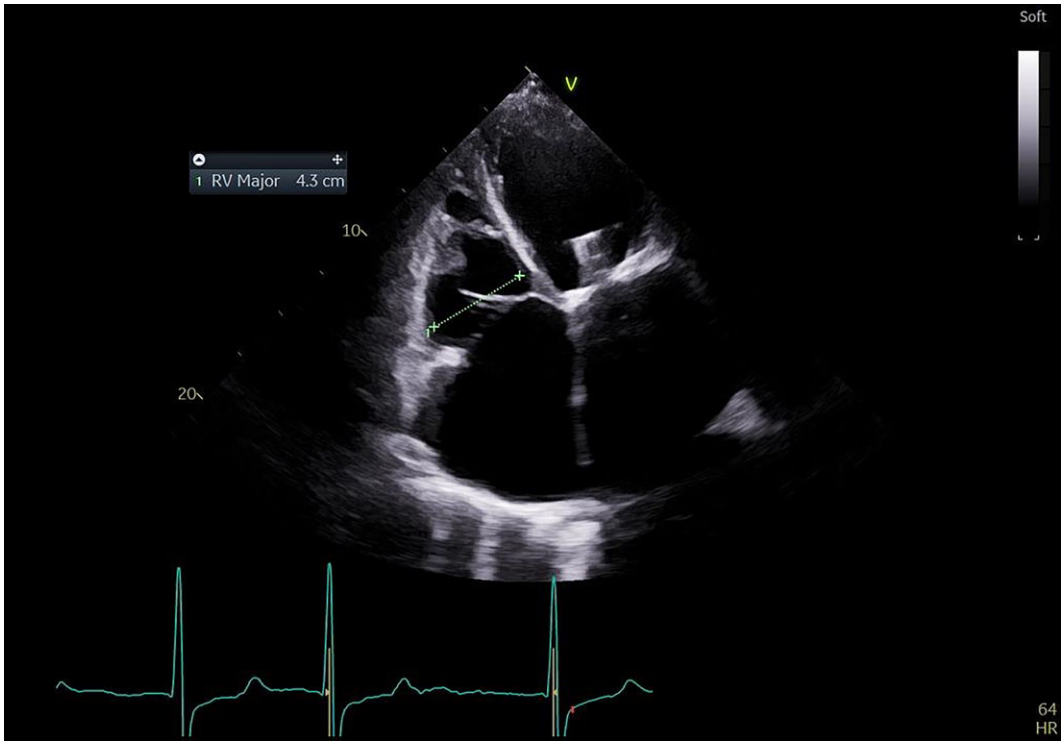

Figure 5 Transthoracic echocardiogram apical-4-chamber view showing reduction of right ventricle basal diameter from 5.2 to 4.3 cm within 3 months.

Table 1 Factors associated with extraction procedure complications and longer-term mortality[8]

Factor	Associated risk
Age	1.05-fold increased mortality
Female sex	4.5-fold increased risk of major complications
Low body mass index (<25 kg/m^2)	1.5-fold increased risk of 30-day mortality, increased number of procedure-related complications
History of cerebrovascular accident	Two-fold increased risk of major complications
Severe LV dysfunction	Two-fold increased risk of major complications
Advanced heart failure	1.3- to 8.5-fold increased risk of 30-day mortality, three-fold increased 1-year mortality
Renal dysfunction	ESKD: 4.8-fold increased risk of 30-day mortality
Diabetes mellitus	Increased in-hospital mortality, 1.71-fold increased mortality
Platelet count	Low platelet count: 1.7-fold increased risk of major complications
Coagulopathy	Elevated INR: 2.7-fold increased risk of major complications and 1.3-fold increased risk of 30-day mortality
Anaemia	3.3-fold increased risk of 30-day mortality
Number of leads extracted	3.5-fold increased risk of any complication, 1.6-fold increased long-term mortality
Presence of dual-coil ICD	2.7-fold increased risk of 30-day mortality
Extraction for infection	2.7- to 30-fold increased risk of 30-day mortality
Operator experience	2.6-fold increased number of procedure-related complications
Prior open-heart surgery	Decreased risk of major complications

infectious indications where mortality benefit of lead extraction is well established, non-infectious indications remain debatable due to the lack of robust randomized data. This controversy is perpetuated by inherent complexities and periprocedural risk of lead extraction resulting from dense adhesions and calcified fibrosis.[8] In fact, the risk of traumatic TV injury has been reported as 3.5–19%.[9] Other factors associated with procedural complications and mortality are listed in *Table 1*.[8]

Symptomatic benefit of CIED-lead extraction for severe TR appears to be heterogenous, with Polewczyk *et al.*[10] reporting 75% clinical improvement vs. Nazmul *et al.*[11] reporting no clinical improvement at

all. We believe that determining the mechanism of TR may delineate the likelihood of symptomatic benefit. The TV apparatus is complex and imaging is notoriously challenging to visualize all three leaflets in a single two-dimensional plane, consequently making it difficult at times to diagnose CIED-induced vs. CIED-associated TR. Adequate assessment of the TV involves a combination of both comprehensive TTE and TOE as described by Hahn,[12] with three-dimensional TOE imaging as the mainstay of diagnosing mechanisms of CIED-induced TR.

This highlights our belief that the mechanism and relationship between the CIED-lead and TR should drive individualized decision-making. We recommend that in cases whereby severe TR is caused by direct lead impingement of TV leaflets preventing adequate coaptation, careful consideration should be given to lead extraction as a means to reduce TR severity and progression. Unless the mechanical obstruction of the CIED-lead is relieved, medical therapy alone is unlikely to resolve the issue, and in time will be insufficient to adequately manage progressively worsening symptoms and the morbidity of living with severe TR. This is in line with recommendations by Chang *et al.*[5] who advocate for lead extraction in the case of lead interference with TV coaptation, although we put forward that the RV and annular dimensions play a smaller role compared with the understanding of TR mechanism in outlining management strategy, as long as the procedural risk of lead extraction is accounted for.

This case emphasizes the importance of understanding the underlying cause of TR in any patient, but especially so in patients with CIED-induced TR. We believe that detailed understanding of the predominant mechanism of CIED-induced TR and the degree of its contribution to the overall magnitude of regurgitation should guide management strategy. We advocate for a mechanistic approach incorporating the CIED-lead interaction with the TV apparatus as the underlying principle in developing future management guidelines for CIED-induced TR.

Lead author biography

Dr Lynn Khor graduated from the University of Adelaide. She is currently completing advanced training in cardiology across Nepean Hospital and Macquarie University Hospital, Sydney, Australia.

Supplementary material

Supplementary material is available at *European Heart Journal—Case Reports* online.

Slide sets: A fully edited slide set detailing these cases and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

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