The added value of three-dimensional echocardiography in the late diagnosis of a pacemaker complication in a patient with severe congestive heart failure: a case report

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Background
Three-dimensional echocardiography (3DE) presents an increasingly important role in the management of interventional cardiac procedures, overcoming limitations of conventional two-dimensional echocardiography (2DE). Early use of 3DE might have an added value in the diagnosis of device-related complications, such as lead induced tricuspid regurgitation (LITR), by providing better understanding of its mechanisms and ensuring a prompt and individually tailored treatment strategy.

Case summary
We report the case of a female patient with repeated hospitalizations for congestive heart failure in the past 2 years, who had a permanent single-chamber ventricular pacemaker (PM) implanted 10 years ago and a misleading diagnosis of severe tricuspid regurgitation (TR) secondary to annular dilation, based on 2DE. Conversely, current 3DE assessment of the TR mechanisms revealed that the PM lead was not placed between the commissures, but in the middle segment of the septal leaflet, causing impingement of the leaflet and severe TR.

Discussion
Given the growing indication for cardiac devices, it is necessary to better define LITR and to establish its impact on patient prognosis. Due to lacking in proper diagnostic techniques, LITR is generally recognized as a late complication of PM/ICD implantation. Two-dimensional echocardiography has important limitations, whereas 3DE provides more accurate information on the TV apparatus in relation to the endocardial leads. Our case shows the usefulness of 3DE for a correct diagnosis of a device-related complication. Its utility in the follow-up of patients receiving cardiac devices remains to be determined, as well as its potential value in the guidance of lead insertion.

Keywords
Lead induced tricuspid regurgitation • Three-dimensional echocardiography • Congestive heart failure • Case report

Learning points
• Two-dimensional echocardiography is key to diagnose lead induced tricuspid regurgitation (TR), but has important limitations in assessing its mechanisms.
• When facing with a patient with un-explicable congestive heart failure, significant TR and history of lead implant, three-dimensional echocardiography becomes essential in assessing TR mechanisms.
Introduction

Three-dimensional echocardiography (3DE) presents an increasingly important role in the assessment of valvular heart disease and the management of interventional cardiac procedures, overcoming the limitations of conventional two-dimensional echocardiography (2DE).1–4 Three-dimensional echocardiography can be useful both for planning and guiding procedures, and also for the follow-up of patients. The early use of 3DE might have added value in the diagnosis of device-related complications, such as lead induced tricuspid regurgitation (LITR) in patients receiving pacing or resynchronization therapy.5 Three-dimensional echocardiography provides better understanding of the LITR mechanisms, and thus ensuring a prompt and individually tailored treatment strategy.6

Case presentation

A 76-year-old woman presented to the emergency department with progressive signs and symptoms of congestive heart failure in the past month. The patient had been repeatedly admitted for decompensated congestive heart failure in the past 2 years, and discharged after standard treatment of care. She had a history of permanent atrial fibrillation, a single-chamber ventricular pacemaker (PM) implant for complete atrioventricular block 10 years earlier, and pacing induced moderate left ventricular (LV) systolic dysfunction. The patient received oxygen, loop diuretics in continuous perfusion (100 mg of furosemide/day), spironolactone (50 mg/day), acenocumarol in doses monitored by using the INR, perindopril (5 mg/day), and digitalis (0.25 mg/day). After 3 days of favourable evolution, she was switched on oral furosemide (60 mg/day).

Clinical examination revealed regular cardiac rhythm (70 b.p.m.), normal blood pressure (100/60 mmHg), polypnoea, and mild hypoxaemia in ambient air (SaO2 95%), holosystolic murmur at the lower}

Timeline

| Ten years prior to presentation | Medical history of complete atrioventricular block. |
|-------------------------------|--------------------------------------------------|
|                               | Two-dimensional echocardiogram showed normal left ventricular systolic function, mild mitral, and tricuspid regurgitation (TR). |
|                               | Received a single-chamber pacemaker (PM), VVI mode, 70 b.p.m. |
| Two years prior to presentation | Admission for congestive heart failure, with shortness of breath, orthopnoea and lower limbs oedema; BNP level of 5250 ng/dL (normal values of maximum 100 ng/dL). |
|                               | Two-dimensional echocardiogram showed mild left ventricular function, moderate mitral regurgitation, severe TR, and severe dilation of the right cardiac chambers. |
|                               | Normally functioning PM, 70 b.p.m., atrial fibrillation. |
|                               | IV, then oral diuretics; Spironolactone; ACE inhibitor; oral anticoagulant. |
| Up to present                 | Further two hospitalizations for decompensated heart failure similarly treated. |
| Present                      | Two-dimensional echocardiogram showed severe dilation of the tricuspid annulus, coaptation loss, and severe ‘functional’ TR. |
| Present                      | Another episode of decompensated congestive heart failure. BNP level was 3520 ng/dL (normal values of maximum 100 ng/dL). |
|                              | Clinical examination: regular cardiac rhythm (70 b.p.m.), normal blood pressure (100/60 mmHg), polypnoea and mild hypoxaemia in ambient air (SaO2 95%). |
|                              | (1) Two-dimensional echocardiogram showed severe dilation of the tricuspid annulus, coaptation loss, and severe ‘functional’ TR. |
|                              | (2) Workup including three-dimensional echocardiography revealed that the pacing lead was not located between the tricuspid valve commissures, but caused the impingement of the septal leaflet, with secondary coaptation deficit and severe secondary TR. Due to longstanding pacing, the lead was adherent to surrounding tissue. |
|                              | Patient was discharged on Day 10 of therapy with oxygen, iv diuretics, spironolactone, ACE inhibitor, and anticoagulants. |
|                              | Patient was scheduled for cardiac surgery and upgrade to resynchronization therapy with an epicardial lead. Patient’s request was for a temporization of the procedure. She died 2 months after the last hospitalization, with severe and irreversible heart failure. |
Figure 1  Two-dimensional echocardiographic assessment of the tricuspid regurgitation. (A) Colour flow assessment of the severe tricuspid regurgitation, with large PISA and vena contracta. (B) Colour mode of the tricuspid regurgitation, showing a holosystolic regurgitant jet. (C) Continuous-wave Doppler, showing a dense triangular envelope of the tricuspid regurgitation jet.
sternal border, and prominent signs of right heart failure (jugular congestion, hepatomegaly, and peripheral oedema).

The electrocardiogram showed atrial fibrillation and a heart rate of 70 b.p.m., and QRS corresponding to pacing morphology. Complete blood cell count and chemistry panel were normal, except for mild hyponatraemia and a high NT-pro-BNP (3520 ng/mL, normal values of max 100 ng/dL). Chest X-ray showed cardiomegaly and bilateral pleural effusions. Abdominal computed tomography was remarkable for hepatic congestion, dilated inferior vena cava, and small peritoneal effusion. On PM interrogation we found that the right ventricle (RV) had been paced 90% of the time, with normal impedance and thresholds.

Two-dimensional transthoracic echocardiography revealed moderately dilated LV (87 mL/m²), severely dilated left atrium (101 mL/m²), and severe LV global systolic dysfunction (LV ejection fraction of 30%). Two-dimensional echocardiography also showed interventricular and intraventricular dyssynchrony and moderate functional MR. Moreover, 2DE showed severe dilation of the right atrium.

Figure 2 Three-dimensional echocardiographic view of the right atrium and the right ventricle. (A) A short-axis view of the tricuspid valve, seen from the right atrium. Two-dimensional longitudinal and transversal cut-planes help in the anatomical orientation of the tricuspid leaflets and in visualizing the pacemaker lead. The tricuspid leaflets are the anterior (AL), posterior (PL), and septal (SL). The red arrows point to the coaptation defect, the yellow arrows to the pacemaker lead, and the blue arrows to the restricted septal leaflet on each analysed cut-plane. In real time, the septal is moving beneath the pacemaker lead. (B) A longitudinal cut-plane of the right cardiac chambers, with the pacemaker lead and the tricuspid leaflets visualized in an opened position. The yellow arrows point to the pacemaker lead, and the blue arrows to the opened tricuspid leaflets.
(81 mL/m²) and of the RV (RV end-diastolic area of 32 cm²), severely dilated tricuspid annulus (31 mm²/m²), and non-coapting tricuspid valve (TV) leaflets. In conclusion, 2DE suggested severe functional tricuspid regurgitation (TR) caused by the important dilation of the tricuspid annulus, with flow reversal into the hepatic veins, and vena contracta of 12 mm and EROA of 51 mm derived from 2DE PISA method (Figure 1).

A multi-beat full-volume of the TV was acquired using an echo machine equipped with a 3DE probe (GE Healthcare, Horten, Norway) and post-processed in order to measure the coaptation defect and to further analyse the mechanisms of the TR. Three-dimensional echocardiography analysis using flexi-slice tool and multiple cut-planes at the level of the TV revealed that the PM lead was not placed between the commissures of the valve, but at the middle of the septal leaflet, causing impingement of the leaflet, coaptation deficit, and severe secondary TR (Figure 2).

Therefore, 10 years after the PM implant, the patient was diagnosed with severe TR due to a coaptation deficit secondary to impingement by the PM lead. Unfortunately, given the late diagnosis of this post-implant complication, the lead could not be repositioned and the patient was referred for cardiac surgery. Interventional extraction of the lead was considered very risky in this case, considering the long duration after the initial implant, which most probably led to the adherence of the lead to the tricuspid leaflet and to the venous walls. Additionally, the patient was a candidate for an upgrade to a biventricular device considering the severe LV dysfunction and congestive heart failure, which could be accomplished by placing of an epicardial lead during cardiac surgery.

Patient’s delayed the procedure for personal reasons. Therefore, patient was discharged after ten days of receiving oxygen, iv then oral diuretics, antialdosteronic, ACE-inhibitors and anticoagulant. She died 2 months after the last hospitalization, with severe and irreversible heart failure.

**Discussion**

Tricuspid regurgitation due to permanent PM/internal cardioverter defibrillator (ICD) lead implantation is an insufficiently studied entity. Given the growing indication for these devices, it is necessary to better define LITR and to establish its impact on patient management and prognosis. Probably due to a lack in diagnostic techniques or improper follow-up of these patients, lead induced TR is generally recognized as a late complication of PM/ICD implantation, resulting from a combination of pathogenic mechanisms. Most frequently, LITR is the consequence of mechanical factors such as lead impingement, adherence, entanglement or perforation, but it can also be the result of RV dyssynchrony during pacing. The diagnosis of LITR is clinically suspected in patients implanted with a PM or ICD, but is essentially established by echocardiography. A prompt diagnosis of LITR due to lead mal-position in relation to the TV leaflets may result in repositioning of the lead in the electrophysiology department with the improvement or even the resolution of the regurgitation.

Two-dimensional echocardiography has important limitations in the diagnosis of LITR, whereas 3DE provides more accurate information on the TV apparatus, in relation to the endocardial leads. In our case, the diagnosis of severe LITR by lead impingement was certified with the aid of 3DE, as two-dimensional imaging was inaccurate in determining the underlying mechanism for so many years. Right ventricular dysynchrony was probably another contributing factor in the pathogenesis of the TR, given the duration of RV pacing, which later resulted in right chamber dilation.

**Conclusions**

Our clinical case report shows the usefulness of 3DE, in addition to 2DE, for a correct and early diagnosis of a complication related to cardiac devices. Its utility in the management and follow-up of patients receiving a PM or ICD remains to be determined, as well as its potential value in the guidance of lead insertion. However, in our particular case, death could have been prevented by an early diagnosis of LITR, as a PM-related complication.

**Supplementary material**

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

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

**Consent:** The author’s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

**Conflict of interest:** none declared.

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