iMDT corner on Novel Concepts in Cardiopulmonary and Structural Heart Disease

Cardiovascular implantable electronic device-related tricuspid regurgitation: a multidisciplinary team approach

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Submitted Dec 20, 2019. Accepted for publication Jan 17, 2020.
doi: 10.21037/jtd.2020.01.64
View this article at: http://dx.doi.org/10.21037/jtd.2020.01.64

Introduction

Cardiovascular Implantable Electronic Device (CIED)-related tricuspid regurgitation (TR) complicates between 10% and 20% of implantable cardioverter defibrillator (ICD) and pacemaker implantations and is independently associated with increased cardiovascular morbidity and mortality (1,2). This condition is multifactorial in etiology and thus management can be challenging.

Case presentation

A 46-year-old male with a history of non-ischemic dilated cardiomyopathy, dual-chamber ICD, persistent atrial fibrillation, and end-stage renal disease was transferred to our institution for atrioventricular node ablation as well as cardiac resynchronization therapy device upgrade. Both procedures were performed uneventfully. However, in the post-operative period, the patient experienced anasarca and congestive hepatopathy requiring ICU admission. A transthoracic echocardiogram (TTE) was obtained which showed severe TR with obstruction of the posterior and septal leaflets by the ICD lead and lead entrapment into the sub-valvular apparatus, as well as right ventricular systolic dysfunction with tricuspid valve annular dilation measuring at least 4.4 cm (*Figure 1*).

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Expert opinion 1 (Electrophysiology—Dr. Zedan)
Because 3D echocardiography provides spatial reconstruction of the three tricuspid leaflets and of their interaction with the device lead, it is the preferred mode of imaging for diagnosis of CIED-related TR (3,4). 3D echocardiography also allows for precise lead localization, which aids in identifying the mechanism of TR; a lead in the favorable position of the inferior-septal leaflet commissure is unlikely to cause valve dysfunction (4).

Although in this particular case, the degree of obstruction was such that the 2D TTE images were diagnostic, 2D TTE and TEE tend to underestimate TR severity. This is due to acoustic shadowing from the device causing signal attenuation, as well as the eccentric nature of the TR jet from asymmetric impairment of leaflet mobility. Thus, the sensitivity of 2D TTE for detection of significant CIED-related TR has been reported as low as 12–17% (4). Reliance on specific parameters such as holosytolic hepatic vein flow reversal is helpful to establish the diagnosis. Due to the same potential for local imaging artifact from the lead, cardiac MRI is unlikely to supersede 3D echocardiography for the evaluation of CIED-related TR (4).

Expert opinion 2 (Echocardiography—Dr. Guigui, Dr. Horvath, Dr. Arenas, Dr. Mihos)
Many theories exist for the mechanisms of CIED-TR. This includes mechanical causes such as suboptimal location of the lead passing through the valve, development of scar tissue on the leaflets, leaflet perforation or damage, and adverse right ventricular remodeling due to RV pacing and geometry changes (5,6). Regardless of the cause, CIED-TR

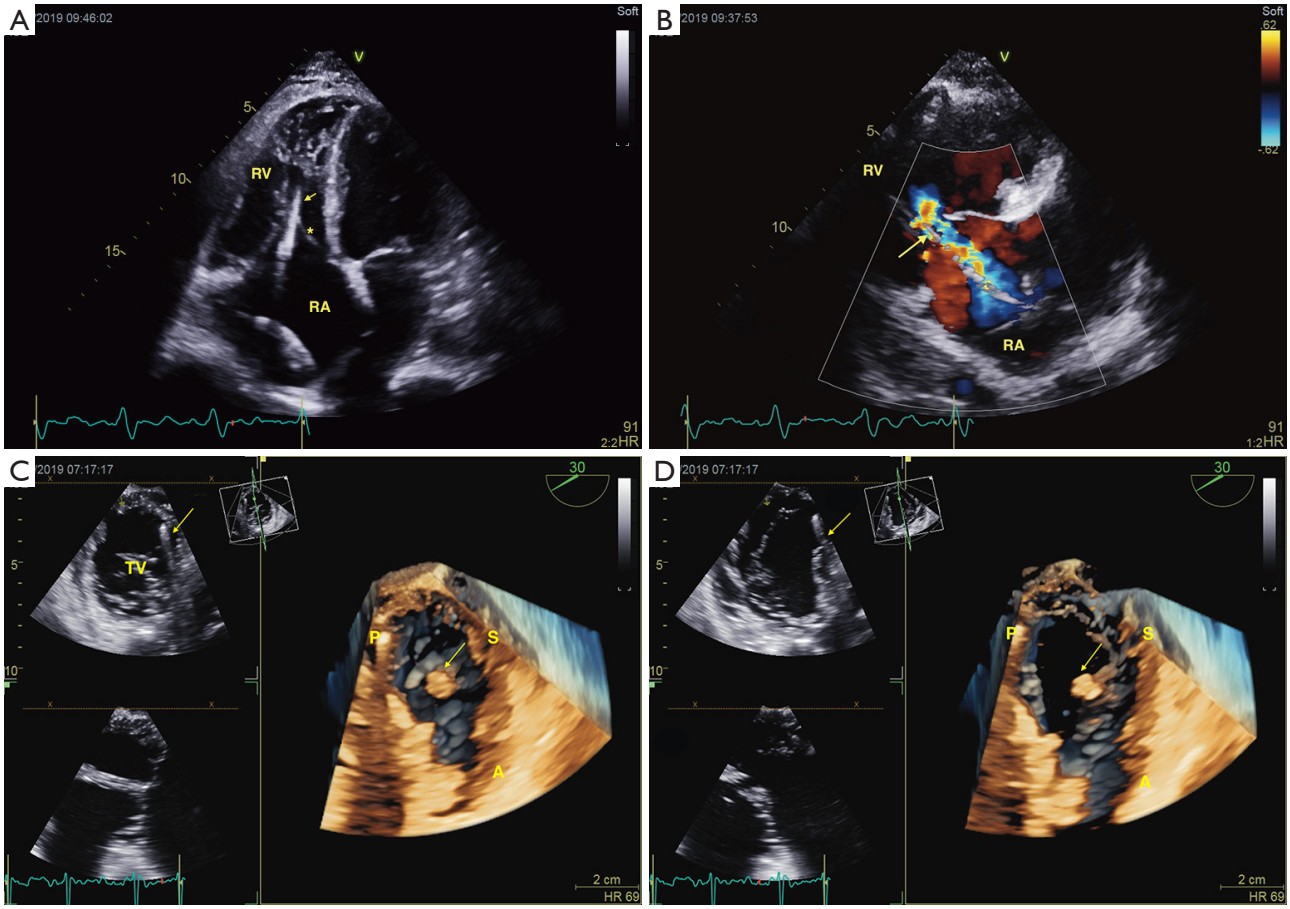

Figure 1 CIED-related tricuspid regurgitation. (A) The apical four-chamber view demonstrates lead impingement of the septal leaflet of the tricuspid valve (star) in systole, as well as entrapment of the lead (arrow) into the sub-valvular apparatus and RV trabeculations. The RV itself appears severely enlarged and displays a moderately impaired systolic function. Other findings on echocardiogram (not shown) included a dilated and non-compressible IVC. (B) Obstruction of the posterior leaflet of the tricuspid valve by the endocardial lead (arrow) is seen on the parasternal long axis—RV inflow view. (C) 3D reconstruction of the tricuspid valve and ICD lead (arrow) in systole in the following views: en face view (right); transgastric view (upper left); transgastric long axis view (lower left). (D) 3D reconstruction of the tricuspid valve and ICD lead in diastole in the following views: en face view (right); transgastric view (upper left); transgastric long axis view (lower left). CIED, Cardiovascular Implantable Electronic Device; ICD, implantable cardioverter defibrillator; P, posterior leaflet; S, septal leaflet; A, anterior leaflet; TV, tricuspid valve; RA, right atrium; IVC, inferior vena cava; RV, right ventricle.

can be debilitating and requires an index of suspicion for diagnosis and aggressive treatment.

Medical therapy is directed at treating the symptoms of TR and right heart dilatation with diuretics is the mainstay of treatment. Surgical therapy requires device lead extraction either percutaneously with traction or laser device, with possible need for open tricuspid valve repair or replacement with extravasation of the lead around a new tricuspid annular ring or valve (4). Other pacing options in an open technique include an epicardial pacing system or placement of a coronary sinus lead only to replace the right ventricular lead. The latter option has been our preferred approach.

Expert opinion 3 (Cardiac Surgery—Dr. LaPietra)

One other important risk factor for TR is during lead extraction procedures. About 10% of ICD/pacemaker leads require extraction—with or without reimplantation of new

leads—either due to infected or malfunctioning leads (7). Around 24,000 transvenous lead extraction procedures occur yearly worldwide (8). With leads being in place for years, leaflets tend to adhere to and encapsulate the leads over time, leading to tricuspid valve damage and even leaflet avulsion during the extraction procedure (9). Removing more than one lead in old age patients and extracting leads with endocarditis involving the tricuspid valve carries the worst prognostic risk for worsening TR during lead extraction procedure (10).

Treatment and follow-up

RHC was performed and was consistent with WHO group 2 pulmonary hypertension. The patient was treated via hemodialysis for volume management. His heart failure medication regimen was optimized.

Discussion

In this case, the etiology of the TR is probably mixed, resulting from a combination of CIED lead impingement on the septal and posterior leaflets of the tricuspid valve as well as RV systolic dysfunction. The management of TR in those cases is complex as corrective intervention usually in the form of transvenous lead extraction with or without TV repair or replacement is associated with its own set of morbidities and mortality. The decision-making process should take into account several factors at once including the severity of RV dysfunction and TV annular dilation (11), the nature of the interaction between the leaflet(s) and the lead, the degree of leaflet tethering, and the presence of leaflet damage (4). In this case, given the presence of significant WHO group 2 pulmonary hypertension, right ventricular functional impairment, and tricuspid valve annular dilation, the patient was at prohibitive for isolated tricuspid valve surgery and optimizing medical management was deemed the safest strategy. Ongoing clinical trials and registry data are showing promising developments in the percutaneous treatment of high-risk patients with severe TR, including edge-to-edge repair and annuloplasty systems (12,13).

Conclusions

CIED-related TR is a relatively prevalent occurrence complicating 10–20% of all CIED cases. It can be best diagnosed with 3D echocardiography which provides spatial reconstruction of the tricuspid leaflets and their interaction with the CIED lead. Identifying the exact mechanism of TR is critical in the management of this condition to determine the necessity and safety of corrective intervention.

Acknowledgments

Funding: None.

Footnote

Provenance and Peer Review: This article was commissioned by the Guest Editors (Christos G. Mihos) for the series "Novel Concepts in Cardiopulmonary and Structural Heart Disease" published in *Journal of Thoracic Disease*. The article was sent for external peer review organized by the Guest Editor and the editorial office.

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at http://dx.doi.org/10.21037/jtd.2020.01.64). The series "Novel Concepts in Cardiopulmonary and Structural Heart Disease" was commissioned by the editorial office without any funding or sponsorship. CGM served as the unpaid Guest Editor of the series and serves as an unpaid editorial member of *Journal of Thoracic Disease* from Jan 2019 to Dec 2020. The other authors have no other conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. Written informed consent was obtained from all patients.

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Cite this article as: Guigui S, Zedan A, LaPietra A, Horvath SA, Arenas IA, Mihos CG. Cardiovascular implantable electronic device-related tricuspid regurgitation: a multidisciplinary team approach. J Thorac Dis 2020;12(5):2986-2989. doi: 10.21037/jtd.2020.01.64

