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Cardiac Implantable Electronic Device Induced Tricuspid Regurgitation: A Mini Review

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ABSTRACT

The continuing application of cardiac implantable electronic devices (CIED) has led to an increasing concern regarding disturbances in the tricuspid valve (TV). The most prevalent TV issue related to lead implantation is tricuspid regurgitation. CIED-induced tricuspid regurgitation is associated with emerging or worsening preexisting heart failure and increased mortality rate. Because discontinuing the implantation of these instruments is not feasible, further knowledge of their mechanical problems may lead to advancements. This review addresses the available data regarding CIED-induced tricuspid regurgitation, elucidating its plausible pathomechanisms, diagnostic methods, and prospective treatments.

Keywords: Cardiac implantable electronic device; tricuspid regurgitation; heart failure.

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Трикуспидальная регургитация, индуцированная сердечными имплантируемыми электронными устройствами (краткий обзор)

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АННОТАЦИЯ

Активное применение сердечных имплантируемых электронных устройств (СИЭУ) вызывает растущую обеспокоенность по поводу нарушений в работе трикуспидального клапана. Наиболее распространенной проблемой, связанной с имплантацией электродов, является трикуспидальная регургитация, которая приводит к возникновению или усугублению уже имеющейся сердечной недостаточности, а следовательно, к повышению уровня смертности. Поскольку отказ от имплантации этих устройств нецелесообразен, дальнейшее изучение механических проблем, связанных с их работой, может привести к улучшению ситуации. В данном обзоре систематизированы имеющиеся данные о трикуспидальной регургитации, вызванной имплантацией СИЭУ, описаны вероятные механизмы развития этой патологии, методы диагностики и перспективные направления в лечении.

Ключевые слова: сердечное имплантируемое электронное устройство; трикуспидальная регургитация; сердечная недостаточность.

Как цитировать

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INTRODUCTION

Cardiac implantable electronic device (CIED) usage has become prevalent for cardiac rhythm detection and management. CIEDs typically involve inserting a lead through the tricuspid valve (TV) and fixing its end to the right ventricle (RV). Numerous publications have described the link connecting the device lead and the TV apparatus, leading to severe tricuspid regurgitation (TR). TV regurgitation is the most common TV malfunction associated with lead implantation. The interval between implantation and clinical appearance can range from a few weeks to up to 30 years [1–3].

TR progression is significantly noticeable in patients with a higher ejection fraction after progressing from no TV disease to mild TR. Nevertheless, TR progression is more crucial in individuals with advanced heart failure (HF) because it is associated with a considerably greater incidence of severe TV illness. HF therapies may not be effective in managing lead-induced TR, which could worsen the prognosis. Therefore, improved prevention and treatment are pivotal for identifying the patients most susceptible to the effects of TR [4, 5].

CIED-induced TR is becoming more widely acknowledged as a significant clinical disorder associated with an increased risk of HF and mortality. Poor clinical outcomes may result from the underestimation of TR severity or late diagnosis of worsening TR, regardless of the morphological varieties. This study aimed to review the information currently available on CIED-induced TR, describing its potential pathomechanisms, diagnostic methods, and therapeutic options [1, 2, 6].

METHODS

An extensive electronic search was conducted using search engines such as Google Scholar, ScienceDirect, and PubMed. The search was limited to English-language articles published between 2014 and 2023 using “cardiac implantable electronic device” AND “tricuspid regurgitation” as the keywords. The search results included reviews, original papers, and case reports. Articles with restricted access and those authored in languages other than English were excluded. The extracted articles were managed using the Mendeley software. After arranging the search results based on the titles and abstracts, the full texts of the publications were examined, and those that matched the exclusion criteria were eliminated. A total of 1.233 articles were retrieved through the search strategy, and 14 articles met the criteria. The literature search process is shown in Figure 1.

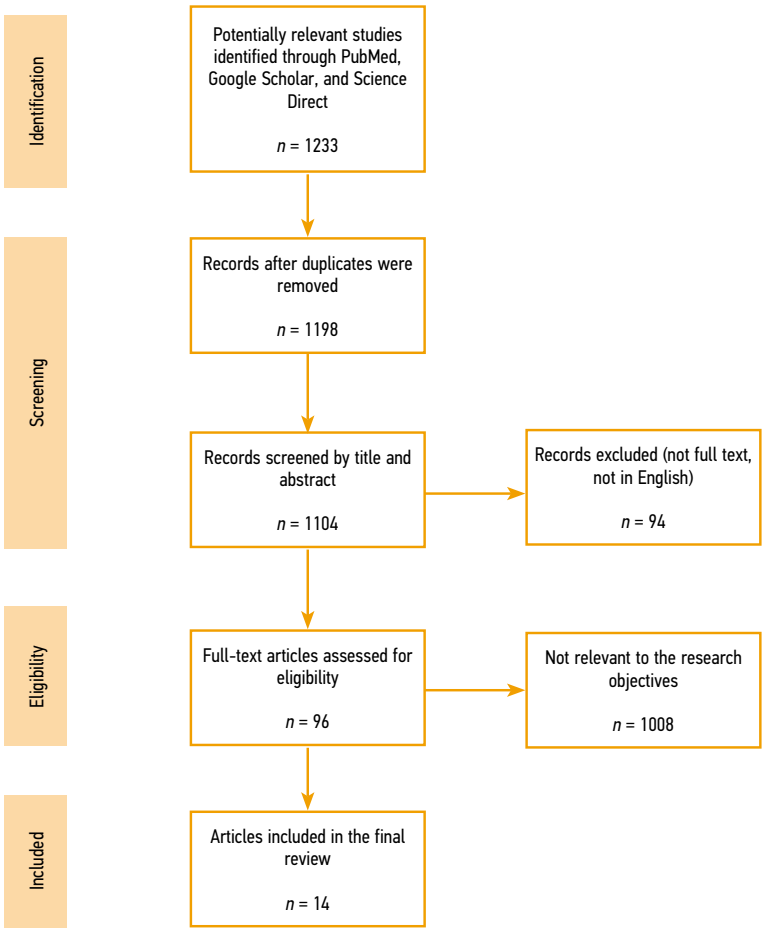

Fig. Diagram ilustrating study selection process
Рис. Диаграмма, иллюстрирующая процесс отбора исследований

RESULTS AND DISCUSSION

Definition

CIEDs, such as permanent pacemakers (PPMs), implantable cardiac defibrillators (ICDs), and cardiac resynchronization therapy devices, are increasingly being used in patients with severe cardiac disorders. The lead in CIEDs is usually placed extending across the TV and anchors in the ventricle, whereas leadless cardiac pacemakers (LCPM) are inserted right into the RV [7, 8].

CIEDs can lead to TV malfunction, which includes regurgitation and, less typically, stenosis. TR provoked or intensified by a right ventricular lead after pacemaker placement is referred to as cardiac implantable electronic device-induced TR. Various mechanisms have been proposed to precipitate TR and right ventricular dysfunction after lead implantation [3, 4, 6].

For CIED implantation, three methods are most often used: prolapsing, direct crossing, and dropping down. Of the three main methods of right ventricular lead placement, the prolapsing technique may be less likely to result in leaflet perforation or rupture than other techniques because of less damage to the leaflets and subvalvular tissue. In addition, because leads are often inserted with some excess intraventricular lead loop length to allow movement of the arm, the lead loop may cause anterior leaflet entrapment [1, 9].

Epidemiology

The prevalence of CIED-associated TR varies from 7% to 45%, depending on the research methodology and population observed. The different standards used in the studies, such as wide variation in follow-up evaluation, imaging technique, availability of baseline echocardiograms, study design, and divergent interpretation for “significant” postprocedural TR, caused several prevalence [1, 10, 11].

No substantial disparity was noted between the implanted device type (ICD or PPM) and the likelihood of developing postimplantation TR. An increased risk of TR was associated with all devices. Patients’ TR severity increases by one or two grades from pre- to postpermanent lead implantation. Novel postimplant moderate or severe TR is associated with poor right ventricular function and long-term (>10 years) survival rates [12–14].

Hospitalizations for HF and all-cause mortality risk were related to CIED-associated TR. After CIED placement, there may be an increase in TR symptoms between 1 and 12 months, whereas hospitalization for HF only became relevant more than a year after CIED implantation [15, 16].

Identifying risk factors for TR development after CIED implantation has been the focus of numerous studies. Atrial fibrillation and right ventricular systolic pressures were linked to significant TR progression in a study by Van de Heyning et al. Atrial fibrillation remained the only independent predictor after adjusting for the baseline TR grade. Zhang et al. found no association between TR and baseline atrial fibrillation and mild TR, age, or left ventricular ejection fraction. In contrast, the time that passed since the implantation and lead interference were risk factors for worsening TR [6, 17].

Mechanism

Previously, the interaction between the device lead and TV leaflets was considered a primary cause of TR. Nonetheless, researchers have reclassified CIED-related TR as a distinct etiologic category because of the numerous causes of TR in the existence of a CIED. Mechanisms underlying CIED-related TR can be classified into implantation, pacing, and device-related [1, 8].

Conventional CIEDs require the implantation of a lead through the TV, which may contribute to TR generation. The most frequently mentioned mechanism among implantation-related TR is lead impingement, which is the mechanical interference of the ventricular lead with leaflet movement. Additional implantation mechanisms include leaflet perforation, impairment of the subvalvular apparatus, entangled or ruptured chordae tendinae, and perforated papillary muscles [1, 2, 4].

Frequently, TR is brought on by or made worse by typical functional causes. Pacing-induced TR is a pathological process triggered by electrical stimulation of the RV. In the absence of mechanical leaflet interference, dyssynchrony brought on by right ventricular stimulation appears to create geometric alterations in the RV that lead to insufficient mitral and TV coaptation. In this situation, nonapical right ventricular pacing — pacing of the interventricular septum or right ventricular outflow tract — may result in less dyssynchrony and more natural ventricular activity than apical pacing. It may also be linked to a decreased risk of TR worsening. The lead’s position in the RV — apical vs. nonapical — influences the lead–leaflet relationship during its crossing over the TV [1].

TR progresses at different rates depending on the mechanism after CIED implantation. Mechanical impingement/restriction of the leaflets or damage to the TV apparatus are possible causes of acute TR alterations. Significant changes in heart inflammation were also noted a few days after surgery. Furthermore, endocarditis or thrombus formation may be more likely to be caused by the device [8, 18].

For severe TR or lead-related infections, transvenous lead extraction (TLE) is a laborious treatment option. The fundamental problem with TLE is that because of considerable fibrous tissue growth along with lead attachment to the TV apparatus, there is a high likelihood of TV avulsion with increasing TR. The main risk associated with TLE operations is TV tissue avulsion throughout manual traction for lead expulsion, which can intensify TR severity [1].

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Leadless pacemakers potentially intensify or even develop TR because of their functional effects and mechanical disruption of the TV subvalvular apparatus. Because of the potential of leadless devices to become entangled in the chordae tendineae or to interact directly with leaflets, septal insertion of these devices has been demonstrated to have a fivefold increased risk of intensifying tendinopathy (TR) compared with apical implantation [11].

Because of lower left ventricular filling and elevated right ventricular pressure, CIED-induced TR may present as either left- or right-sided HF. Notable differences were observed in the responses to HF therapies between lead-induced and lead-nonrelated TR. If significant TR has occurred during follow-up, the underlying cause should be determined. HF therapies may not be effective in managing HF associated with lead-induced TR, which could worsen the prognosis [7, 15].

Diagnosis

The first imaging modalities for CIED implantation are chest X-ray or ultrasonography. Chest radiography is performed to verify the continuity of the leads and determine the dislocation of the leads and their position relative to each other. The gold standard for diagnosing and classifying TR severity is echocardiography along with associated imaging modalities. All available echocardiographic techniques must be employed for the correct diagnosis of lead-related TR and to distinguish the mechanism causing TR [1, 9].

Identifying a new or deteriorating TR after implantation can be arduous if a baseline echocardiogram (before implantation) is unavailable. In preparation for CIED implantation, candidates should optimally undergo a thorough baseline echocardiogram with the assessment of significance on TV and right ventricular performance. In addition, routine echocardiography after CIED implantation should be performed to establish the presence of TV remodeling and risky lead placement, both of which may result in lead-induced severe TR [5, 8].

Two-dimensional (2D) transthoracic echocardiography (TTE) has been the initial method for identifying and classifying TR and assessing its hemodynamic effects. 2D imaging is applicable to determine the cause of TR, grading of its severity, and evaluating how it affects right ventricular performance. Since only two TV leaflets may be seen at a time on the unusual parasternal view, conventional 2D TTE had limited ability to analyze the anatomy of the TV, all the more, figuring out how the leaflets and a CIED lead interact [1, 9].

The primary shortcomings of 2D echocardiography in evaluating lead-related TR have been resolved by three-dimensional echocardiography (3DE). All TV leaflets and the pacing lead position can be observed concurrently with 3D imaging. 3DE is critical for understanding the pathophysiological pathways that cause lead-related diseases. “En face” imagery from the ventricular and atrial viewpoints during TTE and 3DE can accomplish a thorough TV evaluation [1, 14].

The following steps are involved in diagnosing TR associated with lead: 1) Using a direct comparison of pre- and postimplant TTE studies, the presence of TR is determined; (2) TR is graded based on the most recommendations; (3) using 2D echocardiography and 3D imaging to show mechanical damage on the TV leaflets or apparatus; (4) assessing the hemodynamic effect on the RV if TR is greater than moderate; and (5) determining whether early TLE or surgical treatment is necessary and feasible [1].

When 3D TTE visualization of leaflets is insufficient, transgastric 2D or 3D transesophageal echocardiography (TEE) and cine cardiac CT can be used to provide short-axis TV imaging. On the condition that the lead position cannot be established with certainty, TEE should be considered an additional imaging modality. The use of cardiac magnetic resonance imaging (MRI) for diagnostic purposes is limited because notable local local artifacts in the vicinity of the CIED leads affect cardiovascular MRI and frequently obscure the view of the lead, valve, and related TR. Therefore, 3D echocardiography is the preferred imaging modality for diagnosing and planning interventional therapy for TR associated with CIED [9, 11, 19].

Management

Treatment options include medical therapy and percutaneous and surgical interventions. Medical therapy is aimed at alleviating TR symptoms and right heart dilatation, with diuretics as the primary treatment. Aldosterone antagonists are recommended as helpful supplemental medications, particularly for patients with hepatic congestion and secondary aldosterone rise, whereas loop diuretics are frequently used in severe TR and symptomatic right HF.[4]

The definitive therapy may require lead repositioning or removal, either surgically or percutaneously, depending on the expertise of each medical facility. Treating lead-related TR with TLE may be appealing. Given the lack of defined guidelines for the use of TLE in patients with pertinent TR, a comprehensive risk–benefit analysis is crucial. Although rare, significant damage to the TV apparatus may occur throughout TLE, with a reported incidence of 2.5% across over 2600 procedures. Furthermore, the clinical reason for stimulation or pacing when lead extraction is required must be reassessed, and alternative CIED techniques such as subcutaneous ICDs, leadless pacemakers, epicardial, His-bundle pacing, and coronary sinus lead positioning must be considered. Nonetheless, mechanical issues that result in substantial TR can still affect devices such as leadless pacemakers [1, 8, 20].

In addition to severity and irreversible TV leaflet impairment, risk for progressive tricuspid annular dilatation, right ventricular enlargement or malfunction, and right ventricular HF increased if CIED-related TR is not identified and treated quickly. Most often, these cases require surgical

valve replacement or repair. The current criteria for surgical valve repair or replacement when CIED-induced TR occurs consider the degree of regurgitation, presence of symptoms, and right ventricular functionality [20].

The 2021 ESC Guidelines for the Management of Valvular Heart Disease state that valve repair is preferred over valve replacement when there is neither substantial TV degradation nor annulus dilatation. No discernible difference in durability was observed between CIED-induced and CIED-associated TR, and TV repair was still feasible in 63% of the cases with satisfactory long-term results. In 30% of the cases, TV replacement was unavoidable. Several procedures have been employed to repair the valve in patients with CIED-induced TR. In some cases, a fibrotic reaction resulting in lead encapsulation in the TV leaflet was observed and removed. Typically, this process was adequate for the leaflet to move freely [10, 21].

Therapy including transcatheter TV replacement (TTVR) or percutaneous transcatheter edge-to-edge TV repair (T-TEER) for severe TR has recently become available as a nonsurgical option to reduce TR severity in high-risk patients. TTVR in patients with CIED achieves procedural performance and TR reduction similar to those in patients without CIED [22].

Limitation

The primary limitation to the generalization of these results is the heterogeneity of the research methodologies and populations observed in the studies. This article does not restrict the standards used in each investigation, such as study design, evaluation period, imaging technique, and availability of baseline echocardiography. Nonetheless, these findings must be interpreted with caution, and certain limitations should be considered.

CONCLUSION

Patients with CIED have a higher risk of TV disorders, particularly regurgitation. CIED-related TR is recognized as a particular etiologic group because of multiple causes, including implantation, pacing, and device-related. To facilitate the diagnosis of CIED-induced TR, candidates should undergo a comprehensive baseline echocardiogram in preparation for CIED installation and routine echocardiographic follow-ups. Medicinal treatments and percutaneous and surgical procedures are available for treating CIED-induced TR. A definitive treatment may necessitate lead repositioning or removal. Determining the precise mechanism of TR is basic for managing this illness, and determining whether corrective intervention is necessary and safe.

ADDITIONAL INFORMATION

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Contribution of the authors. RZA and SLP are responsible for manuscript preparation, data collection, data analysis, manuscript editing, and manuscript review. SLP is responsible for concept design, final review, and as the corresponding author.

Consent for publication. Written consent was obtained from the patient's parents for publication of relevant medical information and all of accompanying images within the manuscript.

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