JOURNAL OF COMMUNITY HOSPITAL INTERNAL MEDICINE PERSPECTIVES
2021, VOL. 11, NO. 6, 793–798
https://doi.org/10.1080/20009666.2021.1967569

Tricuspid insufficiency after cardiac-implantable electronic device placement

Nismat Javed [a], Raafe Iqbal[b], Jahanzeb Malik [c], Ghazanfar Rana [d], Waheed Akhtar[e] and Syed Muhammad Jawad Zaidi [f]

[a]Department of Medicine, Shifa Tameer-e-Millat University, Islamabad, Pakistan; [b]Department of Cardiology, Pakistan Ordinances Factory Hospital, Wah Cantt, Pakistan; [c]Department of Cardiology, Rawalpindi Institute of Cardiology, Rawalpindi, Pakistan; [d]Department of Cardiology, St. Lukes General Hospital, Kilkenny, Ireland; [e]Department of Cardiology, Abbas Institute of Medical Sciences, Muzaffarabad, Pakistan; [f]Department of Medicine, Rawalpindi Medical University, Rawalpindi, Pakistan

ABSTRACT

Objective

Device-related **e**stimates of incidence and significance of tricuspid regurgitation (TR) is mainly based on case reports and small observational studies. We sought to determine whether right-heart device implantation increased the risk of TR in this interventional study.

Methods

All patients who underwent permanent pacemaker (PPM) or other device implantation were assessed for degree of TR at one year. The data collected was analyzed on IBM SPSS version 26. Descriptive statistics were applied for qualitative variables. Mean and standard deviation were applied for quantitative variables. Regression analysis and paired t-tests were applied for the degree of change and predictors of TR.

Results

Out of 165 participants, 73.94% were male. The mean age of the participants was 59.86 ± 12.03 years. Dual-chamber pacemaker (DDDR) was the most common device implanted (78.18%) causing significant TR and drop in left ventricular ejection fraction as compared to other devices (p-value < 0.05). The paired t-test for changes in ejection fraction (LVEF) and TR were also significant (p-value < 0.05). A regression model predicted significant TR to depend on baseline LVEF (p-value < 0.05).

Conclusion

Device-related worsening of TR is related to mechanical mechanisms. It is significantly associated with DDDR pacemakers after a 1-year follow-up.

ARTICLE HISTORY
Received 27 May 2021
Accepted 9 August 2021

KEYWORDS
Tricuspid regurgitation; ejection fraction; implantation devices; heart block; heart failure

1. Introduction

The technology of endocardial transvenous lead for permanent cardiac pacing has advanced since the advent of this procedure [1]. These pacing leads have become thin, highly flexible due to multi-helical alloy with long-lasting covering sheaths being used [2]. However, despite its size and easy maneuverability, the anatomy mandates that the lead be placed across the tricuspid valve [3]. This is associated with the risk of tricuspid valve dysfunction [2,4].

Tricuspid regurgitation (TR) is a frequently seen valvular lesion on echocardiography with about 1.6 million people in the USA being affected. The incidence is less than 0.1% worldwide [5]. Several isolated presentations of pulvonic valve vegetation are also seen with right heart device placement [6]. Significant TR is associated with increased risk for

CONTACT Jahanzeb Malik Heartdoc86@gmail.com Rawalpindi Institute of Cardiology, Rawal Road, Rawalpindi 46000, Pakistan

developing a variety of complications, which include congestive cardiac failure, elevated mortality, decreased functional class, and peripheral edema [7].

The mechanisms include interference of valve coaptation, perforation of the valve by lead entrapment, and asynchrony of the right and left ventricular activation by the permanent pacemaker (PPM) [8]. Asynchrony is specifically true for patients with single-chamber devices in situ as it causes pseudo-regurgitation as a result of atrial contraction against a closed tricuspid valve [1,9]. Despite, correction by upgrading the single chamber device in place, significant TR requires emergent lead retraction [10–12].

Therefore, the objective of this study was to determine whether right-heart device implantation increased the risk of TR. The study also aimed at determining predictors for the possible change.

2. Methods

This prospective, interventional study was conducted at Abbas Institute of Medical Sciences between November 2019 to November 2020. All patients provided informed written consent, and Institutional Ethical Board approval was granted before data collection (Study ID#AIMS/08/19). The sample size was calculated using a software. A preliminary report from the registered patients for pacemaker placement revealed about 286 patients who had received implantation devices. Using this expected number, the sample size was about 165 patients when assuming an anticipated frequency of 0.5 and confidence intervals of 95%. The exclusion criteria, apart from patients' age less than 18 years and inability to give consent, for the study were as follows:

- Previous known regurgitation valve disease
- Known structural heart disease, such as Hypertrophic Obstructive Cardiomyopathy (HOCM), Ebstein's anomaly, flail leaflet, rupture of valve or prolapse
- Prior coronary artery by-pass
- Prior device in place including permanent pacemaker, implantable cardioverter defibrillator, and cardiac resynchronization device
- Multiple leads in place from previous devices,
- Previous device explantation and reimplantation
- History of infected wound from device implantation
- History of lead associated endocarditis or native valve endocarditis
- History of rheumatic fever
- Suboptimal visualization of the heart, such as suboptimal window of acoustic artifacts due to leads or otherwise.

All the devices were put in the hospital electrophysiology lab. In dual-chamber, rate-modulated pacemaker (DDDR) and implantable cardioverter defibrillator (ICD), one lead was placed in right ventricular (RV) apex and other lead in right atrial appendage (RAA). In single chamber pacemaker (VVIR), a single lead was placed in RV apex. As for cardiac resynchronization device (CRT), a third lead was placed in the coronary sinus. The devices were inserted according to the broad categories of heart block and failure with DDDR and VVIR used for heart blocks and CRT used for heart failure.

The baseline variables were recorded in the in-house data entry system (HMIS). This included age, gender, comorbid, cause of device insertion, and the type of device to be implanted. The number of leads inserted were also assessed as demonstrated by chest X-ray in the hospital image viewer (PACS) or by implantation report. All participants underwent standard two-dimensional echocardiography with the same echocardiography machine (Vivid 7, General Electric Ultrasound) and 2.5 to 3 MHz transducer was used to assess the pertinent cardiac structures and measurements. Images included parasternal, apical, and subcostal views. Tricuspid valve regurgitation was estimated visually, by a qualitative method of eyeballing the jet of tricuspid regurgitation and calculating the jet area, using color flow Doppler. The regurgitation was also approximated quantitatively using vena contracta width. There were different categories for width used to assess grade of tricuspid regurgitation; vena contracta <0.50 cm was grade 1+, ≥0.50 but <0.69 cm was grade 2+ and ≥0.69 cm was grade 3+ [13]. In addition to TR, echocardiographic characteristics included left ventricular ejection fraction (LVEF) by visual estimation and modified Simpson's method. Other parameters such as left atrial volume index (LAVI) and right atrial volume index (RAVI) were also measured. Patients with acoustic shadows and artifacts due to leads were eliminated by taking measurements in right ventricular outflow tract (RVOT) view, parasternal short axis (PSAX), substernal and four chamber view. The prevalence of significant TR was assessed by comparing the echocardiograms before device implantation and following device implantation. Significant TR was defined as an increase of more than one degree from baseline, that is, an increase ≥2. Echocardiogram was followed up after 1 year to see the degree of TR change overtime.

Descriptive statistics were used for qualitative variables. Mean and standard deviation were used for variables such as age. The proportion of participants with significant TR using both quantitative and qualitative techniques were compared using chi-square test. Student's t-test was used to determine the significance of changes in TR and ejection fraction before and after intervention for all devices. Regression analysis was done to determine the association of predictors with the risk of significant TR.

The data were analyzed using IBM Statistical Software for Social Sciences (SPSS) version 26. A p-value of <0.05 was considered significant.

3. Results

There were 165 participants in the study. Out of 165 participants, 122 participants (73.94%) were male and 43 participants were female (26.06%). The mean age of the participants was 59.86 ± 12.03 years. The characteristics of the patients are shown in Table 1.

There were no patients with significant TR at the start of the study. There were 11 participants (6.67%) with atrial fibrillation at the start of the study. After the intervention, 5 additional participants (9.69%) had developed atrial fibrillation. This increase in the number of participants was not significant ($p > 0.05$). 41.20% of the participants had developed significant TR at the 1-year follow-up ($p < 0.05$).

The indications and proportions of different devices used in the study are shown in Table 2.

The differences in grades of TR did not vary when both the vena contracta technique and qualitative technique were compared ($p > 0.05$). The change in mean LVEF at 1-year follow-up was significant ($p < 0.05$). The paired t-test for the mean vena contracta width, at baseline and at follow-up, was significant ($p < 0.05$). The independent samples test for changes in TR and TR before intervention revealed that there were significant differences in the distribution of participants ($p < 0.05$). The changes in LVEF and TR were not significantly determined by gender, type of device, cause of device insertion, and comorbid conditions ($p > 0.05$). The changes in regurgitation and LVEF for the devices are shown in Table 3.

The paired t-tests for mean RAVI, LAVI and annular tricuspid diameter, at baseline and at 1-year follow-up, were all significant ($p < 0.05$). The difference in changes in left atrium volume index at 1-year follow-up was significant for patients who had either ischemic heart disease or smoked ($p < 0.05$). The changes in echocardiographic parameters across all devices are shown in Table 4.

A logistic regression model was used to determine predictors of significant TR. The model predicted significant TR from many independent variables. After adjustments, the independent predictor for the outcome was baseline LVEF. However, not all the variables significantly added to the results as shown in Table 5. No statistical difference was observed in level of TR between devices.

The patients were managed conservatively except for cases which had either grade 3 tricuspid regurgitation or symptoms of right heart failure in which cardiac surgery consultation was requested for tricuspid repair or lead extraction. However, the management of tricuspid regurgitation was beyond the focus of the study.

Table 1. Baseline and follow-up characteristics.

Characteristics (Chronic)		
Age; mean ± SD	59.86 ± 12.03	
Females; n (%)	43 (26.06%)	
Co-morbid conditions n (%)		
Hypertension	77 (46.67)	
Ischemic Heart Disease	73 (44.20)	
Chronic Kidney Disease	15 (9.13)	
Smokers	54 (32.70)	
Characteristics (Clinical)	Baseline	1-year follow-up
Severity of TR; n (%)		
None (0)	140 (84.80)	50 (30.30)
Mild (1+)	25 (15.20)	46 (27.90)
Moderate (2+)	0 (0.00)	40 (24.20)
Severe (3+)	0 (0.00)	29 (17.60)
LA volume (Mean±S.D)	27.16 ± 7.57	30.81 ± 15.91
RA volume (Mean±S.D)	25.86 ± 3.15	31.07 ± 6.30
Annular tricuspid diameter (Mean±S.D; cm)	25.71 ± 2.98	27.32 ± 4.11
LVEF (Mean±S.D; %)	37.24 ± 14.55	33.09 ± 14.21
Vena contracta width (Mean±S.D; cm)	0.07 ± 0.01	0.45 ± 0.20

TR-tricuspid regurgitation, LA-left atrium, RA-right atrium, LVEF-left ventricular ejection fraction, SD-standard deviation, n-frequency.

Table 2. Devices used and their indications.

	Devices			
Indications	DDDR (n, %)	VVIR (n, %)	ICD (n, %)	CRT (n, %)
AV block	128 (77.58)	14 (8.48)	0 (0.00)	0 (0.00)
Scar VT	1 (0.61)	0 (0.00)	5 (3.03)	0 (0.00)
Heart Failure	0 (0.00)	0 (0.00)	0 (0.00)	5 (3.03)
Ischemic VT	0 (0.00)	0 (0.00)	12 (7.27)	0 (0.00)

DDDR- dual-chamber, rate-modulated pacemaker, VVIR- single chamber pacemaker, ICD- implantable cardioverter defibrillator, CRT- cardiac resynchronization device, n-frequency.

4. Discussion

In our study, we found that there was about a 70:30 ratio of male to female participants. The female participants (26.06%) were similar to the percentage of participants (22.00%) in another study focusing on electrical failure secondary to pacing leads [14]. The mean age of our participants was 59.86 ± 12.03 years. In contrast, a 2015 study reported higher mean age of 74.00 ± 14.00 years [15]. The ejection fraction in our study decreased after the placement of leads from 37% to about 33%. A similar result was noted in another study that determined left ventricular ejection fraction less than 40% and mid-range ejection fraction as predictors for deterioration of cardiac function [16].

In our study, the grade of tricuspid regurgitation had increased at a 1-year follow-up and 41.20% of the participants had developed a significant tricuspid regurgitation at 1-year follow-up. This was in contrast to another study which concluded that 10% of the participants had developed significant tricuspid regurgitation and had an increase of two grades in the phenomenon [17]. This was surprising considering the age of the participants as less than 60 years. The possible reasons for the findings include the presence

Table 3. Left ventricular ejection fraction and tricuspid regurgitation across devices.

	LVEF			TR		
Device	Baseline LVEF (Mean±S.D)	1-year follow-up LVEF (Mean±S.D)	*p*-value	Baseline vena contracta (Mean±S.D)	1-year follow-up vena contracta (Mean±S.D)	*p*-value
DDDR	37.87 ± 15.04	33.91 ± 14.37	0.03	0.08 ± 0.03	0.45 ± 0.20	<0.05
VVIR	35.36 ± 12.48	27.86 ± 14.10	0.15	0.00 ± 0.00	0.51 ± 0.17	<0.05
ICD	36.47 ± 12.47	32.35 ± 13.00	0.35	0.09 ± 0.02	0.48 ± 0.21	<0.05
CRT	29.00 ± 14.32	27.50 ± 13.52	0.87	0.20 ± 0.10	0.30 ± 0.17	0.29

DDDR- dual-chamber, rate-modulated pacemaker, VVIR- single chamber pacemaker, ICD- implantable cardioverter defibrillator, CRT- cardiac resynchronization device, LVEF-left ventricular ejection fraction, TR- tricuspid regurgitation, S.D.-standard deviation.

Table 4. LAVI, RAVI and tricuspid annular diameter across devices.

Parameter	Device	Baseline (Mean±S.D)	1-year followup (Mean±S.D)	*p*-value
LAVI	DDDR	25.26 ± 4.29	29.24 ± 16.76	<0.05
	VVIR	47.57 ± 3.23	48.50 ± 2.77	0.40
	ICD	24.47 ± 4.80	27.41 ± 5.28	0.09
	CRT	28.20 ± 5.54	33.40 ± 4.56	0.14
RAVI	DDDR	26.04 ± 3.04	31.14 ± 6.23	<0.05
	VVIR	24.71 ± 2.40	32.14 ± 6.89	<0.05
	ICD	25.06 ± 3.05	30.47 ± 7.05	<0.05
	CRT	27.20 ± 3.56	28.40 ± 4.39	0.65
Tricuspid annular diameter (cm)	DDDR	25.46 ± 2.92	26.95 ± 4.17	<0.05
	VVIR	26.57 ± 3.18	27.86 ± 3.94	0.33
	ICD	26.82 ± 3.45	29.63 ± 3.74	<0.05
	CRT	27.00 ± 2.12	27.80 ± 1.64	0.52

LA-left atrial volume index, RA-right atrial volume index, DDDR- dual-chamber, rate-modulated pacemaker, VVIR- single chamber pacemaker, ICD- implantable cardioverter defibrillator, CRT- cardiac resynchronization device, S.D.-standard deviation.

Table 5. Regression analysis.

	Adjusted Estimates			Unadjusted estimates		
Variables	Risk	95% CI	*p*-value	Risk	95% CI	*p*-value
Age	0.99	0.97–1.02	0.68	1.00	0.97–1.03	0.94
Gender	1.37	0.65–2.88	0.41	1.04	0.45–2.42	0.93
Type 2 Diabetes Mellitus	1.58	0.80–3.15	0.18	2.11	0.99–4.51	0.54
Hypertension	1.70	0.79–3.62	0.17	2.15	0.91–5.10	0.08
Chronic Kidney Disease	0.48	0.17–1.33	0.16	0.33	0.10–1.09	0.06
Ischemic heart disease	1.21	0.62–2.37	0.58	1.18	0.56–2.48	0.67
Smoking	0.89	0.44–1.08	0.75	1.01	0.44–2.29	0.99
Baseline AF	0.43	0.13–1.51	0.19	0.72	0.11–4.99	0.74
Baseline LVEF	1.06	1.03–1.09	0.00	1.05	1.03–1.09	0.00
Baseline TR	7.37	2.59–20.95	0.00	3.26	0.95–10.99	0.06
Baseline LAVI	0.98	0.94–1.02	0.27	0.99	0.93–1.06	0.73
Baseline tricuspid annular diameter	0.96	0.86–1.07	0.47	0.97	0.85–1.09	0.58

AF-atrial fibrillation, LVEF-left ventricular ejection fraction, TR-tricuspid regurgitation, LA-left atrial volume index.

of multiple comorbid conditions amongst the patients. There is a significant risk of tricuspid regurgitation (26.50%) with left heart ischemic disease [18].

The most common device used was DDDR. This device had decreased ejection fraction significantly by about 4% in our study. This finding is comparable to a study that investigated cardiomyopathy due to pacemaker leads and DDDR was associated with a high incidence of cardiomyopathy characterized by a fall in ejection fraction by 10% [19]. In contrast to our study findings of a non-significant decrease in ejection fraction in patients with CRT, a recent study had shown a significant improvement in LVEF [20]. In our study, except for CRT, all devices were associated with an increase in tricuspid regurgitation which is in contrast to a previous study suggesting VVIR as the least acceptable device due to the risk of significant TR ($p < 0.05$) [21].

Our study showed that DDDR insertion was associated with significantly increased LAVI, RAVI and tricuspid annular diameter, VVIR insertion was associated with significantly increased RAVI and ICD was associated with significantly increased RAVI and tricuspid diameter. These findings were in contrast to a 2019 study which mentioned that device implantation did not significantly impact the dimensional volumes of the chambers [20]. The possible reason for the finding in our study is related to LV dysfunction as seen by reduced LVEF ejection fractions. Owing to LV dysfunction and positive remodeling, there is gradual dilation of cardiac chambers leading to both mitral and tricuspid regurgitation.

Our study did not determine age to be a significant predictor of worsening TR after lead placement as compared to another study [17]. The number of leads did not predict TR and the position of leads was more important [22]. The leads impinging on the anterior leaflet, the anteroposterior or anteroseptal commissure, and interfering with leaflet mobility were associated with a higher risk [23]. Baseline LVEF was an important predictor as explained by previous findings. This is attributable to some mechanisms including technical difficulties experienced during the pacing technique, pacing burden load, lead location, and even, number [24]. However, in contrast to a recent study, atrial fibrillation status was not an important predictor for significant TR [20]. The possible explanation for this is that in case of atrial fibrillation, the central pacemaker is implanted and in that case, the device implantation, not atrial fibrillation, would be a significant predictor for TR.

Although not extensively explored in our study, surgery, including lead extraction or valve repair, has been considered an efficacious procedure in patients with class IV heart failure secondary to tricuspid regurgitation, and therefore, these specific cases were sent for a surgery review [25].

In contemporary electrophysiology, introduction of leadless pacemakers has demonstrated safety and efficacy in short and intermediate follow-up as an alternative to transvenous pacing. This technology shows promise for cardiac pacing devices in the future. The LEADLESS trial was the first human trial for leadless pacing using a Nanostim device. The trial enrolled 33 patients with successful implantation achieved in 97% patients. The complication free rate was 94% at three months. Several other clinical trials have been conducted till now and show benefit of this technology for select patients [26].

5. Study limitations

The limitations of the study include a small sample size and center-specific study. Additionally, the time for lead insertion and its subsequent position were not investigated. The results of the study were based on techniques which yielded similar results for significant TR. Another limitation of the study was that right ventricular systolic pressures were not measured because the centre does not include the parameter under normal circumstances, considering that the values for these pressures have not been well-defined. The management for device-related TR was beyond the scope of this study and therefore, the data as well as analysis were not extended to that stage.

6. Conclusion

Worsening of TR is a common finding from baseline to 1-year with a device in the right ventricle, especially after PPM implantation. This is due to either mechanical or physiological mechanisms. Clinical consideration includes the extraction of the offending leads. Large, well-controlled prospective studies are need to assess the incidence and timing of TR along with associated prognosis and mortality.

Disclosure statement

The authors declare that they have no competing interests.

Availability of data and material

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Author contributions

NJ; concept, methodology, analysis, first draft, final draft; RI; data curation, methodology, first draft; JM; concept, first draft; GR; data curation, supervision; WA; data curation, final draft; SMJZ; first draft, final draft.

ORCID

Nismat Javed http://orcid.org/0000-0002-8756-927X
Jahanzeb Malik http://orcid.org/0000-0002-9343-4187
Ghazanfar Rana http://orcid.org/0000-0002-1449-916X
Syed Muhammad Jawad Zaidi http://orcid.org/0000-0002-3513-6974

References

[1] Najib MQ, Vittala SS, Challa S, et al. Predictors of severe tricuspid regurgitation in patients with permanent pacemaker or automatic implantable cardioverter-defibrillator leads. Tex Heart Inst J. 2013;40(5):529–533.
[2] Pokorney SD, Parzynski CS, Daubert JP, et al. Temporal trends in and factors associated with use of single- versus dual-coil implantable cardioverter-defibrillator leads: data from the NCDR ICD registry. JACC Clin Electrophysiol. 2017;3(6):612–619.
[3] Delling FN, Hassan ZK, Piatkowski G, et al. Tricuspid regurgitation and mortality in patients with transvenous permanent pacemaker leads. Am J Cardiol. 2016;117(6):988–992.
[4] Carlson MD. ICD leads and postmarketing surveillance. N Engl J Med. 2012;366(10):967.
[5] Pappalardo OA, Votta E, Selmi M, et al. 4D MDCT in the assessment of the tricuspid valve and its spatial relationship with the right coronary artery: a customized tool based on computed tomography for the planning of percutaneous procedures. J Cardiovasc Comput Tomogr. 2020;14(6):520–523.
[6] Goud A, Abdelqader A, Dahagam C, et al. Isolated pulmonic valve endocarditis presenting as neck pain. J

Community Hosp Intern Med Perspect. 2015;5 (6):29647.
[7] Lee JH, Kim TH, Kim WS. Permanent pacemaker lead induced severe tricuspid regurgitation in patient undergoing multiple valve surgery. Korean J Thorac Cardiovasc Surg. 2015;48:129–133.
[8] Saran N, Said SM, Schaff HV, et al. Outcome of tricuspid valve surgery in the presence of permanent pacemaker. J Thorac Cardiovasc Surg. 2018;155 (4):1498–1508.e3.
[9] Dahou A, Magne J, Clavel MA, et al. Tricuspid regurgitation is associated with increased risk of mortality in patients with low-flow low-gradient aortic stenosis and reduced ejection fraction: results of the multicenter TOPAS study (True or Pseudo-Severe Aortic Stenosis). JACC Cardiovasc Interv. 2015;8(4):588–596.
[10] Hayashi T, Ono H, Kaneko Y. Echocardiographic assessment of ventricular contraction and synchrony in children with isolated complete atrioventricular block and epicardial pacing: implications of interventricular mechanical delay. Echocardiography. 2018;35:1370–1377.
[11] Thibault B, Ritter P, Bode K, et al. Dynamic programming of atrioventricular delay improves electrical synchrony in a multicenter cardiac resynchronization therapy study. Heart Rhythm. 2019;16(7):1047–1056.
[12] Mazzone P, Melillo F, Radinovic A, et al. Use of the new rotating dilator sheath TightRail™ for lead extraction: a bicentric experience. J Arrhythm. 2020;36(2):343–350.
[13] Eleid MF. A step forward for transcatheter tricuspid valve repair. JACC Cardiovasc Interv. 2017;10 (19):2004–2005.
[14] Khattak F, Gupta A, Alluri K, et al. Rate and predictors of electrical failure in non-recalled defibrillator leads. Indian Pacing Electrophysiol J. 2019;19:100–103.
[15] Fanari Z, Hammami S, Hammami MB, et al. The effects of right ventricular apical pacing with transvenous pacemaker and implantable cardioverter defibrillator on mitral and tricuspid regurgitation. J Electrocardiol. 2015;48:791-797.
[16] He H, Li X, Ke B, et al. Midrange ejection fraction as a risk factor for deterioration of cardiofunction after permanent pacemaker implantation. J Interv Card Electrophysiol. 2019;55:213-24.
[17] Lee RC, Friedman SE, Kono AT, et al. Tricuspid regurgitation following implantation of endocardial leads: incidence and predictors. Pacing Clin Electrophysiol. 2015;38:1267-1274.
[18] Bohbot Y, Chadha G, Delabre J, et al. Characteristics and prognosis of patients with significant tricuspid regurgitation. Arch Cardiovasc Dis. 2019;112:604-614.
[19] Bansal R, Parakh N, Gupta A, et al. Incidence and predictors of pacemaker-induced cardiomyopathy with comparison between apical and non-apical right ventricular pacing sites. J Interv Card Electrophysiol. 2019;56(1):63–70.
[20] Van De Heyning CM, Elbarasi E, Masiero S, et al. Prospective study of tricuspid regurgitation associated with permanent leads after cardiac rhythm device implantation. Can J Cardiol. 2019;35(4):389–395.
[21] Sulke N, Chambers J, Dritsas A, et al. A randomized double-blind crossover comparison of four rate-responsive pacing modes. J Am Coll Cardiol. 1991;17:696-706.
[22] Al-Bawardy R, Krishnaswamy A, Rajeswaran J, et al. Tricuspid regurgitation and implantable devices. Pacing Clin Electrophysiol. 2015;38(2):259–266.
[23] Mediratta A, Addetia K, Yamat M, et al. 3D echocardiographic location of implantable device leads and mechanism of associated tricuspid regurgitation. JACC Cardiovasc Imaging. 2014;7 (4):337–347.
[24] Addetia K, Harb SC, Hahn RT, et al. Cardiac implantable electronic device lead-induced tricuspid regurgitation. JACC Cardiovasc Imaging. 2019;12 (4):622–636.
[25] Polewczyk A, Kutarski A, Tomaszewski A, et al. Lead dependent tricuspid dysfunction: analysis of the mechanism and management in patients referred for transvenous lead extraction. Cardiol J. 2013;20 (4):402–410.
[26] Reddy VY, Knops RE, Sperzel J, et al. Permanent leadless cardiac pacing: results of the LEADLESS trial. Circulation. 2014;129(14):1466–1471.