Pacing-Induced Cardiomyopathy in Right Ventricular Pacemaker Patients: Prevalence and Risk Factors in an Iranian Cohort

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Abstract

Background: Right ventricular pacemakers (RVPs) are widely used to manage advanced atrioventricular blocks, significantly improving patient survival and quality of life. However, pacing-induced cardiomyopathy (PiCM) remains a prevalent and under-recognized complication.

Objectives: The present study aims to assess the prevalence of PiCM in an Iranian population with RVPs and to identify associated risk factors.

Methods: This prospective observational study included 105 patients who received right ventricular apical pacemakers from March 2021 to March 2023. Transthoracic echocardiography was performed before pacemaker implantation and six months post-implantation. The PiCM was defined as either a left ventricular ejection fraction (LVEF) $\leq$ 40% if baseline LVEF was $\geq$ 50%, or a reduction of $\geq$ 10% in LVEF.

Results: At six months, 17.1% (n = 18) of patients developed PiCM. A higher prevalence of diabetes mellitus (DM), ischemic heart disease (IHD), and chronic kidney disease (CKD) was observed among the PiCM group. Diabetes mellitus (OR = 3.53; 95% CI 1.78 - 6.45; P = 0.012), IHD (OR = 8.07; 95% CI 5.20 - 29.64; P < 0.001), CKD (OR = 5.29; 95% CI 1.47 - 11.29; P = 0.028), baseline LVEF <5 0% (OR = 5.67; 95% CI 1.15 - 16.38; P = 0.017), paced QRS duration (pQRSd) (OR = 1.01; 95% CI 1.001 - 1.08; P = 0.038), and increased pacing burden (OR = 1.06; 95% CI 1.008 - 1.26; P = 0.036) were identified as independent risk factors for PiCM. Receiver operator curve analysis demonstrated that a pacing burden > 65% can accurately predict PiCM (AUC = 0.651; P = 0.033).

Conclusions: The prevalence of PiCM among Iranian patients with RVPs aligns with global reports. These findings underscore the importance of routine echocardiographic surveillance and risk stratification in patients with RVPs. Early recognition of high-risk individuals, particularly those with comorbidities or elevated pacing burden, may inform pacing strategies and guide follow-up to reduce the incidence of PiCM.

Keywords: Risk Factor, Heart Failure, Pacemaker Induced Cardiomyopathy, Right Ventricular Pacing

1. Background

The use of right ventricular pacemakers (RVPs) is a critical component in the management of advanced atrioventricular blocks (1), providing life-saving therapy and significantly enhancing the quality of life for millions of patients worldwide. Globally, more than 700,000 pacemakers are implanted annually (2), with a substantial proportion being RVP devices (1). While the clinical benefits of RVPs are well-established, there is increasing recognition of its significant complications, including pacing-induced cardiomyopathy (PiCM) (3), atrial fibrillation (AF) (4), and heart failure (HF) (5). The PiCM is a relatively common yet under-recognized complication of RVPs. Current estimates suggest that PiCM may develop in more than 10% of patients with RVP, depending on the population studied and the diagnostic criteria employed (6). Patients with PiCM typically present with new-onset HF symptoms after pacemaker implantation (1), leading to increased healthcare utilization and reduced quality of life. Moreover, the insidious progression of PiCM often goes

undiagnosed due to the lack of unanimous diagnostic criteria, highlighting the need for early recognition and intervention, particularly before a significant reduction in left ventricular ejection fraction (LVEF) occurs. A major challenge in clinical practice and research is the lack of a universally accepted definition of PiCM, which limits comparability across studies and may delay diagnosis [7]. The PiCM is usually defined by either a reduction of ≥ 10% in LVEF from baseline without any identifiable cause other than right ventricular pacing or an LVEF of ≤ 40% if the baseline LVEF was ≥ 50% [8]. While the pathophysiology of PiCM is primarily attributed to pacing-induced ventricular dyssynchrony, often due to right ventricular apical pacing [9-11], its development is likely multifactorial. Contributing factors include patient characteristics and device-related variables such as pacing burden and lead position [12]. Despite extensive research on PiCM, the predictors of this condition remain incompletely defined, underscoring the need for further investigation.

2. Objectives

The present study aims to assess the prevalence and determine independent risk factors of PiCM in an Iranian population with RVPs.

3. Methods

3.1. Study Protocol

This prospective observational study included patients who underwent right ventricle apical pacemaker implantation at Golestan and Imam Khomeini hospitals (tertiary centers in Ahvaz, Iran) between March 2021 to March 2023, using convenience sampling. The study followed the Declaration of Helsinki and was approved by Ahvaz Jundishapur University of Medical Sciences (IR.AJUMS.HGOLESTAN.REC.1401.064). After a thorough explanation of the study protocol, informed consent was obtained from participants. Demographic and medical history data were collected from records. All participants underwent transthoracic echocardiography by a board-certified echocardiologist before implantation and at six months post-implantation.

3.2. Echocardiographic Study

The LVEF was assessed using a Vivid E9 ultrasound machine (GE Vingmed Ultrasound, Horten, Norway) applying the biplane Simpson's method of disc summation. This method was used in accordance with the guidelines of the American Society of Echocardiography and the European Association of Cardiovascular Imaging [13].

3.3. Definition

The PiCM was defined using the following criteria: (1) An LVEF ≤ 40% in patients with a baseline LVEF of ≥ 50% prior to pacemaker implantation; or (2) a reduction in LVEF of ≥ 10% in patients with a baseline LVEF < 50% prior to implantation. The chronic kidney disease (CKD) was defined as an estimated glomerular filtration rate of less than $60 \text{ mL/min/1.73 m}^2$ and/or an albumin-to-creatinine ratio of ≥ 30 mg/g, calculated using the CKD-EPI equation [14-16]. Diabetes mellitus (DM) was defined by a history of glucose-lowering medication use or lab results meeting American Diabetes Association diagnostic criteria [17]. Hyperlipidemia (HLP) was defined by current use of lipid-lowering therapy or meeting diagnostic criteria by the National Cholesterol Education Program ATP III and ACC/AHA guidelines [18].

3.4. Exclusion Criteria

Patients were excluded if they developed new-onset HF or cardiomyopathy due to causes other than PiCM, such as myocardial infarction, uncontrolled hypertension (HTN), and acute valvular diseases. Those unable to attend the six-month follow-up echocardiography were also excluded from the final analysis.

3.5. Statistical Analysis

Continuous data were expressed as means and standard deviations, while categorical data were presented as frequencies and percentages. The Kolmogorov-Smirnov test was employed to assess data normality. Group comparisons used independent samples *t*-tests or Mann-Whitney U tests (for non-normal data), and chi-square tests for categorical variables. Regression analysis evaluated associations with PiCM. Analyses were performed using SPSS version 27.0 (IBM Corp., Armonk, NY), with $P < 0.05$ considered statistically significant.

4. Results

Initially, 110 patients were enrolled in the study. One patient was excluded due to myocardial infarction during follow-up, and four patients were excluded due to missed follow-up, leaving 105 for final analysis. The mean age of the participants was 71.2 ± 8.96 years, with a female predominance (n = 63, 60.0%). Primarily, 59

Table 1. Demographic Findings of Study Participants [a]

Variables	Total Population (N = 105)	PiCM (n = 18)	Non-PiCM (n = 87)	P-Value
Age (y)	71.25 ± 8.96	73.05 ± 9.40	70.39 ± 9.07	0.262
Sex				0.341
Male	42 (40.0)	9 (50.0)	33 (38.0)	
Female	63 (60.0)	9 (50.0)	54 (62.0)	
HTN	75 (71.4)	13 (72.2)	62 (71.2)	0.935
DM	49 (46.7)	16 (88.8)	33 (37.9)	< 0.001
Previous history of HF	32 (30.5)	8 (44.4)	24 (27.5)	0.157
IHD	28 (26.7)	12 (66.6)	16 (18.4)	< 0.001
HLP	10 (9.5)	2 (11.1)	8 (9.2)	0.672
CKD	5 (4.7)	3 (16.7)	2 (2.3)	0.009
Smoking	21 (20.0)	6 (33.3)	15 (17.2)	0.120

Abbreviations: PiCM, pacing-induced cardiomyopathy; HTN, hypertension; DM, diabetes mellitus; HF, heart failure; IHD, ischemic heart disease; HLP, hyperlipidemia; CKD, chronic kidney disease.

[a] Values are expressed as No. (%) or mean ± SD.

patients (56.2%) had complete heart block, 40 had high-grade atrioventricular block (38.1%), and the remaining six patients had either low response AF or sick sinus syndrome. The most prevalent comorbidity among the study population was HTN (n = 75, 71.4%), followed by DM (n = 49, 46.7%) and HF (n = 32, 30.5%). At six months, 18 patients (17.1%) developed PiCM (Table 1). No significant differences in demographics or baseline LVEF were found between included and excluded patients (Appendix 1 in Supplementary File).

4.1. Comparison Between Pacing-Induced Cardiomyopathy and Non-pacing-Induced Cardiomyopathy Patients

In this study, PiCM patients had significantly higher rates of DM (P < 0.001), IHD (P < 0.001), and CKD (P = 0.009). They also exhibited lower LVEF after six months (P = 0.021), longer paced QRS duration (pQRSd) (P = 0.042), and higher pacing burden (P = 0.032). No significant differences were observed between the PiCM and non-PiCM groups regarding age, gender, HTN, HF, HLP, smoking, type of AV block, or native QRS duration (nQRSd) (All P > 0.05) (Tables 1 and 2).

4.2. Predictors of Pacing-Induced Cardiomyopathy

Univariable regression revealed significant associations between PiCM and DM (OR = 13.09; 95% CI 2.82 - 60.60; P = 0.001), IHD (OR = 11.87; 95% CI 6.16 - 57.68; P < 0.001), CKD (OR = 8.50; 95% CI 1.30 - 55.23; P = 0.025), baseline LVEF < 50% (OR = 2.93; 95% CI 1.04 - 8.27; P = 0.042), pQRSd (OR = 1.04; 95% CI 1.002 - 1.09; P = 0.049), and pacing burden (OR = 1.06; 95% CI 1.005 - 1.12; P = 0.032) (Table 3). In multivariable analysis, independent predictors of PiCM included DM (OR = 3.53; 95% CI 1.78 -

6.45; P = 0.012), IHD (OR = 8.07; 95% CI 5.20 - 29.64; P < 0.001), and CKD (OR = 5.29; 95% CI 1.47 - 11.29; P = 0.028). Baseline LVEF < 50% increased PiCM risk 5.67-fold (95% CI 1.15 - 16.38; P = 0.017). Each one millisecond increase in pQRSd was associated with a 1% increase in PiCM odds (95% CI 1.001 - 1.08; P = 0.038) and each percentage increase in pacing burden raised the risk by 6% (95% CI 1.008 - 1.26; P = 0.036) (Table 3). Receiver operator curve analysis demonstrated that pacing burden > 65% can predict PiCM (AUC = 0.651; 95% CI 0.552 - 0.742; P = 0.033) with 94.4% sensitivity and 28.7% specificity.

5. Discussion

In this study, 17.1% of patients who underwent RVP developed PiCM within six months. These patients had significantly higher rates of DM, IHD, and CKD, and greater pacing burden. Independent risk factors included baseline LVEF < 50%, and histories of CKD, IHD, and DM. While RVP remains a standard treatment for atrioventricular block, especially with an aging population, it poses risks, including PiCM. Diagnosis is primarily based on LVEF assessment via conventional methods, which are widely accessible and reasonably reproducible (19). Previous studies report PiCM prevalence ranging from 6% to 25%, depending on definitions and follow-up durations (20-25). Our observed rate of 17.1% in apical RVP patients aligns with these findings. While no significant differences were found between the PiCM and non-PiCM groups in our population, consistent with some studies (26-28), other research has identified baseline LVEF as a distinguishing factor (22-24, 29). In our study, patients with baseline LVEF < 50% showed higher odds of developing PiCM.

Table 2. Clinical Findings of Study Participants [a]

Variables	Total Population (N = 105)	PiCM (n = 18)	Non-PiCM (n = 87)	P-Value
Primary rhythm				0.094
Complete heart block	59 (56.2)	13 (72.2)	46 (52.9)	
High grade AV block	40 (38.1)	3 (16.7)	37 (43.5)	
Low-response AF/sick sinus syndrome	6 (5.7)	2 (11.1)	4 (4.6)	
Baseline LVEF (%)	48.27 ± 8.65	46.38 ± 9.20	48.67 ± 8.54	0.309
LVEF at 6-months follow-up (%)	49.46 ± 11.92	43.61 ± 17.72	50.68 ± 10.06	0.021
nQRSd (ms)	106.31 ± 11.76	104.09 ± 17.14	106.78 ± 10.65	0.387
pQRSd (ms)	145.33 ± 11.62	150.43 ± 14.64	144.28 ± 11.0	0.042
Pacing burden (%)	72.80 ± 11.62	78.22 ± 11.59	71.68 ± 11.68	0.032

Abbreviations: PiCM, pacing-induced cardiomyopathy; AV, atrioventricular; AF, atrial fibrillation; LVEF, left ventricular ejection fraction; nQRSd, native QRS duration; pQRSd, paced QRS duration.

[a] Values are expressed as No. (%) or mean ± SD.

Similarly, previous studies reported that higher baseline LVEF was associated with a lower PiCM risk, confirming LVEF as an independent predictor (23-25, 29). Variations in baseline LVEF findings could be attributed to methodological differences. Some studies included only patients with LVEF ≥ 50% to exclude those with pre-existing HF, although HF with reduced ejection fraction is typically defined as LVEF ≤ 40%, according to the AHA/ACC/HFSA guidelines. The absence of a standardized PiCM definition further complicates comparisons. While some define PiCM as a ≥ 10% LVEF reduction from a baseline ≥ 50%, others include patients with baseline LVEF < 50% if a further 5 - 10% decline occurs (8). A unified definition could improve early diagnosis and identification of high-risk patients.

In our study, RV pacing burden was significantly higher in patients who developed PiCM, with each percentage increase linked to a 6% rise in risk of PiCM. This aligns with prior studies, including a meta-analysis by Somma et al., which found a 2% increase in PiCM odds per 1% increase in pacing burden (12). While high RV pacing is a recognized risk factor, the optimal threshold remains debated. We identified 65% as a predictive cut-off, compared to 60% reported by Bansal et al. (28), 40% in other studies (8, 30), and even 20% by Kiehl et al. (29). Our higher threshold may reflect the generally elevated pacing burden in our cohort. Although an AUC of 0.651 reflects modest discrimination, this threshold may still aid broader clinical risk assessment. Pacing burden alone is not definitive and should be interpreted alongside baseline LVEF, comorbidities, and QRS duration. Including it in a multifactorial model may improve early identification of at-risk patients. Larger prospective studies are needed to refine pacing burden thresholds for PiCM risk stratification.

The effects of native and pQRSd on the development of PiCM remain controversial, with heterogeneous findings reported in the literature. Khurshid et al. identified nQRSd as a significant predictor of PiCM, while pQRSd was not associated with PiCM in their analysis (31). However, in a subsequent study by the same authors in 2016, no association was found between nQRSd and PiCM, whereas pQRSd was significantly related to PiCM development (32). Similarly, Cho et al. demonstrated that pQRSd was an independent risk factor for PiCM, with no significant association observed for nQRSd (22). A 15-year study by Kim et al. on the South Korean population further supported pQRSd as an independent predictor of PiCM (26). Similarly, we also observed that pQRSd was independently associated with PiCM, while nQRSd did not show any significant association with PiCM development. In contrast with the above-mentioned studies and our findings, a recent meta-analysis by Somma et al. reported that both nQRSd and pQRSd were potential risk factors for PiCM (12). Current evidence suggests that increased pQRSd plays a critical role in PiCM risk, though the findings for nQRSd remain inconclusive. Larger studies are needed to clarify the role of nQRSd, as it has been unexplored in existing literature; for instance, Somma et al.'s meta-analysis included ten studies on pQRSd but only three on nQRSd.

In addition to pacing burden and pQRSd, comorbidities such as DM, IHD, and CKD have been proposed earlier as risk factors of PiCM (12, 22, 33, 34). Other studies have suggested atrioventricular blocks, AF, and prolonged QRS duration prior to pacing with increased PiCM risk (5, 12, 35, 36). Our findings align with these observations, as patients with a history of DM, IHD, or CKD demonstrated significantly increased risk of PiCM. However, some studies have not reported such associations (26-28), likely due to heterogeneity among

Table 3. Associated Factors with Developing Pacing-Induced Cardiomyopathy After Six Months

Variables	Univariable Regression Analysis				Multivariable Regression Analysis			
	OR	95% CI		P-Value	OR	95% CI		P-Value
		Lower	Upper			Lower	Upper	
Age	1.03	0.97	1.09	0.261	-	-	-	-
Male gender	1.63	0.59	4.54	0.344	-	-	-	-
DM	13.09	2.82	60.60	0.001	3.53	1.78	6.45	0.012
HTN	1.04	0.33	3.24	0.935	-	-	-	-
IHD	11.87	6.16	57.68	< 0.001	8.07	5.20	29.64	< 0.001
HLP	1.42	0.27	7.51	0.674	-	-	-	-
CKD	8.50	1.30	55.23	0.025	5.29	1.47	11.29	0.028
Previous history of HF	2.10	0.74	5.95	0.163	-	-	-	-
Smoking	2.40	0.77	7.40	0.128	-	-	-	-
Baseline LVEF < 50%	2.93	1.04	8.27	0.042	5.67	1.15	16.38	0.017
Having complete heart block	2.31	0.76	7.06	0.139	-	-	-	-
nQRSd (each 1 ms)	0.994	0.96	1.01	0.580	-	-	-	-
pQRSd (each 1 ms)	1.04	1.002	1.09	0.049	1.01	1.001	1.08	0.038
Pacing burden (each 1%)	1.05	1.005	1.12	0.032	1.06	1.008	1.26	0.036

Abbreviations: OR, odds ratio; CI, confidence interval; DM, diabetes mellitus; HTN, hypertension; IHD, ischemic heart disease; HLP, hyperlipidemia; CKD, chronic kidney disease; HF, heart failure; LVEF, left ventricular ejection fraction; nQRSd, native QRS duration; pQRSd, paced QRS duration; ms, Millisecond.

study populations, varying PiCM definitions, and differences in follow-up durations. Currently, our ability to discriminate patients with high risk of PiCM from others is limited, and these inconsistencies highlight the need for more research to clarify the role of comorbidities in PiCM risk and to improve patient monitoring and outcomes.

5.1. Conclusions

The prevalence of PiCM in the Iranian population is consistent with global data. Diabetes mellitus, IHD, CKD, baseline LVEF < 50%, prolonged pQRSd, and increased pacing burden were identified as independent risk factors in patients with right ventricular apical pacemakers. These findings highlight the need for early detection and close monitoring of high-risk patients to mitigate PiCM-related complications. Identifying at-risk individuals may also prompt consideration of alternative pacing strategies, such as cardiac resynchronization therapy or left bundle branch area pacing, instead of right ventricular apical pacing for atrioventricular block management.

5.2. Study Limitations

To our knowledge, this is the first study to assess PiCM risk factors in an Iranian population. However, several limitations must be acknowledged. The relatively small sample size and recruitment from a single region may limit generalizability. A limited sample may also contribute to overfitting in regression models. The six-month follow-up might have been too short to capture all outcomes; longer follow-up could provide more definitive insights. Selection bias may have occurred due to the exclusion of patients lost to follow-up. Although baseline characteristics between included and excluded patients showed no significant differences, this could still affect external validity. The sample size also prevented formal sensitivity analyses to test the robustness of our findings. Lastly, we did not assess reproducibility (e.g., interobserver variability in echocardiographic measurements), which may limit evaluation consistency.

Supplementary Material

Supplementary material(s) is available here [To read supplementary materials, please refer to the journal website and open PDF/HTML].

Footnotes

Authors' Contribution: Study concept and design: B. P. and M. Z.; Acquisition of data: M. Z. and A. M.; Analysis and interpretation of data: M. Z. and A. M.; Drafting of manuscript: B. P., A. M., and M. Z.; Critical revision of the manuscript for important intellectual content: A. M. and B. P.; Statistical analysis: A. M. and M. Z.; Administrative,

Technical, and material support: B. P., M. E. M., and N. A.; Study supervision: B. P., N. A., and M. E. M.

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References

1. Naqvi TZ, Chao CJ. Adverse effects of right ventricular pacing on cardiac function: prevalence, prevention and treatment with physiologic pacing. *Trends Cardiovasc Med*. 2023;**33**(2):109-22. [PubMed ID: 34742888]. https://doi.org/10.1016/j.tcm.2021.10.013.

2. Bernard ML. Pacing Without Wires: Leadless Cardiac Pacing. *Ochsner J*. 2016;**16**(3):238-42. [PubMed ID: 27660571]. [PubMed Central ID: PMC5024804].

3. Hussain MA, Furuya-Kanamori L, Kaye G, Clark J, Doi SA. The Effect of Right Ventricular Apical and Nonapical Pacing on the Short- and Long-Term Changes in Left Ventricular Ejection Fraction: A Systematic Review and Meta-Analysis of Randomized-Controlled Trials. *Pacing Clin Electrophysiol*. 2015;**38**(9):1121-36. [PubMed ID: 26096902]. https://doi.org/10.1111/pace.12681.

4. Kreimer F, Gotzmann M. Pacemaker-induced atrial fibrillation reconsidered-associations with different pacing sites and prevention approaches. *Front Cardiovasc Med*. 2024;**11**:1412283. [PubMed ID: 38957332]. [PubMed Central ID: PMC11217490]. https://doi.org/10.3389/fcvm.2024.1412283.

5. Merchant FM, Hoskins MH, Musat DL, Prillinger JB, Roberts GJ, Nabutovsky Y, et al. Incidence and Time Course for Developing Heart Failure With High-Burden Right Ventricular Pacing. *Circ Cardiovasc Qual Outcomes*. 2017;**10**(6). [PubMed ID: 28630373]. https://doi.org/10.1161/CIRCOUTCOMES.117.003564.

6. Laksono S, Setianto B, Iqbal M, Prawara AS. Understanding Pacemaker-Induced Cardiomyopathy Incidence and Predictors in Patients with Right Ventricular Pacing: A Systematic Review. *Int J Angiol*. 2022;**31**(1):10-5. [PubMed ID: 35221847]. [PubMed Central ID: PMC8881105]. https://doi.org/10.1055/s-0041-1735206.

7. Gavaghan C. Pacemaker Induced Cardiomyopathy: An Overview of Current Literature. *Curr Cardiol Rev*. 2022;**18**(3). e010921196020. [PubMed ID: 34468302]. [PubMed Central ID: PMC9615218]. https://doi.org/10.2174/1573403X17666210901111616.

8. Kaye G, Ng JY, Ahmed S, Valencia D, Harrop D, Ng ACT. The Prevalence of Pacing-Induced Cardiomyopathy (PICM) in Patients With Long Term Right Ventricular Pacing - Is it a Matter Of Definition? *Heart Lung Circ*. 2019;**28**(7):1027-33. [PubMed ID: 30017634]. https://doi.org/10.1016/j.hlc.2018.05.196.

9. Ghani A, Delnoy PP, Ottervanger JP, Ramdat Misier AR, Smit JJ, Elvan A. Assessment of left ventricular dyssynchrony in pacing-induced left

10. bundle branch block compared with intrinsic left bundle branch block. *Europace*. 2011;**13**(10):1504-7. [PubMed ID: 21527389]. https://doi.org/10.1093/europace/eur117.

11. Varma N. Left ventricular conduction delays induced by right ventricular apical pacing: effect of left ventricular dysfunction and bundle branch block. *J Cardiovasc Electrophysiol*. 2008;**19**(2):114-22. [PubMed ID: 17971145]. https://doi.org/10.1111/j.1540-8167.2007.00995.x.

12. Vernooy K, Dijkman B, Cheriex EC, Prinzen FW, Crijns HJ. Ventricular remodeling during long-term right ventricular pacing following His bundle ablation. *Am J Cardiol*. 2006;**97**(8):1223-7. [PubMed ID: 16616030]. https://doi.org/10.1016/j.amjcard.2005.11.044.

13. Somma V, Ha FJ, Palmer S, Mohamed U, Agarwal S. Pacing-induced cardiomyopathy: A systematic review and meta-analysis of definition, prevalence, risk factors, and management. *Heart Rhythm*. 2023;**20**(2):282-90. [PubMed ID: 36356656]. https://doi.org/10.1016/j.hrthm.2022.09.019.

14. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr*. 2015;**28**(1):1-39 e14. [PubMed ID: 25559473]. https://doi.org/10.1016/j.echo.2014.10.003.

15. Kidney Disease: Improving Global Outcomes CG. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. *Kidney Int*. 2024;**105**(4S):S117-314. [PubMed ID: 38490803]. https://doi.org/10.1016/j.kint.2023.10.018.

16. Inker LA, Eneanya ND, Coresh J, Tighiouart H, Wang D, Sang Y, et al. New Creatinine- and Cystatin C-Based Equations to Estimate GFR without Race. *N Engl J Med*. 2021;**385**(19):1737-49. [PubMed ID: 34554658]. [PubMed Central ID: PMC8822996]. https://doi.org/10.1056/NEJMoa2102953.

17. Mousavi FS, Bagheri B, Jalalian R, Nabati M, Moradi A, Mousavi F, et al. Assessing renal function as a predictor of adverse outcomes in diabetic patients undergoing percutaneous coronary intervention. *Acta Cardiol*. 2024;**79**(7):824-32. [PubMed ID: 39377142]. https://doi.org/10.1080/00015385.2024.2410603.

18. American Diabetes Association Professional Practice C. 2. Diagnosis and Classification of Diabetes: Standards of Care in Diabetes-2025. *Diabetes Care*. 2025;**48**(1 Suppl 1):S27-49. [PubMed ID: 39651986]. [PubMed Central ID: PMC11635041]. https://doi.org/10.2337/dc25-S002.

19. Expert Panel on Detection E, Treatment of High Blood Cholesterol in A. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). *JAMA*. 2001;**285**(19):2486-97. [PubMed ID: 11368702]. https://doi.org/10.1001/jama.285.19.2486.

20. Kim SS, Park HW, Jeong HK. Post-Implant Global Longitudinal Strain as a Predictor of Pacing-Induced Cardiomyopathy in Patients with Preserved Ejection Fraction Undergoing Pacemaker Placement. *Rev Cardiovasc Med*. 2025;**26**(1):26173. [PubMed ID: 39867201]. [PubMed Central ID: PMC11760551]. https://doi.org/10.31083/RCM26173.

21. Dor O, Haim M, Barrett O, Novack V, Konstantino Y. Incidence and Clinical Outcomes of Pacing Induced Cardiomyopathy in Patients With Normal Left Ventricular Systolic Function and Atrioventricular Block. *Am J Cardiol*. 2020;**128**:174-80. [PubMed ID: 32650916]. https://doi.org/10.1016/j.amjcard.2020.05.017.

22. Safak E, Ince H, Gkouvatsou L, Schultheiss HP, Ortak J, Caglayan E, et al. Pacing-induced cardiomyopathy in chronic right ventricular apical pacing: a midterm follow-up study. *Eur J Med Res*. 2019;**24**(1):23. [PubMed ID: 31331388]. [PubMed Central ID: PMC6643303]. https://doi.org/10.1186/s40001-019-0386-5.

23. Cho SW, Gwag HB, Hwang JK, Chun KJ, Park KM, On YK, et al. Clinical features, predictors, and long-term prognosis of pacing-induced

cardiomyopathy. *Eur J Heart Fail.* 2019;**21**(5):643-51. [PubMed ID: 30734436]. https://doi.org/10.1002/ejhf.1427.

23. Balli M, Cetin M, Koksal F, Sag FE, Katkat F, Tekin EE, et al. Predictors of Pacemaker-Induced Cardiomyopathy and Importance of Nutritional Status and Prognostic Nutritional Index. *Acta Cardiol Sin.* 2022;**38**(2):151-8. [PubMed ID: 35273436]. [PubMed Central ID: PMC8888326]. https://doi.org/10.6515/ACS.202203_38(2).20211117A.

24. Perla HT, Chandra Srinath Patloori S, Manickavasagam A, Chase D, Roshan J. Do the predictors of right ventricular pacing-induced cardiomyopathy add up? *Indian Heart J.* 2021;**73**(5):582-7. [PubMed ID: 34627573]. [PubMed Central ID: PMC8514412]. https://doi.org/10.1016/j.ihj.2021.07.011.

25. Li DL, Yoneda ZT, Issa TZ, Shoemaker MB, Montgomery JA. Prevalence and predictors of pacing-induced cardiomyopathy in young adult patients (<60 years) with pacemakers. *J Cardiovasc Electrophysiol.* 2021;**32**(7):1961-8. [PubMed ID: 33825250]. https://doi.org/10.1111/jce.15029.

26. Kim JH, Kang KW, Chin JY, Kim TS, Park JH, Choi YJ. Major determinant of the occurrence of pacing-induced cardiomyopathy in complete atrioventricular block: a multicentre, retrospective analysis over a 15-year period in South Korea. *BMJ Open.* 2018;**8**(2). e019048. [PubMed ID: 29439074]. [PubMed Central ID: PMC5829811]. https://doi.org/10.1136/bmjopen-2017-019048.

27. Abdin A, Yalin K, Zink MD, Napp A, Gramlich M, Marx N, et al. Incidence and predictors of pacemaker induced cardiomyopathy: A single-center experience. *J Electrocardiol.* 2019;**57**:31-4. [PubMed ID: 31473477]. https://doi.org/10.1016/j.jelectrocard.2019.08.016.

28. Bansal R, Parakh N, Gupta A, Juneja R, Naik N, Yadav R, 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. [PubMed ID: 31363943]. https://doi.org/10.1007/s10840-019-00602-2.

29. Kiehl EL, Makki T, Kumar R, Gumber D, Kwon DH, Rickard JW, et al. Incidence and predictors of right ventricular pacing-induced cardiomyopathy in patients with complete atrioventricular block and preserved left ventricular systolic function. *Heart Rhythm.* 2016;**13**(12):2272-8. [PubMed ID: 27855853]. https://doi.org/10.1016/j.hrthm.2016.09.027.

30. Sharma AD, Rizo-Patron C, Hallstrom AP, O'Neill GP, Rothbart S, Martins JB, et al. Percent right ventricular pacing predicts outcomes in the DAVID trial. *Heart Rhythm.* 2005;**2**(8):830-4. [PubMed ID: 16051118]. https://doi.org/10.1016/j.hrthm.2005.05.015.

31. Khurshid S, Epstein AE, Verdino RJ, Lin D, Goldberg LR, Marchlinski FE, et al. Incidence and predictors of right ventricular pacing-induced cardiomyopathy. *Heart Rhythm.* 2014;**11**(9):1619-25. [PubMed ID: 24893122]. https://doi.org/10.1016/j.hrthm.2014.05.040.

32. Khurshid S, Liang JJ, Owens A, Lin D, Schaller R, Epstein AE, et al. Longer Paced QRS Duration is Associated With Increased Prevalence of Right Ventricular Pacing-Induced Cardiomyopathy. *J Cardiovasc Electrophysiol.* 2016;**27**(10):1174-9. [PubMed ID: 27457998]. https://doi.org/10.1111/jce.13045.

33. Tayal B, Fruelund P, Sogaard P, Riahi S, Polcwiartek C, Atwater BD, et al. Incidence of heart failure after pacemaker implantation: a nationwide Danish Registry-based follow-up study. *Eur Heart J.* 2019;**40**(44):3641-8. [PubMed ID: 31504437]. https://doi.org/10.1093/eurheartj/ehz584.

34. Oida M, Hasumi E, Kohsaku G, Kunihiro K, Oshima T, Matsubara TJ, et al. The estimated glomerular filtration rate predicts pacemaker-induced cardiomyopathy. *Sci Rep.* 2023;**13**(1):16514. [PubMed ID: 37783787]. [PubMed Central ID: PMC10545821]. https://doi.org/10.1038/s41598-023-43953-7.

35. Merchant FM, Mittal S. Pacing-Induced Cardiomyopathy. *Card Electrophysiol Clin.* 2018;**10**(3):437-45. [PubMed ID: 30172280]. https://doi.org/10.1016/j.ccep.2018.05.005.

36. Zhang H, Zhou YJ, Zeng YJ. Prognostic factors of pacing-induced cardiomyopathy. *Chin Med J (Engl).* 2020;**133**(13):1533-9. [PubMed ID: 32568868]. [PubMed Central ID: PMC7386339]. https://doi.org/10.1097/CM9.0000000000000856.