ORIGINAL ARTICLE

Braz J Cardiovasc Surg 2026;41(5):e20250335
https://doi.org/10.21470/1678-9741-2025-0335
ISSN: 1678-9741

Association Between Invasive Coronary Investigation Before Permanent Pacemaker Implantation and Mortality in a Historical Cohort with a One-Year Follow-Up: CorInPace Study

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This study was carried out at the Instituto de Ritmologia Cardíaca, Itajaí, Santa Catarina, Brazil.

ABSTRACT

Introduction: Permanent pacemaker (PPM) implantation is standard therapy for symptomatic bradyarrhythmias. In emergencies, limited assessment may lead to underdiagnosis of concomitant coronary artery disease (CAD).
Objective: To evaluate the impact of coronary cineangiography (CAG) before urgent PPM implantation on mortality.
Methods: This cohort included patients undergoing urgent PPM implantation between July 2016 and June 2021, divided into those undergoing CAG (Group A) and those not undergoing CAG (Group B). The primary endpoint was all-cause mortality; secondary endpoints included mortality/readmission, acute coronary syndrome (ACS), and hospital stay.
Results: Of 895 patients, 841 were included (Group A: 160; Group B: 681). Group A patients were younger (70.2 *vs.* 75.2 years; $P < 0.001$), more frequently male (63.1% *vs.* 51.8%; $P = 0.010$), had more CAD (36.2% *vs.* 19%; $P < 0.001$), and less frequently had estimated glomerular filtration rate < 45 ml/min/1.73 m^2 (10.2% *vs.* 22.9%; $P < 0.001$). Mortality (hazard ratio [HR] 0.96, 95% confidence interval [CI] 0.56 – 1.66; $P = 0.898$) and mortality/readmission (HR 1.05, 95% CI 0.74 – 1.50; $P = 0.776$) did not differ between groups. Multivariate analysis showed no significant impact of CAG on either outcome. ACS was more frequent in Group A (7.5% *vs.* 2.7%; $P = 0.005$), and hospital stay was shorter in Group B (6 *vs.* 9 days; $P < 0.001$).
Conclusion: In our study, CAG before PPM implantation was not associated with mortality or mortality/readmission but was associated with longer hospitalization and a higher incidence of ACS
Keywords: Pacemaker. Bradycardia. Coronary Disease. Coronary Cineangiography. Acute Coronary Syndrome.

Abbreviations, Acronyms & Symbols

ACS	= Acute coronary syndrome	GFR	= Glomerular filtration rate
AVB	= Atrioventricular block	HR	= Hazard ratio
CABG	= Coronary artery bypass grafting	IQR	= Interquartile range
CAD	= Coronary artery disease	PCI	= Percutaneous coronary intervention
CAG	= Coronary cineangiography	PPM	= Permanent pacemaker
CI	= Confidence interval	SD	= Standard deviation
CKD	= Chronic kidney disease	SND	= Sinus node dysfunction
EF	= Ejection fraction		

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Editor-in-chief: Henrique Murad
Associate Editor: Acrisio Sales Valente

How to cite: Silvestrini TL, Ronsoni RDM, Lima HN, Passuello GG, Fontoura SCP, Leiria TLL. Association Between Invasive Coronary Investigation Before Permanent Pacemaker Implantation and Mortality in a Historical Cohort with a One-Year Follow-Up: CorInPace Study. Braz J Cardiovasc Surg. 2026;41(5):e20250335. doi: 10.21470/1678-9741-2025-0335.

Article received on October 6th, 2025.
Article peer reviewed on January 5th, 2026, and February 4th, 2026.
Article accepted on February 21st, 2026.

INTRODUCTION

Permanent pacemaker (PPM) implantation is a well-established intervention for symptomatic bradyarrhythmia, whether it is due to sinus node dysfunction (SND) or atrioventricular block (AVB)[1-3]. A significant proportion of patients receive this treatment during urgent hospitalizations due to bradycardia symptoms, such as dyspnea, lightheadedness, and syncope, which lead to clinical instability.

Coronary artery disease (CAD) is a potential cause of bradycardia and may present symptoms similar to those of bradyarrhythmias or, conversely, bradycardia may mask the symptoms of CAD, complicating the differential diagnosis between these conditions. In emergency situations, this distinction becomes critical since noninvasive methods for evaluating CAD often lack high predictive value, with stratification typically performed through coronary cineangiography (CAG)[4,5]. Except in cases of acute myocardial infarction, revascularization has a minimal impact on the recovery of the rhythm[2,5-7].

The literature does not clearly state whether an invasive investigation approach with CAG during hospitalization for bradyarrhythmia requiring PPM implantation — and consequently the diagnosis and appropriate treatment of CAD — provides benefits in reducing cardiovascular outcomes in the short and medium term. Consistent data on mortality and hospital admissions in patients with significant CAD undiagnosed by CAG are scarce[8,9]. Although not routinely recommended by guidelines, CAG is occasionally performed in non-acute coronary syndrome (ACS) settings based on physician preference, reflecting real-world practice.

The primary objective of this study was to evaluate whether performing CAG prior to urgent PPM implantation for symptomatic bradycardia influences all-cause mortality during the follow-up.

METHODS

A historical cohort study was conducted at two Brazilian centers, analyzing data from medical records, patient charts, and telephone follow-ups for patients who underwent urgent PPM implantation.

Study Population

The study included patients who received PPM implantation due to symptomatic SND or AVB and sought emergency care for bradycardia at Hospital and Maternidade Marieta Konder Bornhausen, Itajaí (Santa Catarina/Brazil), and the Instituto de Cardiologia, Porto Alegre (Rio Grande do Sul, Brazil), between July 2016 and June 2021. Initially, we screened patients who underwent PPM implantation during this period via procedure-related codes from public and private healthcare systems. From this initial list, we conducted a secondary screening to identify which patients underwent CAG. Two comparison groups were defined: Group A, patients who underwent PPM implantation with CAG, and Group B, patients who underwent PPM implantation without CAG.

The decision to perform CAG was not protocol-driven and was based on the treating physician's clinical judgment, reflecting real-world practice rather than a standardized institutional strategy.

Outcomes

The primary endpoint was all-cause mortality. The secondary outcomes included the composite of all-cause mortality and readmission, CAG performance rate and prevalence of significant lesion, incidence of ACS, and length of hospital stay.

Data Collection

Patients were followed up in the artificial cardiac pacing outpatient clinic of each institution, with scheduled follow-ups during the first year at months one, three, six, and 12. The outcomes of interest were assessed on the basis of medical records. For patients without regular follow-up, three separate attempts were made to contact patients or their families by phone to collect outcome information. The occurrence of death was also verified through the death registration website https://crccidadao.com.br/crcCidadao. Hospital admissions with a diagnosis of ACS were identified per published guidelines[10].

Demographic and cardiovascular comorbidity data were collected from patient medical records. Previous CAD was defined on the basis of a history of percutaneous coronary intervention, coronary artery bypass grafting, or clinical treatment with the presence of lesions identified on CAG, regardless of the degree of obstruction. Aortic and mitral valve diseases were considered in patients with a history of previous valve surgery or moderate to severe lesions identified on echocardiogram.

Exclusion Criteria

Patients with a diagnosis of ACS per defined criteria[10] at the time of hospitalization leading to PPM implantation and those with other reversible causes of bradycardia[1] were excluded. Patients who underwent PPM implantation in an outpatient setting, those under 18 years, and patients who died within 48 hours of PPM implantation were also excluded from the final analysis to minimize reverse causality bias, as early deaths were unlikely to be influenced by CAG status.

Sample Size

We estimated that approximately 50% of patients undergoing CAG would have significant CAD, with a similar proportion in the group not undergoing CAG[4,5,11,12]. Significant CAD was defined as lesions > 70% in major coronary arteries and their first branches. Considering that diagnosed CAD patients might benefit from lifestyle changes, medication, and potentially percutaneous or surgical revascularization, we estimated a mortality risk of 5 – 10% for undiagnosed and untreated patients[13], which might decrease to 1 – 2% for diagnosed and treated patients[14]. On the basis of a preliminary evaluation of data from our first 100 patients, we observed a CAG status ratio of 1:4.5 among these patients. Taking these assumptions into account, it was determined that a sample size of 700 patients was needed to achieve a 95% significance level with 80% power. Allowing for 20% data or follow-up loss, we estimated that the final sample size should be 840 patients.

Statistical Analysis

Categorical variables are described by their frequencies and percentages. Quantitative variables are described by their mean and standard deviation or median and interquartile range on the basis of their distribution according to the Kolmogorov-Smirnov test. We used chi-square tests and Student's *t*-tests or Mann-Whitney tests to compare the frequencies or means of variables for the

primary exposure and outcome. Initially, we assessed the crude association between primary exposure and death 12 months post-PPM implantation and then adjusted for potential confounders in a bivariate manner via Cox regression. The multivariable model considered all variables that modified the effect between primary exposure and outcome by 10% or more and known variables associated with the outcome. Similarly, the effect of primary exposure was evaluated for the composite outcome of death and readmission within 12 months post-PPM implantation. All the data were analyzed via STATA/IC 15.1 software.

This study was conducted in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (or STROBE) statement to ensure comprehensive and transparent reporting of the observational study design and with the principles of the Declaration of Helsinki. This study was approved by the research ethics committee under protocol number 5.786.753, that waived the requirement for patient consent

RESULTS

During the study period, 895 patients, with a mean age of 74.2 years (± 11.3), underwent PPM implantation, and 54% of them were male. A flowchart of the study patients is shown in Figure 1. The characteristics of patients who underwent the final statistical analysis are presented in Table 1.

Patients in Group A were significantly younger than those in Group B (70.2 *vs.* 75.2 years; $P < 0.001$). The percentage of male patients was greater in Group A (63.1% *vs.* 51.08%; $P = 0.010$). Group A had nearly twice the prevalence of prior CAD (36.2% *vs.* 19%, $P < 0.001$). Group B had a greater prevalence of glomerular filtration rare (GFR) < 45 ml/min/1.73 m^2 (22.9% *vs.* 10.2%; $P < 0.001$). And the echocardiographic ejection fraction (EF) was greater in Group B (62% *vs.* 56%; $P = 0.007$).

The population of patients who survived had a lower prevalence of heart failure (14.6% *vs.* 36.0%; $P < 0.001$), a higher baseline EF (57.8% *vs.* 46.9%; $P < 0.001$), a lower prevalence of estimated GFR < 45 ml/min/1.73 m^2 (18.2% *vs.* 39.5%), and a shorter length of stay during their initial hospitalization (6 *vs.* 9 days; $P < 0.001$) (Table A – Appendices).

The outcomes related to the length of hospital stay, mortality, hospital readmission, and admission for ACS are presented in Table 2.

The length of hospital stay was shorter for Group B patients: six days (interquartile range [IQR] 1 – 5) *vs.* nine days (IQR 2 – 8) ($P < 0.001$). The time from hospital admission to PPM implantation

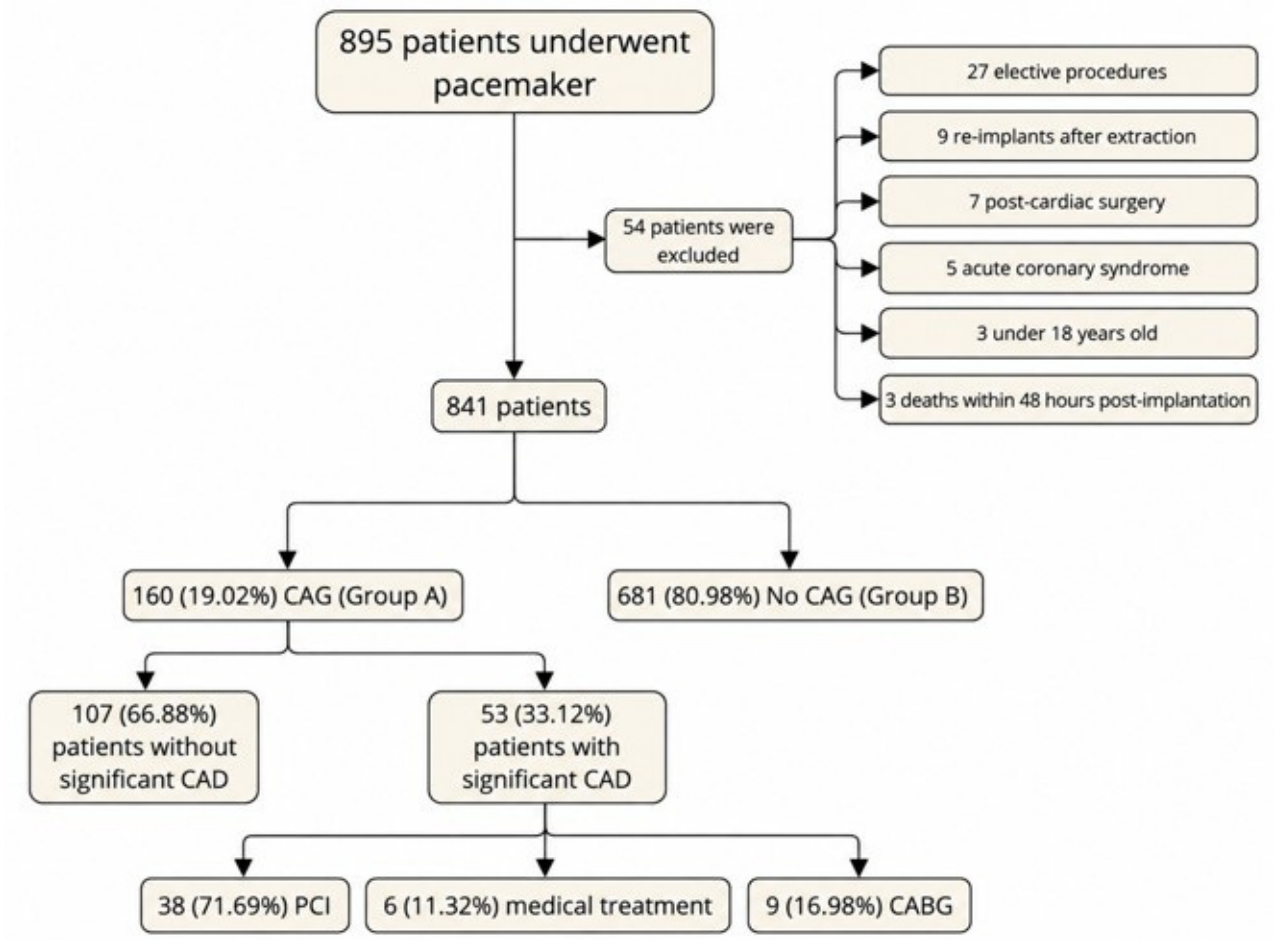

Fig. 1. *Patient flowchart. CABG=coronary artery bypass grafting; CAD=coronary artery disease; CAG=coronary cineangiography; PCI=percutaneous coronary intervention.*

Table 1. General characteristics of the sample stratified by the presence of CAG before pacemaker implantation.

General characteristics	Overall sample (n = 841)	Group A (with CAG) (n = 160)	Group B (without CAG) (n = 681)	*P*-value
Age, years; mean ± SD	74.2 ± 11.3	70.2 ± 9.8	75.2 ± 11.5	< 0.001
Male sex; total (%)	454 (54.0%)	101 (63.1%)	353 (51.8%)	0.010
Hypertension, yes; total (%), n = 828	649 (78.4%)	127 (79.9%)	522 (78%)	0.611
Heart failure, yes; total (%), n = 828	139 (16.8%)	27 (17%)	112 (16.7%)	0.942
Coronary artery disease, yes; total (%), n = 830	185 (22.3%)	58 (36.2%)	127 (19%)	< 0.001
Diabetes mellitus, yes; total (%), n = 828	251 (30.3%)	49 (30.8%)	202 (30.2%)	0.878
Aortic valvulopathy, yes; total (%), n = 816	45 (5.5%)	8 (5%)	37 (5.6%)	0.766
Mitral valvulopathy, yes; total (%), n = 816	35 (4.3%)	8 (5%)	27 (4.1%)	0.607
Estimated GFR‡, total (%), n = 812				< 0.001
≥ 45 ml/min/1.73 m^2	646 (79.6%)	141 (89.8%)	507 (77.2%)	
< 45 ml/min/1.73 m^2	166 (20.4%)	16 (10.2%)	150 (22.9%)	
Escape QRS, yes; total (%), n = 693				0.488
Narrow	210 (30.3%)	37 (27.8%)	173 (30.9%)	
Wide	483 (69.7%)	96 (72.2%)	387 (69.1%)	
Escape heart rate, median (IQR§), n = 691	39 (34/45)	40 (35/48)	38 (34/45)	0.098
Potassium, mEq/L; mean ± SD, n = 808	4.4 ± 0.6	4.5 ± 0.5	4.4 ± 0.6	0.653
Ejection fraction, %; mean ± SD, n = 432	56.6 ± 13.7	56 (44/65)	62 (49/68)	0.007
Disease; total (%)				0.786
Sick sinus syndrome	207 (25.4%)	38 (24.2%)	169 (25.6%)	
Atrioventricular block	609 (74.6%)	119 (75.8%)	490 (74.4%)	
Type of pacemaker; total (%), n = 835				0.401
Single-chamber	93 (11.2%)	14 (8.9%)	79 (11.6%)	
Dual-chamber	742 (88.8%)	143 (91.1%)	599 (88.4%)	

‡GFR (Chronic Kidney Disease Epidemiology Collaboration formula); §IQR (25th percentile/75th percentile)
CAG=coronary cineangiography; GFR=glomerular filtration rate; IQR=interquartile range; SD=standard deviation

also differed: three days (IQR 1/5) for Group B and four days (IQR 2/8) for Group A ($P < 0.001$). A significantly greater incidence of ACS was observed in Group A (7.5% *vs.* 2.7%, $P = 0.005$).

The estimated 12-month survival following PPM implantation did not differ between the groups. This percentage was 89.6% (95% confidence interval [CI] 87.0% - 91.6%) for patients in Group B and 90.0% (95% CI 84.2% - 93.7%) for patients in Group A (Figure 2).

The crude and adjusted risk analysis of the effect of CAG on mortality after PPM implantation shows that patients in Group A had a hazard ratio (HR) for all-cause mortality of 0.96 (95% CI 0.56 - 1.66, $P = 0.898$), with no significant difference compared with Group B even when adjusted for other variables in a bivariate manner (Table B – Appendices).

The multivariate analysis of the effect of CAG exposure did not impact mortality, with an HR of 0.92 (95% CI 0.52 - 1.65, $P = 0.793$), even after adjusting for other confounding variables (Table C – Appendices).

The survival curves for the composite outcomes of mortality and readmission after 12 months of PPM implantation are depicted in Figure 3. In Group B, 77.1% (95% CI 73.7% - 80.1%) of patients remained event-free, and in Group A, 76.9% (95% CI 69.5% - 82.7%) remained event-free, indicating no significant difference.

The crude and adjusted risk analyses demonstrated that patients in Group A had a total HR of 1.05 (95% CI 0.74 - 1.50, $P = 0.776$) for the composite outcome of mortality/readmission, showing no significant difference compared with Group B, even when the models were adjusted for other variables in a bivariate manner (Table D – Appendices).

Similarly, the multivariate analysis of the effect of CAG exposure on the composite outcome of mortality and readmission revealed that CAG did not affect the mortality/readmission outcome, with an HR of 0.92 (95% CI 0.63 – 1.34; $P = 0.671$), even after multivariate adjustment for confounding variables (Table E – Appendices).

DISCUSSION

In this retrospective cohort study, we evaluated the association between performing CAG prior to urgent PPM implantation and

Table 2. Outcomes in the overall sample, in patients who underwent CAG and in patients who did not undergo CAG.

Outcomes	Overall sample (n = 841)	Group A (with CAG) (n = 160)	Group B (without CAG) (n = 681)	*P*-value
Time to first hospitalization, days; median (IQR†)	6 (4/11)	9 (5/15)	6 (4/10)	< 0.001
Time from hospitalization to surgery, days; median (IQR†)	3 (1/5)	4 (2/8)	3 (1/5)	< 0.001
Deaths, yes; total (%)	87 (10.3%)	16 (10%)	71 (10.4%)	0.874
Readmission, yes; total (%)	144 (17.1%)	28 (17.5%)	116 (17.0%)	0.888
Death/readmission, yes; total (%)	196 (23.3%)	38 (23.7%)	159 (23.2%)	0.883
Acute coronary syndrome, yes; total (%), n = 778	28 (3.6%)	11 (7.5%)	17 (2.7%)	0.005

†IQR (25th percentile/75th percentile)
CAG=coronary cineangiography; IQR=interquartile range

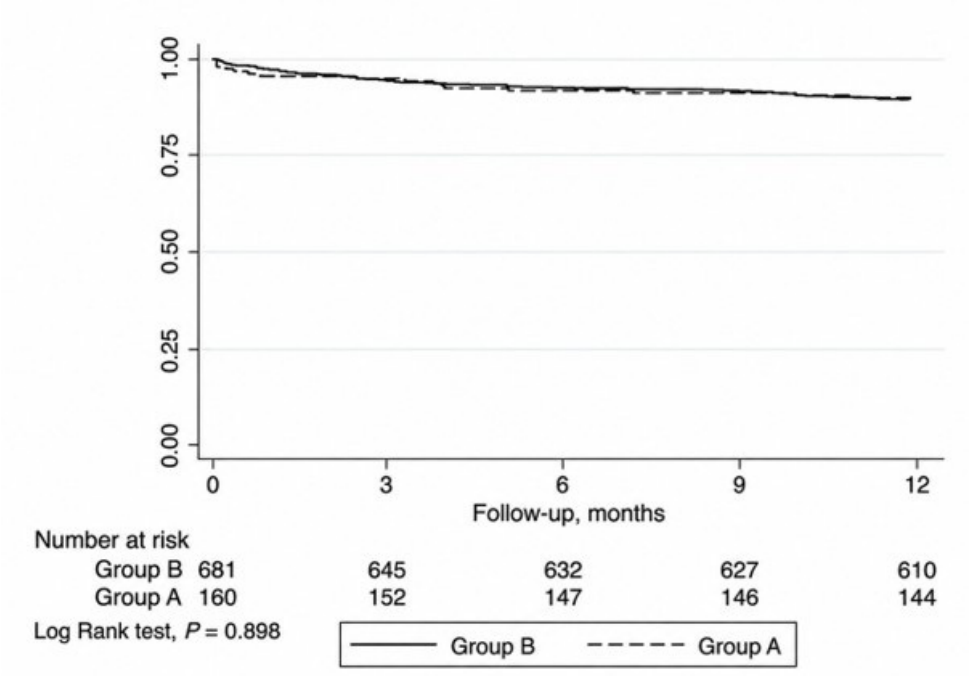

Fig. 2. *Cumulative survival to death post-permanent pacemaker implantation between patients who underwent coronary cineangiography (Group A) and those who did not undergo coronary cineangiography (Group B), assessed via Kaplan–Meier analysis.*

clinical outcomes, including mortality and hospital readmission at 12 months. Our main findings indicate that CAG — including the eventual diagnosis of CAD and its treatment when appropriate — was not associated with a reduction in mortality or the composite outcome of death and readmission. Conversely, CAG was linked to longer hospital stays, delays in pacemaker implantation, and a higher incidence of ischemic events during follow-up.

The baseline characteristics revealed a clinical pattern that may have influenced physicians' decisions to request CAG. Patients undergoing CAG were, on average, younger, had a higher prevalence of known CAD, and a lower rate of renal dysfunction. These findings suggest a selection bias favoring the invasive approach in patients perceived to have a more favorable clinical profile, longer life expectancy, and lower risk of contrast-related complications. The higher proportion of male patients in the CAG group further supports the hypothesis of selective testing, consistent with patterns previously described in the literature[15,16].

Despite this potentially more favorable profile, patients who underwent CAG did not demonstrate better clinical outcomes. The one-year mortality rate was similar between groups (approx-

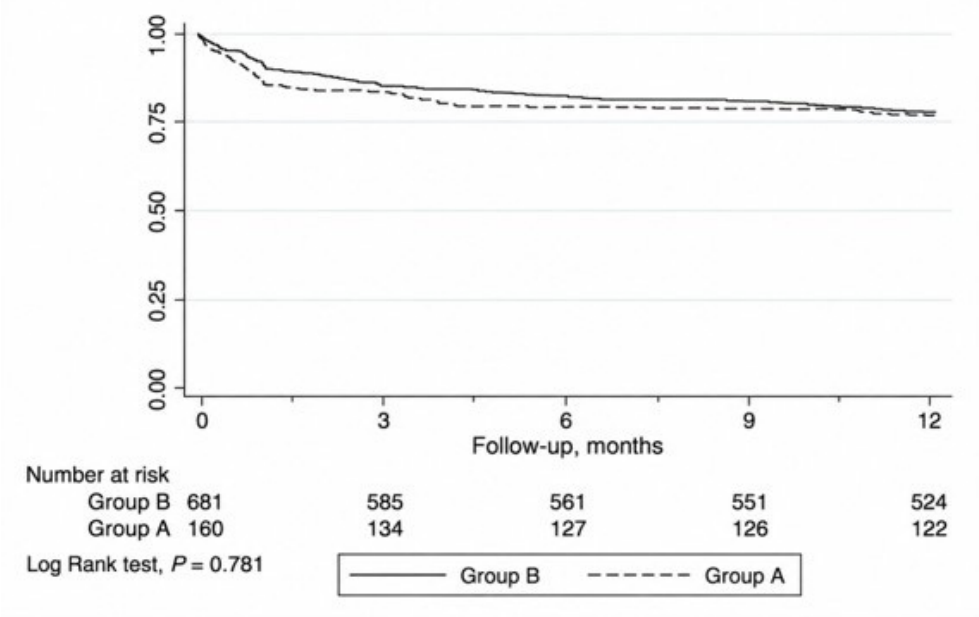

Fig. 3. *Cumulative survival to death and/or readmission post-permanent pacemaker implantation between patients who underwent coronary cineangiography (Group A) and those who did not undergo coronary cineangiography (Group B), assessed via Kaplan–Meier analysis.*

imately 10%) and remained nonsignificant after adjustment for confounders such as age, sex, heart failure, renal dysfunction, and prior CAD. The multivariate analysis yielded a HR of 0.92 (95% CI 0.52 – 1.65; $P = 0.793$), confirming the absence of association between CAG and mortality. This clinical neutrality suggests that, in patients presenting with symptomatic bradyarrhythmias without overt signs of ACS, invasive CAD investigation does not alter short- to mid-term prognosis.

From a pathophysiological perspective, our data reinforce the notion that although CAD may coexist with bradyarrhythmias — especially in older adults — it is not necessarily the direct cause of conduction disturbances. Thus, diagnosing and treating CAD in this context may not have a meaningful impact on the clinical events that led to PPM implantation. Furthermore, patients with previously diagnosed CAD are often already receiving optimized secondary prevention, which may further diminish the incremental value of CAG.

The one-year mortality rates in our cohort are consistent with those reported in other studies on PPM implantation, although none specifically assessed the role of CAG. The FOLLOWPACE study showed a 93% one-year survival rate in a Dutch cohort, slightly higher than ours, and identified prior CAD as a mortality predictor, but did not evaluate CAG[17-19].

In the REPACE study, one-year mortality was 11.7% among over 82,000 patients, reaching 68% at ten years, with CAD responsible for 31% of deaths; CAG use was not assessed[20].

Bradshaw et al.[21] found 9% one-year mortality in Australia; although revascularization during hospitalization improved five-year survival, no benefit was seen at one year, and CAG data were lacking.

The MOST trial reported 20% mortality over nearly three years, with CAD accounting for 21% of deaths[22], while Pyatt et al.[23] observed a 13.7% one-year mortality rate without assessing CAD or CAG impact.

In Brazil, Alves et al.[24] reported 8.9% one-year mortality, with approximately 11% in initial implants. Elective procedures accounted for 25% of the cohort, and no CAG data in emergency cases were provided.

Regarding the composite outcome of death and hospital readmission, our findings were consistent with the isolated mortality analysis. Event rates were similar between groups (23.7% *vs.* 23.2%; $P = 0.883$), and the adjusted HR was 0.92 (95% CI 0.63 – 1.34; $P = 0.671$), again showing no significant benefit associated with the invasive strategy. These results support the conclusion that CAG does not appear to influence the risk of relevant clinical events in this patient population.

When attempting to compare our findings with the existing literature, we faced significant challenges in identifying studies involving comparable populations and clinical scenarios. While several cohorts have reported hospital readmission and mortality rates following PPM implantation, none have specifically addressed the performance of CAG or its prognostic implications. Gillam et al.[25] described a 90-day readmission rate of 9.3% and mortality of 2.6% following dual-chamber PPM implantation, though their cohort included older patients and a higher prevalence of CAD. Patel et al.[26] reported a 30-day readmission rate of 12% in a large American cohort, with CAD and myocardial infarction cited as causes of admission in a small proportion of cases, yet no mortality or CAG data were provided. Załuska et al.[27] observed a 24.5% readmission rate over a nine-year follow-up, with CAD being a major contributor and associated with reduced survival[27]. Similarly, Alves et al.[24] found a 16.4% readmission rate, primarily driven by non-cardiovascular causes, without reporting CAD prevalence or the use of CAG.

The diagnosis and treatment of CAD are, in theory, strategies that should reduce the risk of future events when compared to undetected CAD[13,14]. Interestingly, we observed a significantly higher incidence of ACS in the CAG group (7.5% *vs.* 2.7%; $P = 0.005$). This

finding likely reflects the higher baseline atherosclerotic burden in this group, evidenced by a greater prevalence of prior CAD. It may also reflect a diagnostic bias, in which patients who underwent CAG were more likely to be further investigated and monitored for ischemic events. In addition, several studies have also shown that patients with diagnosed and treated CAD remain at high risk for recurrent coronary events, reflecting the chronic and progressive nature of the disease[28,29]. In a Danish cohort of 17,111 patients undergoing coronary angioplasty, Hansen et al.[30] (2018) reported a 50.5% hospital readmission rate in the first year, with half of these events related to new ACS.

Another relevant aspect of our findings was the impact of CAG on hospitalization time. Patients in the CAG group had significantly longer hospital stays (median 9 *vs.* 6 days; $P < 0.001$) and experienced longer delays between admission and PPM implantation (median 4 *vs.* 3 days; $P < 0.001$). These differences may be attributed to the logistical complexity of arranging CAG — particularly in public hospitals with limited resources — and to clinical decisions to defer device implantation until CAD status was clarified. While such caution may be clinically justified, our findings suggest that it did not translate into improved outcomes and may have increased the risk of complications associated with prolonged hospitalization, such as infections, delirium, or worsening comorbidities.

These observations raise important questions regarding the clinical management of patients hospitalized for symptomatic bradycardia. The lack of benefit associated with CAG, combined with longer hospital stays and increased ischemic events, highlights the need for careful patient selection. In the absence of specific guidelines, decisions regarding CAG in this setting appear to be driven by individual clinical judgment, resulting in heterogeneous practices and potentially avoidable inefficiencies.

It is also important to consider that identifying stable or subclinical CAD in patients without ischemic symptoms may not warrant immediate intervention. In cases where bradycardia is the primary clinical issue, coexisting CAD may act more as a marker of global cardiovascular risk than as a modifiable cause of arrhythmia or short-term adverse outcomes.

The CAG performance rate in our cohort was 19%, and the prevalence of significant CAD among those who underwent the procedure was 33%, lower than the initially estimated 50%. Prior studies assessing CAD prevalence in patients undergoing PPM implantation reported heterogeneous findings due to varying methodologies and definitions. Brueck et al.[11] performed CAG in patients with cardiovascular risk factors, with a 40% utilization rate and significant CAD in 71%, although CAG was conducted up to two months before or after PPM implantation. In high-risk Indian cohorts, obstructive CAD was found in 29% to 34.4%, with critical lesions (> 90% stenosis) in up to 16.25% of cases[5,31]. Lower prevalence was observed by Tayyebi et al.[4] (13.7%). Variability likely reflects differences in patient profiles, selection criteria, and angiographic thresholds. Our study defined severe CAD as > 70% stenosis in major coronary arteries or first branches, in line with standard clinical definitions.

Limitations

This study has several limitations. Its retrospective design makes it susceptible to selection bias, and the decision to perform CAG was based on unstandardized clinical criteria. Although multivariable analyses were performed, residual confounding cannot be excluded. Additionally, we lacked data on post-discharge medical therapy, such as statins or antiplatelet agents, which could influence long-term outcomes. The 12-month follow-up may also be insufficient to detect delayed benefits from revascularization or intensification of preventive therapy. Lastly, cause-specific mortality and reasons for readmission were not available, limiting a more granular analysis of the relationship between CAD and subsequent clinical events. We excluded three patients who died within 48 hours after pacemaker implantation. This exclusion aimed to avoid reverse causality, as these early deaths were related to critical baseline conditions and were unlikely to be influenced by CAG or its timing.

Despite these limitations, this study has important strengths. The sample size was robust and included a real-world population treated in the urgent care setting, enhancing the external validity of our findings. The inclusion of relevant clinical variables and the use of adjusted Cox regression models support the reliability of the conclusions. Moreover, to our knowledge, this is one of the first studies to systematically evaluate the role of CAG in patients undergoing urgent PPM implantation, addressing a gap in the existing literature.

Further prospective, ideally randomized studies are needed to define which subgroups of patients might benefit from invasive CAD evaluation before PPM implantation, considering individual factors such as cardiovascular risk, life expectancy, renal function, and presence of ischemic symptoms.

Our findings suggest that systematic invasive coronary investigation before urgent PPM implantation does not confer prognostic benefit in the absence of ACS. A more selective approach, guided by clinical presentation rather than routine testing, may reduce unnecessary delays, prolonged hospitalization, and resource utilization.

CONCLUSION

In our study, CAG before PPM implantation did not impact the outcomes of death or the composite outcome of mortality/readmission. Patients who underwent CAG experienced longer hospital stays and delays in surgery.

Data Availability

The authors declare that the data are openly available in the Harvard Dataverse at https://doi.org/10.7910/DVN/1GK1HM.

Artificial Intelligence Usage

The authors declare that no artificial intelligence tools or services were used in the preparation, writing, translation, editing, or revision of this manuscript.

Sources of Funding

The authors declare no external funding to this study.

Potential Conflict of Interest

The authors declare that there is no conflict of interest in this study.

Authors' Roles & Responsibilities

TLS Substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work; drafting the work or revising it critically for important intellectual content; agreement to be 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; final approval of the version to be published

RDMR Substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work; drafting the work or revising it critically for important intellectual content; agreement to be 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; final approval of the version to be published

HNL Substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work; drafting the work or revising it critically for important intellectual content; agreement to be 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; final approval of the version to be published

GGP Substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work; drafting the work or revising it critically for important intellectual content; agreement to be 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; final approval of the version to be published

SCPF Substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work; drafting the work or revising it critically for important intellectual content; agreement to be 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; final approval of the version to be published

TLLL Substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work; drafting the work or revising it critically for important intellectual content; agreement to be 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; final approval of the version to be published

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APPENDICES

Table A. Clinical characteristics of patients who died and those who survived.

General characteristics	Survived (n = 754, 89.7%)	Deceased (n = 87, 10.3%)	*P*-value
Age, years; mean ± SD	73.9 ± 11.4	76.9 ± 10.0	0.023
Male sex; total (%)	402 (53.2%)	52 (59.8%)	0.253
Hypertension, yes; total (%), n = 828	588 (79.2%)	61 (70.9%)	0.076
Heart failure, yes; total (%), n = 828	108 (14.6%)	31 (36.0%)	< 0.001
Coronary artery disease, yes; total (%), n = 830	160 (21.5%)	25 (28.7%)	0.127
Diabetes mellitus, yes; total (%), n = 828	222 (29.9%)	29 (33.7%)	0.468
Aortic valvulopathy, yes; total (%), n = 816	38 (5.2%)	7 (8.3%)	0.232
Mitral valvulopathy, yes; total (%), n = 816	28 (3.8%)	7 (8.3%)	0.053
Estimated GFR‡, total (%), n = 812			< 0.001
≥ 45 ml/min/1.73 m^2	594 (81.8%)	52 (60.5%)	
< 45 ml/min/1.73 m^2	132 (18.2%)	34 (39.5%)	
CAG, yes; total (%)	144 (19.1%)	16 (18.4%)	0.874
Presence of lesion, yes	47 (32.9%)	6 (35.3%)	0.841
Post-CAG management, total (%)			0.544
Clinical treatment	22 (15.3%)	1 (6.2%)	
PCI or CABG	41 (28.5%)	6 (37.5%)	
No treatment	81 (56.2%)	9 (56.2%)	
Escape QRS, yes; total (%), n = 693			0.878
Narrow	187 (30.2%)	23 (31.1%)	
Wide	432 (69.8%)	51 (69.9%)	
Escape heart rate, median (IQR§), n = 691	39 (34/45)	40 (31/48)	0.968
Potassium, mEq/L; mean ± SD, n = 808	4.4 ± 0.6	4.5 ± 0.7	0.274
Ejection fraction, %; mean ± SD, n = 432	57.8 ± 12.8	46.9 ± 16.7	< 0.001
First hospitalization time, days; median (IQR)	6 (4/10)	9 (6/18)	< 0.001
Hospitalization-to-surgery time, days; median (IQR)	3 (1/5)	3 (2/7)	0.053
Acute coronary syndrome, yes; total (%), n = 778	24 (3.5%)	4 (4.6%)	0.596
Readmission, yes; total (%), n = 691	109 (14.5%)	35 (40.2%)	< 0.001

‡GFR (Chronic Kidney Disease Epidemiology Collaboration formula); §IQR (25th percentile/75th percentile)
CABG=coronary artery bypass grafting; CAG=coronary cineangiography; GFR=glomerular filtration rate; IQR=interquartile range; PCI=percutaneous coronary intervention; SD=standard deviation

Table B. Crude and adjusted risk of the effect of coronary cineangiography on mortality after permanent pacemaker implantation, assessed via Cox regression.

Variable	Number of observations	Number of outcomes	Crude HR	95% CI	*P*-value
Coronary cineangiography	841	87			
Group B			1.00	Reference	
Group A			0.96	0.56 - 1.66	0.898
Adjusted HR					
Age	841	87	1.10	0.63 - 1.92	0.726
Sex	841	87	0.94	0.54 - 1.62	0.818
Hypertension	828	86	0.90	0.51 - 1.57	0.711
Diabetes mellitus	828	86	0.89	0.51 - 1.55	0.682
Coronary artery disease	830	87	0.88	0.51 - 1.53	0.660
Aortic valvulopathy	816	84	0.91	0.52 - 1.59	0.738
Mitral valvulopathy	816	84	0.89	0.51 - 1.56	0.688
CKD (estimated GFR < 45 ml/min/1.73 m^2)	812	86	1.12	0.65 - 1.95	0.675
Heart failure	828	86	0.89	0.51 - 1.56	0.692
Ejection fraction	432	49	0.65	0.33 - 1.32	0.235

CI=confidence interval; CKD=chronic kidney disease; GFR=glomerular filtration rate; HR=hazard ratio

Table C. Multivariate analysis of the effect of coronary cineangiography on mortality after permanent pacemaker implantation, assessed via Cox regression (n = 796).

Variable	HR	95% CI	*P*-value
Coronary cineangiography			
Group B	1.00	Reference	
Group A	0.92	0.52 - 1.65	0.793
Age	1.03	1.00 - 1.05	0.016
Sex	1.34	0.85 - 2.09	0.205
Diabetes mellitus	1.06	0.66 - 1.68	0.815
Coronary artery disease	1.14	0.69 - 1.87	0.609
Aortic valvulopathy	1.34	0.61 - 2.93	0.467
CKD (estimated GFR < 45 ml/min/1.73 m^2)	1.57	0.86 - 2.87	0.142
Heart failure	2.87	1.81 - 4.55	< 0.001

CI=confidence interval; CKD=chronic kidney disease; GFR=glomerular filtration rate; HR=hazard ratio

Table D. Crude and adjusted risk of the effect of coronary cineangiography on mortality and/or readmission after permanent pacemaker implantation, assessed via Cox Regression.

Variable	Number of observations	Number of outcomes	Crude HR	95% CI	*P*-value
Coronary cineangiography	841	196			
Group B			1.00	Reference	
Group A			1.05	0.74 - 1.50	0.776
Adjusted HR					
Age	841	196	1.10	0.77 - 1.59	0.586
Sex	841	196	1.03	0.72 - 1.47	0.880
Hypertension	828	194	1.02	0.71 - 1.45	0.925
Diabetes mellitus	828	194	1.01	0.71 - 1.45	0.935
Coronary artery disease	830	195	0.93	0.65 - 1.33	0.689
Aortic valvulopathy	816	193	1.01	0.70 - 1.44	0.960
Mitral valvulopathy	816	193	0.99	0.69 - 1.42	0.981
CKD (estimated GFR < 45 ml/min/1.73 m^2)	812	194	1.14	0.80 - 1.63	0.464
Heart failure	828	194	1.03	0.72 - 1.47	0.890
Ejection fraction	432	121	0.86	0.57 - 1.32	0.488

CI=confidence interval; CKD=chronic kidney disease; GFR=glomerular filtration rate; HR=hazard ratio

Table E. Multivariate analysis of the effect of coronary cineangiography on mortality and/or readmission after permanent pacemaker implantation, assessed via Cox regression.

Variable	HR	95% CI	*P*-value
Coronary cineangiography			
Group B	1.00	Reference	
Group A	0.92	0.63 - 1.34	0.671
Age	1.01	0.99 - 1.02	0.141
Sex	1.18	0.88 - 1.58	0.261
Diabetes mellitus	1.01	0.74 - 1.38	0.929
Coronary artery disease	1.50	1.08 - 2.07	0.014
Aortic valvulopathy	1.39	0.80 - 2.41	0.238
CKD (estimated GFR <45 ml/min/1.73 m^2)	1.72	1.15 - 2.58	0.009
Heart failure	2.14	1.56 - 2.96	< 0.001

CI=confidence interval; CKD=chronic kidney disease; GFR=glomerular filtration rate; HR=hazard ratio