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Prevalence and Predictors of Atrial High Rate Episodes in Patients with Cardiac Implantable Electronic Devices

AMIRA M. AL-AWADY RIZK, M.Sc.; MAZEN T.I. GHANEM, M.D.; HAITHAM A. BADRAN, M.D. and TAREK A. ABDEL FATTAH, M.D.

The Department of Cardiology, Faculty of Medicine, Ain Shams University

Abstract

Background: Atrial fibrillation (AF) is the most common cardiac rhythm disorder encountered in clinical practice.

It is associated with an increased risk of stroke, hospitalization, and mortality, all of which have a significant impact on healthcare economic costs.

AF is often asymptomatic and is frequently underdiagnosed. In particular, paroxysmal episodes may be missed during clinical evaluation and electrocardiogram.

Aim of Study: To investigate the prevalence and predictive factors of atrial high rate episodes in patients with cardiac implantable electronic devices and without a history of AF in order to provide a reliable basis for the clinical identification of patients with a Atrial High Rate Episodes (AHRE) in patients.

Patients and Methods: This is a retrospective, observational data collection study conducted at a single, tertiary care center in the Pacemaker Follow-up clinics at Ain Shams University Hospitals. Patients with dual-chamber CIED (dual-chamber pacemaker, ICD, CRT-P, and CRT-D), visiting the device clinic during the period of May 2022 to November 2022, were eligible for this data collection protocol.

Results: According to the result of our study, coronary artery disease, stroke/Transient ischemic attacks, left atrial volume index >34.73 ml/m^2, left ventricular ejection fraction 48%, atrial pacing percentage >53%, and ventricular pacing percentage >97% are independent factors associated with AHREs in patients with CIEDs and without a history of AF. The clinical outcomes were not evaluated due to the small number of included patients and the short duration of the study.

Conclusion: Coronary artery disease, stroke/Transient ischemic attacks, left atrial volume index >34.73 ml/m^2, left ventricular ejection fraction 48%, atrial pacing percentage >53%, and ventricular pacing percentage >97% are independent factors associated with AHREs in patients with cardiac implantable electronic devices and without a history of AF.

Key Words: *Atrial high rate episodes – Cardiac implantable electronic devices.*

Correspondence to: Dr. Amira Mo. Al-Awady Rizk,
E-Mail: amiraelawadyrizk@gmail.com

Introduction

PACEMAKERS are electrical activity-generating devices used to treat heart failure patients as well as those with slow heart rate or symptomatic heart block **[1]**. A pulse generator, which produces the electrical current needed to stimulate the heart muscle, and one or two electrodes, also known as leads, which are in charge of transferring the electrical activity produced by the pulse generator to the heart muscle, make up the majority of cardiac pacemakers **[2]**.

The most prevalent cardiac arrhythmia is atrial fibrillation (AF), and its incidence is predicted to rise in the coming years due to aging populations as well **[3]**.

According to the guidelines any arrhythmia that has the electrocardiogram (ECG) characteristics of AF and lasts sufficiently long for a 12-lead ECG to be recorded, or at least 30s on a rhythm strip, should be considered clinical AF. The presence of arrhythmia-related symptoms is not required for the definition of clinical AF, which therefore can be symptomatic or asymptomatic **[4]**.

Despite ongoing advancements in AF diagnosis, treatment, and prevention, AF morbidity and fatality rates remain high, particularly in the older population **[5]**.

Nowadays, physicians should acknowledge that a considerable proportion of AF episodes are completely asymptomatic.

When a clinical event occurs (such as a stroke, systemic thromboembolism, etc.), these episodes may or may not be discovered at that time, or they may be discovered by chance during sporadic checks. However, there is still some resistance to

the administration of appropriate prophylaxis with oral anticoagulants (OACs) in patients at risk [6,7].

Through one or more intracavitary catheters, CIEDs may now analyze, record, and perhaps treat various types of arrhythmias [6,8,9]. Additionally, the atrial lead can continuously track atrial activity and document any arrhythmic episode known as an atrial high rate episode (AHRE).

The fact that AHREs are only captured by CIEDs under continuous monitoring and that they include a variety of atrial arrhythmias, including AF, atrial flutter, and atrial tachycardia, as well as frequent transitions between regular and irregular rhythm in the same patient, is a key characteristic of these episodes. These recordings are captured as Intracardiac electrograms (EGMs), which can be stored in the memory of the devices.

Regarding the duration and atrial frequency of the episode, there is currently no agreement on the best definition of an AHRE.

The European Society of Cardiology (ESC) guidelines and the majority of studies in the literature both use the definition that stipulates a time restriction of 5 to 6 minutes and an atrial rate of 175 beats per minute [4].

These cutoffs are designed to reduce the possibility of artifact inclusion since some high atrial rate episodes may represent atrial arrhythmias of clinical importance but rather are just noise signals captured by the atrial lead [7,9].

Aim of the work:

To investigate the prevalence and predictive factors of AHREs in patients with CIEDs and without a history of AF in order to provide a reliable basis for the clinical identification of patients with a high AHRE risk.

Patients and Methods

Study population: This study included 300 patients who were undergoing follow-up of CIEDs and without a history of AF.

Study setting: The study was carried out in the Pacemaker Follow-up clinics at Ain Shams University Hospitals during the period from May 2022 to November 2022.

Inclusion criteria: Consecutive patients aged 18 years or older who underwent CIED implantation: dual chamber pacemaker, dual chamber implantable cardioverter defibrillator, cardiac resynchronization therapy-pacing, and cardiac resynchronization therapy-defibrillator, patients with no clinical evidence of a past AF attack and patients in whom the implanted pacemaker could diagnose and record atrial arrhythmia.

Exclusion criteria: Patients aged <18 years, patients with clinical evidence of AF attacks before pacemaker implantation, malfunctioning of CIEDs due to malsensing, loss of capture, or dislodgment of leads and patients who had a single chamber device (pacemaker or ICD).

Study design: Acohort study in which patients were divided into 2 groups according to the occurrence of AHREs:

- Group 1: No AHREs and group 2: AHREs

Methods:

Data collected included:

1- Patient demographics, comorbidities, and indication for pacemaker implantation: All the following data were collected: Age, gender, special habits, ethnicity/race, occupation, medical history (History of hypertension, diabetes mellitus, ischemic heart disease, ischemic stroke or transient ischemic attack, hyperlipidemia, chronic kidney disease, history of atrial fibrillation, heart failure), medications and presence or absence of symptomatic episodes of tachyarrhythmia (as palpitations or syncopal attacks).

2- 12-lead ECG before the procedure: To detect left atrial enlargement, and left ventricular hypertrophy and to exclude atrial fibrillation.

3- Echocardiography: To estimate left atrial volume, left ventricular hypertrophy, left ventricular dimensions, and left atrial diameter and to evaluate left ventricular systolic function.

4- Pacemaker programming: The pacing mode was set at DDD, with the lower rate limit at 60 b.p.m. and the upper rate limit at 130 b.p.m.

The maximum voltage in a pacemaker battery was 2.8 V. Impedance reading values ranged from 300 to 1000 Ohms.

Atrial lead was set to record signals with amplitude between 1.5 to 5 mV. The ventricular lead was set to record signals with amplitude between 5 to 25mV. The atrial and ventricular bipolar sensitivity was programmed to 0.5 and 2mV respectively.

The pacing output safety margin was calculated by multiplying the amplitude threshold by two.

The maximum tracking rate was individualized and the Mode switch function was activated.

Mode switch occurred when the atrial rate exceeded 180 b.p.m. for a given number of beats or period of time according to the default settings of the manufacturer of the pacemaker. For managing AV delay, auto AV extension algorithms (Medtronic's Search AV + with Auto PVARP) were used to decrease unnecessary right ventricular pacing and guard against retrograde conduction.

The AHREs diagnostic was programmed ON. The atrial tachycardia detection rate (ATDR) was programmed to 175 b.p.m.

The cumulative percentages of atrial and ventricular pacing were recorded. CIEDs recorded AHREs were visually inspected because some AHREs may be electrical artifacts or false positives.

Ethical considerations: The study was approved by the Cardiology Department Council and by the hospital's ethical committee and conducted in accordance with institutional guidelines on 18 th April 2022.

Statistical analysis:

Data were collected, revised, coded, and entered into the Statistical Package for Social Science (IBM SPSS).

The quantitative data were presented as mean, standard deviations, and ranges when parametric and median, and interquartile range

(IQR) when data was found non-parametric. Also, qualitative variables were presented as numbers and percentages.

The comparison between groups regarding qualitative data was done by using the Chi-square test and/or Fisher exact test when the expected count in any cell was found less than 5. The comparison between two groups regarding quantitative data and parametric distribution was done by using an Independent *t*-test while nonparametric distribution was done by using the Mann-Whitney test. The comparison between two paired groups regarding quantitative data nonparametric distribution was done by using the Wilcoxon test. Spearman correlation coefficients were used to assess the correlation between two quantitative parameters in the same group. The receiver operating characteristic curve (ROC) was used to assess the best cut-off point with its sensitivity, specificity, positive predictive value, negative predictive value, and area under the curve (AUC) of the studied marker. The confidence interval was set to 95% and the margin of error accepted was set to 5%.

So, the p-value was considered significant as the following: p-value >0.05: Non significant (N), p-value <0.05:

Significant (S) and p-value <0.01: Highly significant (HS).

Results

The current study included 300 individuals who were recruited from Ain Shams University Hospitals.

All patients were undergoing follow-up of dual chamber cardiac implantable devices in pacemaker follow-up clinics.

Demographic data and comorbidities of the studied patients: age ranged from 19 to 84 years with a mean age of 54.46±16.93 years. Males were 180 (60%) and females were 120 (40%), the occupation of studied patients was as follows: 111 (37%) were housewives, 36 (12%) were employees, 87 (29%) were retired, 15 (5%) were teachers, 12(4%) were workers, 21(7%) were students, 15(5%) were drivers and 3(1%) were interviewers, BMIranged from 21.76 to 36.36 with a mean value of 29.69±3.44 and of the study participants, 75 (25.0%) were smokers, 168 (56.0%) had hypertension, 78 (26.0%) had DM, 48 (16%) had CAD, and 15 (5%) had strokes, 33(11%) were CKD, 132 (44%) had dyslipidemia, 82 (27.3%) had HF.

Table (1): Descriptive of demographic data of the studied patients.

	No. = 300
Age (years):	
Mean ± SD	54.46±16.93
Range	19-84
Sex:	
Male	180 (60.0%)
Female	120 (40.0%)
Smoking:	
Non-smoker	225 (75.0%)
Smoker	75 (25.0%)
Occupation:	111 (37.0%)
Housewife	
Student	21 (7.0%)
Retired	87 (29.0%)
Employee	36 (12.0%)
Worker	12 (4.0%)
Driver	15 (5.0%)
Teacher	15 (5.0%)
Interviewer	3 (1.0%)

Table (2): Descriptive of comorbidities of the studied patients.

	No.	%
HTN:		
Non-hypertensive	132	44.0
Hypertensive	168	56.0
DM:		
Non-diabetic	222	74.0
Diabetic	78	26.0
CAD:		
Non-CAD	252	84.0
CAD	48	16.0
CKD:		
Non-CKD	267	89.0
CKD	33	11.0
Dyslipidemia:		
Non-dyslipidemia	168	56.0
Dyslipidemia	132	44.0
HF:		
Non-HF	218	72.7
HF	82	27.3
Stroke/TIA:		
Non-stroke	285	95.0
Stroke	15	5.0

Indication of CIED simplantation, device type, device mode, device manufacturer, duration, and medications of the studied patients:

Among the studied precipitants, 147 (49%) had complete heart block (CHB), 54 (18%) had sick sinus syndrome (SSS), 45 (15%) had mobitz II, 21 (7%) had trifasicular block, 18 (6%) had syncope, 9 (3%) had heart failure (HF), 3 (1%) had hypertrophic cardiomyopathy (HCM), 3 (1 %) had arrhythmogenic right ventricular cardiomyopathy/ dysplasia (ARVC/D).

The number of the studied patients who were on antihypertensive was 168 (56%), antiDM was 78 (26%), and lipid-lowering agents was 132 (44%), Diuretics was 111 (37%), rate control drugs was 120 (40%), rhythm control drugs was 30 (10%), anticoagulation drugs was 33 (11%), antiplatelets drugs was 69 (23%).

Electrocardiography and Echocardiographic data of thestudiedpatients: LA diameter (cm) ranged from 3.7 to 5.9cm with a mean value of 4.44±0.53, LVID (cm) ranged from 4-7.1cm with a mean value of 5.20±0.65, left atrial volume index (ml/m^2) ranged from 14.28-70.09ml/m^2 with a mean value of 32.39±9.69.

Left ventricular ejection fraction (%) ranged from 15-80% with a mean value of 49.19±11.35.

Table (3): Descriptive of Indication of CIEDs implantation, device type, device mode, device manufacturer and duration of the studied patients.

	No. = 300
Ind. of pacemaker implantation:	
CHB	147 (49.0%)
SSS	54 (18.0%)
Mobitz II	45 (15.0%)
Trifasicular block	21 (7.0%)
Syncope	18 (6.0%)
Heart failure	9 (3.0%)
HCM	3 (1.0%)
ARVC/D	3 (1.0%)
Device type:	
Dual chamber ICD	21 (7.0%)
Dual chamber pacemaker	237 (79.0%)
Biventricular pacemaker	42 (14.0%)
Device mode:	
DDDR	57 (19.0%)
DDD	243 (81.0%)
Device manufacturer:	
Medtronic	75 (25.0%)
St.Jude Medical	198 (66.0%)
Biotronik	21 (7.0%)
Boston Scientific	6 (2.0%)
Device duration (months):	
Median (IQR)	48 (24-84)
Range	1-216

Table (4): Descriptive of medications of the studied patients.

	No.	%
Anti HTN:		
No	132	44.0
Yes	168	56.0
Anti DM:		
No	222	74.0
Yes	78	26.0
Lipid-lowering agents:		
No	168	56.0
Yes	132	44.0
Diuretics:		
No	189	63.0
Yes	111	37.0
Rate control:		
No	180	60.0
Yes	120	40.0
Rhythm control:		
No	270	90.0
Yes	30	10.0
Anticoagulation:		
No	267	89.0
Yes	33	11.0
Anti-platelet:		
No	231	77.0
Yes	69	23.0

Table (5): Descriptive of symptoms of the studied patients.

	No.	%
Palpitation:		
No	243	81.0
Yes	57	19.0
Syncope:		
No	291	97.0
Yes	9	3.0
Chest pain:		
No	255	85.0
Yes	45	15.0

Table (6): Descriptive of electrocardiogram of the studied patients.

	No. = 300
ECG: LAE:	
No	258 (86.0%)
Yes	42 (14.0%)
ECG: LVH:	
No	225 (75.0%)
Yes	75 (25.0%)

LAE: Left atrial enlargement.
LVH: Left ventricular hypertrophy.

Table (7): Descriptive of echocardiographic data of the studied patients.

	No. = 300
LAVI (ml/m^2):	
Mean ± SD	32.39±9.69
Range	14.28-70.09
LAV (ml):	
Mean ± SD	47.57±18.75
Range	26.34-106.8
LVH:	
No	258 (86.0%)
Yes	42 (14.0%)
LV Diam. (cm):	
Mean ± SD	5.20±0.65
Range	4-7.1
LA Diam. (cm):	
Mean ± SD	4.44±0.53
Range	3.7-5.9
LVEF (%):	
Mean ± SD	49.19±11.35
Range	15-80

LAVI : Left atrial volume index.
LAV : Left atrial volume.
LVEF : Left ventricular ejection fraction.
LV Diam.: Left ventricular diameter.
LA Diam.: Left atrial diameter

AHREs attacks, AP% and VP%: The total number of patients who had AHREs was 93 (31 %) and patients who had not AHREs was 207 (69%).

The number of attacks ranged from 1 to 24 with the duration of an attack ranged from 2 to 86861 seconds with a median 12 (6-30).

Atrial pacing percentage ranged from 1-99% with a median 52 (30-53) while ventricular pacing percentage ranged from 1 to 100% with a median 96 (80-99).

Table (8): Descriptive of AHREs, Number of attacks and duration of attack of the studied patients.

	No. = 300
AHREs:	
No	207 (69.0%)
Yes	93 (31.0%)
No. of attacks:	
Median (IQR)	2 (1-10)
Range	1-24
Duration of attack (seconds):	
Median (IQR)	12 (6-30)
Range	2-86861

Table (9): AP% and VP% of the studied patients.

	No. = 300
AP%:	
Median (IQR)	52 (30-53)
Range	1-99
VP%:	
Median (IQR)	96 (80-99)
Range	1-100

Demographic data between the two groups: Age was highly significant between the two groups and occupation was significant between the two groups while sex and smoking were not significant between groups 1 and 2.

Height and BMI were significant while weight was highly significant between the two groups.

DM, CKD, CAD, HF, and stroke/TIA were highly significant between the two groups.

HTN and dyslipidemia were significant between the two groups (Table 12).

Indication of CIEDs implantation, device type, device mode, device manufacturer, duration, and medications of the two groups:

There was no statistically significant difference as regarding the indication of CIEDs implantation, device type, mode, manufacturer, or duration of implantation (Table 13).

The percentage of patients who are on anti-HTN, anti-DM, lipid lowering agents, and anticoagulation drugs was significantly higher in Group 2 than in Group 1.

Palpitation and chest pain were highly significant in Group 2.

Electrocardiography and Echocardiographic data of the 2 groups: LA enlargement was highly significant in group 2 and LAVI (ml/m^2), and LA diameter (cm) were significantly higher in Group 2 than in Group 1.

AP% and VP% of the two groups: The atrial pacing percentage and the ventricular pacing percentage were highly significant in the AHREs group.

The ROC analysis revealed that the best diagnostic cutoff value of the left atrial volume index was 34.73ml/m^2 and the left atrial diameter was more than 4.5cm. The best diagnostic cutoff value of LVEF was less than or equal to 48%.

The atrial pacing percentage and ventricular pacing percentage were significantly higher in the AHREs group with the best diagnostic cutoff being a value was 53% and 97% respectively.

Logistic regression analysis for predictors of AHREs:

In univariate logistic regression analysis, age >52 years, BMI >27.34Kg/m^2, hypertension, diabetes mellitus, coronary artery disease, Dyslipidemia, heart failure, chronic kidney disease, stroke/Transient ischemic attacks, left atrial volume index >34.73ml/m^2, left atrial diameter >4.5cm, left ventricular ejection fraction 48%, atrial pacing percentage> 53% and ventricular pacing percentage >97% were significant factors associated with AHREs. (Table 19).

In multivariate logistic regression analysis, coronary artery disease, stroke/Transient ischemic attacks, palpitation, syncope, left atrial volume index >34.73ml/m^2, left ventricular ejection fraction 48%, atrial pacing percentage >53%, and ventricular pacing percentage >97% were independent factors associated with AHREs. (Table 20).

Table (10): Comparison between two groups as regard Age, sex, and occupations.

	No AHREs No. = 207	AHREs No. = 93	Test value	*p*-value	Sig.
Age (years):					
Mean ± SD	52.67±17.59	58.45±14.67	–2.768•	0.006	HS
Range	19-84	19-80			
Sex:					
Male	128 (61.8%)	52 (55.9%)	0.938*	0.333	NS
Female	79 (38.2%)	41 (44.1%)			
Smoking:					
Non-smoker	156 (75.4%)	69 (74.2%)	0.047 *	0.829	NS
Smoker	51 (24.6%)	24 (25.8%)			
Occupation:					
Housewife	66 (31.9%)	45 (48.4%)	17.703*	0.013	S
Student	18 (8.7%)	3 (3.2%)			
Retired	57 (27.5%)	30 (32.3%)			
Employee	27 (13.0%)	9 (9.7%)			
Worker	9 (4.3%)	3 (3.2%)			
Driver	15 (7.2%)	0 (0.0%)			
Teacher	12 (5.8%)	3 (3.2%)			
Interviewer	3 (1.4%)	0 (0.0%)			

p-value >0.05: Non significant (NS). *: Chi-square test.
p-value <0.05: Significant (S). •: Independent *t*-test.
p-value <0.01: Highly significant (HS).

Table (11): Comparison between two groups regarding weight, height, and BMI.

	No AHREs No. = 207	AHREs No. = 93	Test value•	*p*-value	Sig.
Weight (kg):					
Mean ± SD	80.35±11.57	84.65±9.40	–3.146	0.002	HS
Range	55-100	60-100			
Height (cm):					
Mean ± SD	165.28±5.16	166.71±4.80	–2.273	0.024	S
Range	155-175	158-175			
BMI (Kg/m^2:					
Mean ± SD	29.36±3.63	30.42±2.84	–2.490	0.013	S
Range	21.76-35.69	23.44-36.36			

p-value >0.05: Non significant (NS).
p-value <0.05: Significant (S).
p-value <0.01: Highly significant (HS).
•: Independent *t*-test.

Table (12): Comparison between two groups regarding comorbidities.

	No AHREs		AHREs		Test value•	*p*-value	Sig.
	No.	%	No.	%			
HTN:							
No hypertension	99	47.8	33	35.5	3.967	0.046	S
Hypertension	108	52.2	60	64.5			
DM:							
No DM	165	79.7	57	61.3	11.316	0.001	HS
DM	42	20.3	36	38.7			
CAD:							
No CAD	186	89.9	66	71.0	17.032	0.000	HS
CAD	21	10.1	27	29.0			
CKD:							
No CKD	195	94.2	72	77.4	18.464	0.000	HS
CKD	12	5.8	21	22.6			
Dyslipidemia:							
Nodyslipidemia	126	60.9	42	45.2	6.426	0.011	S
Dyslipidemia	81	39.1	51	54.8			
HF:							
No HF	164	79.2	54	58.1	14.469	0.000	HS
HF	43	20.8	39	41.9			
Stroke/TIA:							
No stroke	204	98.6	81	87.1	17.723	0.000	HS
Stroke	3	1.4	12	12.9			

p-value >0.05: Non significant (NS).
p-value <0.05: Significant (S).
p-value <0.01: Highly significant (HS).
*: Chi-square test.

Table (13): Comparison between two groups as regard indications of CIEDs implantation, device type, mode, manufacturer and duration.

	No AHREs	AHREs	Test value	p-value	Sig.
	No. = 207	No. = 93			
Ind. of pacemaker implantation:					
CHB	105 (50.7%)	42 (45.2%)	10.523*	0.161	NS
Mobitz II	27 (13.0%)	18 (19.4%)			
SSS	39 (18.8%)	15 (16.1%)			
Trifasicular	15 (7.2%)	6 (6.5%)			
Block Syncope	12 (5.8%)	6 (6.5%)			
Heart failure	3 (1.4%)	6 (6.5%)			
HCM	3 (1.4%)	0 (0.0%)			
ARVC/D	3 (1.4%)	0 (0.0%)			
Device type:					
Dual chamber ICD	18 (8.7%)	3 (3.2%)	3.225*	0.199	NS
Dual chamberpacemaker	162 (78.3%)	75 (80.6%)			
Biventricular pacemaker	27 (13.0%)	15 (16.1%)			
Device mode:					
DDDR	42 (20.3%)	15 (16.1%)	0.722*	0.396	NS
DDD	165 (79.7%)	78 (83.9%)			
Device manufacturer:					
Medtronic	51 (24.6%)	24 (25.8%)	2.851 *	0.415	NS
St.Jude Medical	135 (65.2%)	63 (67.7%)			
Biotronik	15 (7.2%)	6 (6.5%)			
Boston Scientific	6 (2.9%)	0 (0.0%)			
Device duration (months):					
Median (IQR)	48 (24-84)	48 (12-72)	–0.955 ‡	0.340	NS
Range	1-192	1-216			

p-value >0.05: Non significant (NS). *: Chi-square test.
p-value <0.05: Significant (S). ‡: Mann Whitney test.
p-value <0.01: Highly significant (HS).

Table (14): Comparison between two groups as regard medications.

	No AHREs		AHREs		Test value*	p-value	Sig.
	No.	%	No.	%			
Anti HTN:							
No	99	47.8	33	35.5	3.967	0.046	S
Yes	108	52.2	60	64.5			
Anti DM:							
No	165	79.7	57	61.3	11.316	0.001	HS
Yes	42	20.3	36	38.7			
Lipid lowering agents:							
No	126	60.9	42	45.2	6.426	0.011	S
Yes	81	39.1	51	54.8			
Diuretics:							
No	138	66.7	51	54.8	3.851	0.050	NS
Yes	69	33.3	42	45.2			
Rate control:							
No	126	60.9	54	58.1	0.210	0.646	NS
Yes	81	39.1	39	41.9			
Rhythm control:							
No	186	89.9	84	90.3	0.016	0.901	NS
Yes	21	10.1	9	9.7			
Anticoagulation:							
No	195	94.2	72	77.4	18.464	0.000	HS
Yes	12	5.8	21	22.6			
Anti-platelet:							
No	156	75.4	75	80.6	1.011	0.315	NS
Yes	51	24.6	18	19.4			

p-value >0.05: Non significant (NS). *p*-value <0.01: Highly significant (HS).
p-value <0.05: Significant (S). *: Chi-square test.

Table (15): Comparison between two groups as regard symptoms.

	No AHREs		AHREs		Test value*	*p*-value	Sig.
	No.	%	No.	%			
Palpitation:							
No	183	88.4	60	64.5	23.797	0.000	HS
Yes	24	11.6	33	35.5			
Syncope:							
No	204	98.6	87	93.5	5.518	0.019	S
Yes	3	1.4	6	6.5			
Chest pain:							
No	189	91.3	66	71.0	20.815	0.000	HS
Yes	18	8.7	27	29.0			

p-value >0.05: Non significant (NS). *p*-value <0.01: Highly significant (HS).
p-value <0.05: Significant (S). *: Chi-square test.

Table (16): Comparison between the two groups as regard electrocardiogram.

	No AHREs	AHREs	Test value	*p*-value	Sig.
	No. = 207	No. = 93			
ECG: LAE					
No	201 (97.1%)	57 (61.3%)	68.350*	0.000	HS
Yes	6 (2.9%)	36 (38.7%)			
ECG: LVH:					
No	156 (75.4%)	69 (74.2%)	0.047*	0.829	NS
Yes	51 (24.6%)	24 (25.8%)			

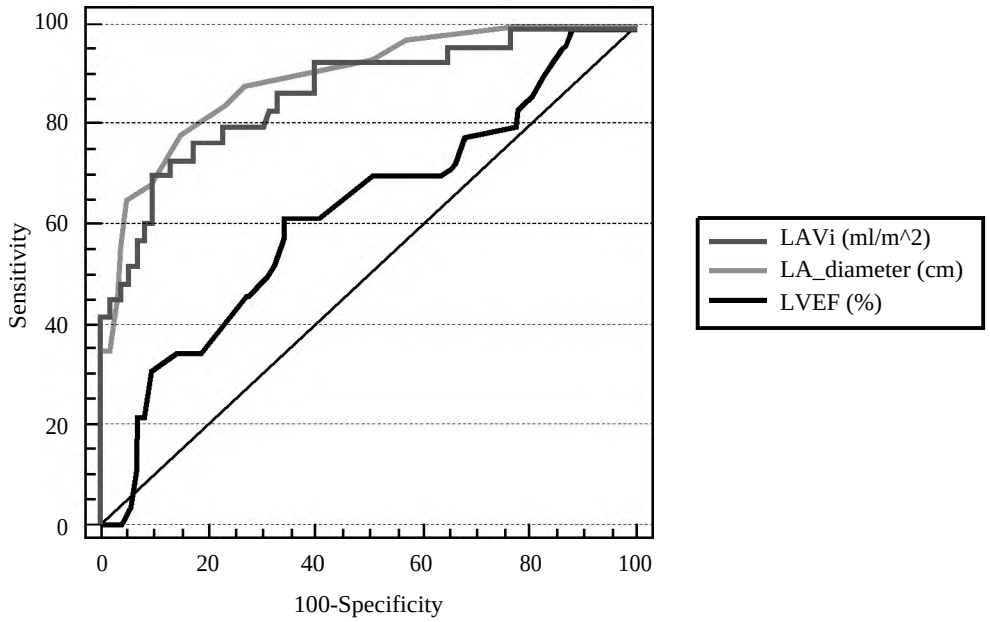
p-value >0.05: Non significant (NS). *: Chi-square test.
p-value <0.05: Significant (S). •: Independent *t*-test.
p-value <0.01: Highly significant (HS).

Table (17): Comparison between the two groups regarding echocardiographic data.

	No AHREs	AHREs	Test value	*p*-value	Sig.
	No. = 207	No. = 93			
LAVI(ml/m^2):					
Mean ± SD	20.34±4.71	41.42±11.67	–13.833•	0.000	HS
Range	14.28-42.96	16.18-70.09			
LAV (ml):					
Mean ± SD	39.20±9.13	66.20±21.14	–15.468•	0.000	HS
Range	26.34-73.12	33.28-106.8			
LVH:					
No	180 (87.0%)	78 (83.9%)	0.507 *	0.476	NS
Yes	27 (13.0%)	15 (16.1%)			
LV Diam. (cm):					
Mean ± SD	5.23±0.65	5.15±0.64	1.052•	0.294	NS
Range	4-7.1	4.1-6.3			
LA Diam. (cm):					
Mean ± SD	4.20±0.31	4.97±0.54	–15.758•	0.000	HS
Range	3.7-5.2	4-5.9			
LVEF (%):					
Mean ± SD	50.63±10.86	45.97±11.80	3.349•	0.001	HS
Range	15-80	21-76			

p-value >0.05: Non significant (NS). *: Chi-square test.
p-value <0.05: Significant (S). ‡: Mann Whitney test.
p-value <0.01: Highly significant (HS).

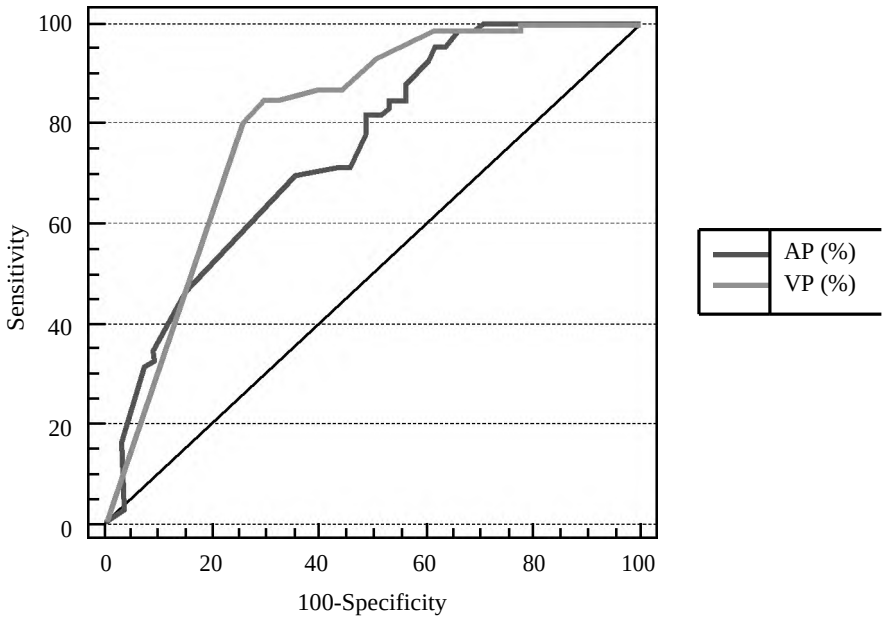
ROC curve of echo as a predictor of AHREs:



Parameter	AUC	Cut of Point	Sensitivity	Specificity	PPV	NPV
LAVI (ml/m^2)	0.863	>34.73	69.89	90.82	77.4	87.0
LA diameter (cm)	0.894	>4.5	77.42	85.51	70.6	89.4
LVEF (%)	0.623	48	61.29	65.70	44.5	79.1

Fig. (1): Receiver-operating characteristic (ROC) curve for ECHO data as predictors of AHRE.

ROC curve of AP (%) and VP (%) as a predictor of AHREs:



Parameter	AUC	Cut of Point	Sensitivity	Specificity	PPV	NPV
AP (%)	0.751	>53	51.61	84.54	60.0	79.5
VP (%)	0.810	>97	84.95	70.53	56.4	91.2

Fig. (2): Receiver-operating characteristic (ROC) curve for AP (%) and VP(%) as predictors of AHRE.

Table (19): Univariate logistic regression analysis for significant factors associated with AHREs.

	Univariate			
	p-value	Odds ratio (OR)	95% C.I. for OR	
			Lower	Upper
Age >52	0.003	2.241	1.326	3.785
BMI >27.34	0.000	6.787	2.823	16.321
HTN	0.047	1.667	1.006	2.761
DM	0.001	2.481	1.450	4.247
CAD	0.000	3.623	1.919	6.842
CKD	0.000	4.740	2.219	10.125
Dyslipidemia	0.012	1.889	1.152	3.098
HF	0.000	2.755	1.619	4.686
Stroke/TIA	0.000	10.074	2.770	36.636
Palpitation	0.000	4.194	2.299	7.651
Syncope	0.032	4.690	1.147	19.179
Chest pain	0.000	4.295	2.222	8.302
ECG: LAE	0.000	21.158	8.492	52.718
LAVI >34.73	0.000	22.970	12.025	43.878
LA Diameter >4.5	0.000	20.229	10.868	37.653
LVEF 48	0.000	3.033	1.828	5.033
AP% >53	0.000	4.883	2.793	8.535
VP% >97	0.000	13.506	7.107	25.668

Table (20): Multivariate logistic regression analysis for independent factors of AHREs.

	Multivariate			
	p-value	Odds ratio (OR)	95% C.I. for OR	
			Lower	Upper
Age >52	0.742	1.335	0.239	7.462
BMI >27.34	0.321	20.246	0.054	7661.282
HTN	0.547	0.633	0.143	2.809
DM	0.341	2.578	0.366	18.130
CAD	0.044	7.291	1.056	50.322
CKD	0.132	4.395	0.640	30.199
Dyslipidemia	0.278	0.381	0.066	2.182
HF	0.514	0.603	0.132	2.756
Stroke/TIA	0.001	3726.175	26.498	523986.06
Palpitation	0.015	7.248	1.475	35.609
Syncope	0.010	0.003	0.000	0.247
Chest pain	0.657	0.617	0.073	5.188
ECG: LAE	0.655	0.619	0.075	5.080
LAVI >34.73	0.010	31.447	2.263	436.976
LA Diameter >4.5	–	–	–	–
LVEF 48	0.012	5.993	1.477	24.316
AP% >53	0.009	5.529	1.532	19.953
VP% >97	0.000	55.072	11.095	273.366

Discussion

In our study, we investigated both the prevalence and predictors of AHREs in patients with CIEDs and without a history of atrial fibrillation (AF). Importantly, studies including patients with the clinical diagnosis AF, which per se have a higher frequency of atrial arrhythmias, found AHRE in 40-70% **[9]**.

Studies excluding patients with known AF have found AHRE in 10-30% of patients **[10]**.

In our study, patients with a previous history of AF were excluded. AHRE was detected in 93/300 patients.

The AHRE incidence was 31 %, which is similar to the incidence reported in the relevant literature **[11]**.

Healey et al., examined 445 patients (25.9% of which had pre-existing AF) who had dual-chamber pacemakers placed for multiple indications including sinoatrial nodal dysfunction (SND). They found that at a mean of 4.3-year follow-up, 55.3% of patients had device-detected AF **[12]**.

Preliminary results from the ASSERT-II trial (The Asymptomatic Atrial Fibrillation and Stroke Evaluation in Pacemaker Patients and the Atrial Fibrillation Reduction Atrial Pacing Trial) **[12]** suggested that among individuals aged >65 years, with risk factors for stroke and evidence of left atrial enlargement, the incidence of AHREs was 34.4% per year. Preliminary data indicate that the incidence of AHREs in the REVEAL-AF study **[13]** was 27% at 1 year and 29% at 18 months. In PREDATE-AF **[14]**, the incidence of AHREs at 15 months was somewhat lower at 22%. The higher incidence of AHREs in ASSERT-II might be related to the inclusion of increased left atrial dimension as one of the entry criteria in that study.

In this study, significant univariate predictors of AHREs development were age >52 years, BMI >27.34Kg/m^2, HTN, diabetes mellitus, CAD, dyslipidemia, HF, CKD, stroke/transient ischemic attack, left atrial volume index >34.73ml/m^2, left atrial diameter >4.5cm, LVEF 48%, atrial pacing percentage > 53% and ventricular pacing percentage >97% while the independent predictors for development of AHRE were CAD (OR 3.474.5, CI 1.919-6.842, p=0.000), Stroke/ TIA (OR 99.023, CI 1.372-7148, p=0.035), LAVI >34.73ml/m^2 (OR 456.420, CI 59.955-3474.5, p=0.000), LVEF 48% (OR 25.613, CI 5.898-111.233, p=0.000), AP >53% (OR 169.186, CI 24.372-1174.452, p= 0.000) and VP >97% (OR 13.506, CI 7.107-25.668, p= 0.000).

Similar to our study, previous population-based studies on outpatients found that increased LA volume and left atrial enlargement were associated with an increased rate of subclinical atrial fibrillation (SCAF) **[15,16]**. Furthermore, older age, HTN and history of HF were found to be independent predictors of SCAF **[12]**.

Proietti et al. **[17]**, calculated pooled estimates for several characteristics comparing patients with SCAF vs. patients without SCAF. The occurrence of SCAF was significantly associated with older age, and clinical history of atrial fibrillation, hypertension, heart failure, and history of stroke/ transient ischemic attack; however, male sex, body mass index, diabetes, and CAD were not associated with SCAF. They did not observe any significant association between SCAF and pharmacological treatments.

Patient characteristics such as increasing age, HTN, diabetes, or vascular disease are already known to be independent predictors of AF. Association of the known risk factors of AF with the occurrence of AHREs may improve the predictability and detection of AHREs in patients with CIEDs; and also, further assist in predicting the increased risk of stroke and systemic embolism through intense monitoring. Almost three decades ago, the landmark Framingham Heart Study has well-established HTN, aging, congestive heart failure, CAD, diabetes mellitus as independent risk factors for AF **[18]**.

Multiple studies have, since then, consistently reported HTN and diabetes as significant independent predictors of AF, adjusting for age and other predisposing conditions **[19]**. Similarly, in our study too, we found significant association of HTN, diabetes, CAD, and stroke/TIA with AHRE occurrence.

In the study of Bukari et al. **[20]**, the strongest predictor was co-existing diabetes mellitus followed by LA size.

AP was also significantly associated with AF, atrial flutter (AFL) and atrial tachycardia (AT). When arrhythmias were evaluated individually, AF was associated with HTN and LA size. AT was significantly associated with HTN, CKD, and atrial pacing percentage. New-onset AF was associated with LA size which was concordant with our study.

Aging has been a known risk factor for new-onset AF Li et al. **[21]**. However, arrhythmia is observed frequently in older patients, it is also reported to be common in young people and those without any comorbidity Wasmer et al. **[22]**.

for the association of age with AHRE occurrence has been conflicting. Some studies have shown that the occurrence of AHRE increases with age, **[12,23]** while others have failed to show any significant relation between them **[24]**.

In our analysis too, age appears to have a highly significant association with AHREs occurrence (p=0.006).

In univariate logistic regression analysis, also an age of more than 52 years was a significant factor associated with AHREs.

Gonzalez et al. **[25]** reported that previous HF was an AHRE predictor, and Wilton et al. **[26]**

revealed that in trials on re synchronization in ambulatory HF, AF/AT after implantation of CRT/D was detected in nearly half of the patients after randomized grouping.

In our study, there was a significant association between HF and AHRE occurrence. The ROC analysis revealed that the best diagnostic cutoff value of LVEF was less than or equal to 48% (AUC =58.023, sensitivity=61.29, specificity=65.70).

Both sinoatrial nodal dysfunction (SND) and AF are associated with atrial remodeling.

The common pathological change is atrial fibrosis, which results in an extensive low-voltage area and slow conduction velocity in the atrium. A recent study revealed that Paired-like homeodomain 2, the first common AF gene locus, is not only involved in the development of the pulmonary vein but also related to the development of sinoatrial node and the asymmetry of the right and left atrium. Among patients with SND, 40-70% have atrial arrhythmias, such as AF. The results of the ASSERT revealed that SND and the resting heart rate decrease were AHRE predictors which were discordant with our study in which There was a non-significant difference in the indication of CIEDs implantation between the two groups (p=0.161). This may be due to several reasons; our study excluded patients with previously diagnosed AF, different sample sizes of studied patients between this study and our study, and ASSERT was a multicenter trial, unlike our study which was a single-center. Also, the percentage of patients with SND in our study was less than ASSERT trial **[12]**.

In this study, left atrial enlargement was significantly associated with AHREs (p=0.000) which suggests that left atrial enlargement is closely related to AHRE occurrence. The ROC analysis revealed that the best diagnostic cut off value of the left atrial volume index was 34.73ml/m^2 (AUC=0.863, sensitivity=69.89, specificity=90.82) and of left atrial diameter was more than 4.5cm (AUC=0.894, sensitivity=77.42, specificity=85.51) which is similar to a previous study that concluded that left atrial enlargement was not only closely related to the occurrence and development of AF but also a predictor of AF recurrence after radiofrequency ablation. Atrial enlargement is accompanied by different degrees of atrial fibrosis, and that atrial fibrosis may be an important characteristic of persistent AF. Kim et al. **[27]** revealed that left atrium enlargement (>41mm) was associated with AHRE occurrence (OR=1.96; 95% CI, 1.00-3.85; and p=0.050). A number of clinical diseases (e.g., HTN, CAD, HF, cardiomyopathy, obesity, and diabetes) can induce atrial fibrosis; this suggests that in AF treatment, attention should also be paid to comprehensively treat patient complications. This is consistent with the guidelines' treatment path **[10]**.

In Kim et al., significant univariate predictors of AF development were prior HF, and LA volume index 38.5mL/m^2

According to a multivariate Cox regression model, the independent predictors for AF development were prior HF (hazard ratio [HR] 2.40; 95% confidence interval [CI] 1.50-3.85; p<0.001), and LA volume index 38.5mL/m^2 (HR 2.01; 95% CI 1.23-3.30; p=0.005).

While the association between LA dimension and AF is best documented in the general population, Healey et al., early study was important in demonstrating an association between LA enlargement and pacemaker-detected AF. Also showed LA size as an OR 1.18 (1.07-1.29, p=0.001) for new-onset AF risk in a mixed population that received pacemaker for either SND or high grade AV block **[12]**.

Atrial pacing increases the occurrence of AHREs; Adelstein and Saba **[28]** revealed that, after CRT implantation, the risk of AF increased 2 folds in patients with atrial pacing than in patients with atrial sensing. Fontenla et al. **[29]** reported that atrial rate-responsive pacing increased the incidence of persistent AF/AT in patients with ICD implants (OR=3.58; 95% CI, 1.82-7.03; and p< 0.001). In the present study, the AP% was significantly higher in the AHREs group. The ROC curve analysis revealed that the best AP% diagnostic cut-off value was 53% (AUC=0.751; sensitivity= 51.61 %; specificity=84.54%; and p<0.001).

Also, these findings are concordant with the results of a small study that showed an increased incidence of AF with atrial pacing in patients undergoing cardiac resynchronization therapy **[28]**.

Unfortunately, the nature of our present study did not allow for a true mechanistic inquiry.

We speculate that the mechanism for our findings may be related to the nonphysiological propagation and conduction time of atrial depolarization during atrial pacing. The subsequent delay in left atrial contraction can diminish left ventricular filling, leading to higher atrial pressures and thus

increasing the risk of AF. In addition, the electrical dispersion caused by atrial appendage pacing may act as a trigger for the formation of re-entrant pathways, particularly in patients with already diseased atria.

The mechanism for the increased incidence of AF with ventricular pacing remains unclear. Right ventricular pacing leads to left ventricular remodelling, increases mitral regurgitation, and modestly reduces ejection fraction. Furthermore, changing the relationship between atrial and ventricular timing, as can occur with ventricular pacing, has been shown to increase atrial pressure and cause stretch-related changes, which may increase the incidence of AF.

Concordant with our study, Aizawa et al. concluded that an increased percentage of ventricular pacing has been associated with an increased risk of developing AF. However, even in patients with dual-chamber pacemakers, where atrioventricular synchrony is preserved, an increased percentage of ventricular pacing has been associated with a higher risk of developing AF. The most likely explanation is that ventricular pacing causes paradoxical septal motion, which alters interventricular synchrony, lowers ejection fraction, and increases filling pressures in the heart chambers. This leads to electric remodelling of the left atrium **[18]**.

Limitations:

This study has several limitations. First, this was a single-center, retrospective, observational study with a relatively small number of patients, and all patients were Egyptian. So the results may not be generalizable to other populations. Second, atrial undersensing can occur during AHREs, which can lead to either a failure to detect an AHRE or the truncation of a single AHRE into multiple shorter episodes.

Third, since all the patients found to have AHREs were only detected on device interrogation and not on electrocardiogram recordings, it can be difficult to differentiate with certainty AF from other forms of atrial tachyarrhythmia.

Conclusion:

Coronary artery disease, stroke/Transient ischemic attacks, left atrial volume index >34.73 ml/m^2, left ventricular ejection fraction 48%, atrial pacing percentage >53%, and ventricular pacing percentage >97% are independent factors associated with AHREs in patients with cardiac implantable electronic devices and without a history of AF.

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دراسة الحدوث والتنبؤات للنوبات عالية المعدل الأذينى فى المرضى المعتمدين على زراعة الأجهزة الإلكترونية لعلاج أمراض القلب والأوعية الدموية

\الخلفية : الرجفان الأذينى (AF) هو اضطراب ضربات القلب الأكثر شيوعاً فى الممارسة السريرية. يرتبط بزيادة خطر الإصابة بالسكتة الدماغية والاستشفاء والوفيات، وكلها لها تأثير كبير على تكاليف الرعاية الصحية الأقتصادية. غالباً ما يكون الرجفان الأذينى بدون أعراض وغالباً ما لا يتم تشخيصه بشكل كافٍ. على وجه الخصوص، قد تفوت نوبات الانتيابية أثناء التقييم السريرى وتخطيط القلب الكهربائى.

\الهدف : للتحقيق فى حدوث والعوامل التنبؤية لـ AHREs فى المرضى الذين يعانون من CIEDs وبدون تاريخ من AF من أجل توفير أساس موثوق به لتحديد المرضى الذين يعانون من مخاطر عالية من AHRE .

\المرضى و\الطرق : هذه دراسة لجمع البيانات بأثر رجعى، أجريت فى مركز رعاية واحد من الدرجة الثالثة فى عيادات متابعة منظم ضربات القلب فى مستشفيات جامعة عين شمس. المرضى الذين يعانون من CIED ثنائى الغرفة (منظم ضربات القلب ثنائى الغرفة، ICD، CRT-P وCRT-D)، الذين يزورون عيادة الجهاز خلال الفترة من مايو ٢٠٢٢ إلى نوفمبر ٢٠٢٢، كانوا مؤهلين لبروتوكول جمع البيانات هذا .

\النتائج : وفقاً لنتائج دراستنا، خلصنا إلى أن مرض الشريان التاجى، والسكتة الدماغية / النوبات الإقفارية العابرة. ومؤشر حجم الأذين الأيسر > ٣٤.٧٣ مل / م ٢٨، وكسر طرد البطين الأيسر ٤٨٪، ونسبة سرعة الأذين >٥٣٪، ونسبة سرعة البطين هى عوامل مستقلة مرتبطة بـ AHREs فى المرضى الذين لديهم أجهزة إلكترونية قابلة للزرع فى القلب وليس لديهم تاريخ من الرجفان الأذينى، لكن لسوء الحظ، لا يمكننا تقييم النتائج السريرية لـ AHREs فى هؤلاء المرضى بسبب قصر مدة الدراسة الحالية والعدد الصغير نسبياً من المرضى الذين تم تضمينهم فى دراستنا .

\الخلاصة : مرض الشريان التاجى، والسكتة الدماغية / النوبات الإقفارية العابرة، ومؤشر حجم الأذين الأيسر > ٣٤.٧٣ مل / م ٢٨، وكسر طرد البطين الأيسر ٤٨٪، ونسبة سرعة الأذين > ٥٣٪، ونسبة سرعة البطين > ٩٧٪ عوامل مستقلة مرتبطة مع AHREs فى المرضى الذين يعانون من أجهزة إلكترونية مزروعة بالقلب وبدون تاريخ من الرجفان الأذينى.