International Journal of Cardiology Sciences

ISSN Print: 2664-9020
ISSN Online: 2664-9039
Impact Factor: RJIF 5.63
IJCS 2025; 7(2): 97-104
www.cardiologyjournals.net
Received: 12-07-2025
Accepted: 17-08-2025

Nourhan Hossameldein Ahmed
Department of Cardiovascular Medicine, Faculty of Medicine, Tanta University, Tanta, Egypt

Seham Fahmy Badr
Department of Cardiovascular Medicine, Faculty of Medicine, Tanta University, Tanta, Egypt

Mai Mohammed Salama
Department of Cardiovascular Medicine, Faculty of Medicine, Tanta University, Tanta, Egypt

Sahr Abdallah Elshedody
Department of Cardiovascular Medicine, Faculty of Medicine, Tanta University, Tanta, Egypt

Mohamed Khalfallah
Department of Cardiovascular Medicine, Faculty of Medicine, Tanta University, Tanta, Egypt

Corresponding Author:
Nourhan Hossameldein Ahmed
Department of Cardiovascular Medicine, Faculty of Medicine, Tanta University, Tanta, Egypt

Registry of patients with ventricular arrhythmia in Tanta university hospitals incidence, risk factors and outcomes

Nourhan Hossameldein Ahmed, Seham Fahmy Badr, Mai Mohammed Salama, Sahr Abdallah Elshedody and Mohamed Khalfallah

DOI: https://www.doi.org/10.33545/26649020.2025.v7.i2b.124

Abstract
Background: Ventricular arrhythmias (VAs) encompass a wide spectrum of rhythm disorders ranging from premature ventricular contractions (PVCs) to sustained ventricular tachycardia (VT) and life-threatening ventricular fibrillation (VF). They are frequently associated with structural heart disease (SHD) and remain a major cause of morbidity and mortality. Despite their clinical importance, data on incidence and outcomes from real-world registries in developing countries are limited.
Objectives: This study aimed to assess the incidence, risk factors, and outcomes of patients presenting with VAs at Tanta University Hospitals.
Methods: This prospective observational registry included 400 consecutive patients presented with VAs. Clinical history, comorbidities, laboratory results, electrocardiogram (ECG), and echocardiographic data were systematically collected. Outcomes at 3 months included cardiac mortality, recurrence of VA, and heart-failure (HF) hospitalization.
Results: The incidence of VAs among patients was 14% over two years. The cohort had a mean age of 53.0±15 years and 59% were male. During follow-up, cardiac mortality occurred in 28 patients (7%), VA recurrence in 62 patients (15.5%), and HF hospitalization in 47 patients (11.7%). In multivariate analysis, cardiac mortality was independently predicted by monomorphic VT (P=0.004; OR 4.16, 95% CI 1.57-11.04) and VF (P=0.003; OR 5.13, 95% CI 1.72-15.25). VA recurrence was strongly associated with a PVC burden >10% (*P*<0.001; OR 1.26, 95% CI 1.15-1.39). HF hospitalization was independently predicted by older age (P=0.020; OR 1.04, 95% CI 1.01-1.08), cardiogenic shock (P=0.045; OR 2.45, 95% CI 1.02-5.88), and non-LBBB morphology (P=0.011; OR 3.24, 95% CI 1.31-8.01).
Conclusions: VA incidence was 14%. Mortality was driven by monomorphic VT and VF; VA recurrence by high PVC burden; and HF hospitalization by older age, cardiogenic shock, and non-LBBB. These factors can guide early risk stratification and follow-up.

Keywords: Ventricular arrhythmia; registry, predictors, PVC burden, heart failure hospitalization

Introduction
Ventricular arrhythmias (VAs) represent a group of cardiac rhythm disturbances originating from the ventricular myocardium or His-Purkinje system. They encompass a spectrum ranging from isolated premature ventricular contractions (PVCs) and non-sustained ventricular tachycardia (NSVT) to sustained VT and life-threatening ventricular fibrillation (VF) [1, 2].
The precise incidence and prevalence of VT within the general population remain uncertain. NSVT can occur in both structurally normal and abnormal hearts, although it is markedly more frequent in the latter. The reported incidence of NSVT ranges from 0% to 4% in the general population, with higher prevalence in older individuals and males [3]. Overall, VAs affect approximately 12 per 10,000 individuals under 55 years, increasing to 20 per 10,000 in females and 59 per 10,000 in males aged ≥65 years [4].
Mechanistically, VAs are primarily mediated via three pathways: abnormal automaticity, triggered activity, and reentry. Abnormal automaticity and triggered activity contribute to PVCs, NSVT, and focal VTs originating from a discrete ventricular site, typically observed in patients with normal hearts as well as in a subset of those with structural heart disease

(SHD) [5, 6]. Conversely, reentry is the predominant mechanism underlying VT in SHD, including dilated cardiomyopathy, ischemic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, and cardiac sarcoidosis [7, 8].

Management of VAs is often complex and individualized. Some patients respond to medical therapy alone, whereas others require interventional approaches, with treatment strategies guided by the underlying etiology and identification of precipitating factors that may exacerbate arrhythmic events [9, 10].

Accordingly, this work aimed to evaluate the incidence, risk factors, and clinical outcomes of VAs in patients managed at Tanta University Hospitals.

Patients and methods

Design and population

This observational registry enrolled 400 consecutive patients presenting with VAs to the Cardiology Department, Tanta University Hospitals (Cardiac Intensive Care Unit or Electrophysiology Clinic) between August 2022 and August 2024. The research protocol received approval from the institutional ethics committee. As a registry research, individual informed consent was not obtained, and all patient data were anonymized to ensure confidentiality.

Patient Selection

Inclusion criteria encompassed patients presenting with PVCs (symptomatic or asymptomatic), VT (sustained or non-sustained), or VF. Patients with wide-complex tachycardia ultimately diagnosed as supraventricular tachycardia with aberrancy or via an accessory pathway were excluded.

Clinical Assessment and Data Collection

All participants underwent structured history taking and physical examination. Demographic data included age, sex, and body mass index (BMI). Cardiovascular risk factors and comorbidities recorded were systemic hypertension, diabetes mellitus, dyslipidemia, smoking status (quantified as pack-years), chronic kidney disease, prior stroke, prior myocardial infarction, prior PCI/CABG, family history of premature coronary artery disease (CAD) or sudden cardiac death (<45 years), thyroid disease, prior cardiac surgery, and prior device implantation (pacemaker, ICD, CRT-P, or CRT-D). Clinical symptoms—including chest pain, palpitations, syncope, and dyspnea—and signs of cardiogenic shock were documented.

Laboratory Testing

Venous blood sampling included complete blood count (hemoglobin), renal function tests (creatinine, urea), electrolytes (K^+, Mg^{2+}, Ca^{2+}, Na^+), thyroid-stimulating hormone, cardiac enzymes (when indicated), lipid profile, and serum digoxin in selected cases.

Electrocardiography

A standard 12-lead ECG recorded heart rate, rhythm, PR interval, QRS duration, and corrected QT interval (QTc). Prolonged QTc was defined as ≥480 ms on repeated recordings, while short QTc was ≤360 ms [11]. Additional ECG findings (LVH, poor R-wave progression, and ischemic changes) were noted [12]. VAs were categorized following standard criteria [13]: as monomorphic VT, RVOT VT, LVOT VT, polymorphic VT, bidirectional VT, accelerated idioventricular rhythm, VF, and fascicular VT (RBBB pattern with left axis deviation).

Echocardiography

Transthoracic echocardiography (Philips CX50, S5-1 transducer) evaluated LV dimensions (EDD, ESD), wall thickness, and left ventricular ejection fraction (LVEF) using Simpson's biplane method from apical 4- and 2-chamber views [14]. EF categories followed ESC guidance: preserved ≥50%, mildly reduced 41-49%, reduced ≤40% [15]. Regional wall motion was qualitatively scored across 17 LV segments [16]. Right-ventricular structure and function were evaluated; TAPSE was measured from apical 4-chamber and graded as normal >1.9 cm, borderline 1.5-1.9 cm, or abnormal <1.5 cm [17].

Follow-Up and Outcomes

Patients were followed for 3 months, with management plans reviewed and major adverse cardiac events (MACE) recorded, including cardiac mortality, arrhythmia recurrence (indexed to presenting VA type), heart-failure hospitalization, and stroke.

Statistical Analysis

Data were analyzed using IBM SPSS v20.0. The Kolmogorov-Smirnov test assessed normality of continuous variables. Quantitative data are presented as range, mean±SD, and median (IQR); categorical variables are reported as frequencies (%). Group comparisons employed non-parametric repeated-measures tests where appropriate and chi-square tests for categorical data. Logistic regression (generalized linear models) identified predictors of categorical outcomes, reported as odds ratios (OR) with 95% confidence intervals (CI). An OR of 1 indicated no effect, OR >1 increased odds, and OR <1 decreased odds. CI width reflected estimate precision, and two-sided $p<0.05$ denoted statistical significance at the 95% CI level.

Results

Demographic data, risk factors, and comorbidities were presented in Table 1.

Table 1: Distribution of the studied cases according to demographic data and risk factors.

		(n = 400)
Sex	Male	236 (59.0%)
	Female	164 (41.0%)
Age		52.99±14.96
BMI		27.37±1.67%
Comorbidity	HTN	154 (38.5%)
	DM	131 (32.8%)
	Smokers	164 (41.0%)
	Dyslipidemia	151 (37.8%)
	Ischemic stroke	23 (5.8%)
Renal impairment	CKD on medical treatment	41 (10.3%)
	CKD on dialysis	5 (1.3%)
Prior coronary intervention	1ry PCI MI (revascularized)	24 (6.0%)
	Previous elective PCI	57 (14.3%)
	CABG	15 (3.8%)
	1ry PCI MI & previous elective PCI	13 (3.3%)
	Previous elective PCI and CABG	5 (1.3%)
Prior device	Pacemaker	21 (5.3%)
	ICD	40 (10.0%)
	CRT-P	1 (0.3%)
	CRT-D	9 (2.3%)
Known cardiomyopathy		59 (14.8%)
Prosthetic cardiac valve		10 (2.5%)
Hypothyroidism		15 (3.8%)
Hyperthyroidism		6 (1.5%)
Family history	Sudden cardiac death	11 (2.8%)
	Coronary artery disease	8 (2.0%)
Drug history	Including AAD	114 (28.5%)
	Not including AAD	117 (29.5%)

BMI: body mass index, **AAD:** antiarrhythmic drugs, **CABG:** coronary artery bypass graft, **CKD:** chronic kidney disease, **CRT-P/D:** cardiac resynchronization therapy pacing/ defibrillator, **DM:** diabetes mellitus **HTN:** hypertension, **ICD:** implantable cardioverter device, **MI:** myocardial infarction, **PCI:** percutaneous coronary intervention.

The reported rhythm was sinus rhythm in 317 patients (79.25%), atrial fibrillation in 51 patients (12.8%) and paced rhythm in 32 patients (8%). The rate ranged from 30 bpm to 160 bpm with a mean of (79.68±25.69) b/m. The PR interval ranged from 100-280 msec with a mean of (145.35±32.49) msec. The QRS duration ranged from 80 to 200 msec with a mean of 118.01±29.03msec. Concerning ECG, bundle branch block was presented in 79 patients, 40 patients (10%) presented with LBBB and 52 patients with non LBBB (13%). LVH was reported in 34 ECG (8.5%). Ischemic changes were reported in 108 ECG (27%). High grade AV block was reported in 42 ECG (10.5%). Poor R wave progression appeared in 64 ECG (16%). The QTc was prolonged in ECG of 13 patients (3.3%). Table 2

Table 2: Distribution of the studied cases according to resting ECG.

	(n = 400)
SR	317 (79.25)
AF	51 (12.8)
Paced rhythm	32 (8.0)
LBBB	40 (10)
Non LBBB	52 (13)
LVH	34 (8.5)
Ischaemic changes	108 (27.0)
High grade AV block	42 (10.5)
Poor R wave progression	64 (16.0)
Prolonged QTc interval	13 (3.3)
Rate (b/m)	79.69±25.69
PR interval (msec) (n = 333)	145.35±32.49
QRS width (mesc) (n = 400)	118.01±29.03

AF: atrial fibrillation, **B/M:** beat per minute, **LBBB:** left bundle branch block, **AV:** atrioventricular, **LVH:** left ventricular hypertrophy, **SR:** sinus rhythm.

Echocardiography showed EDD 35-76 mm (mean 52.45±12.55) and ESD 28-62 mm (mean 40.46±12.54), with EF 25-70% (mean 49.53±13.32). Wall-motion abnormalities were present in 220 patients: segmental 104 (26%) and global 116 (29%). RV morphology was normal in 397 (99.3%) and abnormal in 3 (0.7%). TAPSE was normal in 319 (79.8%), fair in 53 (13.3%), and impaired in 28 (7%). Cardiac structure: normal 148 (37%); IHD 176 (44%; EF > 40%: 53; EF ≤ 40%: 123); non-ischemic heart disease 51 (preserved EF 3, mildly reduced 7, reduced 41); special cardiomyopathies 18—ARVC 2 (0.5%), sarcoidosis 2 (0.5%), non-compaction 1 (0.3%), restrictive 9 (2.3%), HCM 4 (1%); mitral valve prolapse 7 (1.8%). Table 3

Table 3: Distribution of the studied cases according to Echo data and cardiac structure.

		(n = 400)
SWMA	Segmental	104 (26.0%)
	Global	116 (29.0%)
RV morphology	Normal	397 (99.3%)
	Abnormal	3 (0.8%)
TAPSE	Fair	53 (13.3%)
	Average	319 (79.8%)
	Impaired	28 (7.0%)
EDD (mm)		52.45±12.55
ESD (mm)		40.46±12.54
IVSD (mm)		10.54±2.18
EF (%)		49.53±13.32
Normal structure		148 (37.0%)
IHD		176 (44.0%)
Non-ischaemic heart disease	Preserved EF (≥50%)	3 (0.8%)
	Mildly reduced EF (41-49%)	7 (1.75%)
	Reduced EF (≤40)	41 (10.25%)
Special cardiomyopathies	Non-compaction	1 (0.3%)
	Restrictive	9 (2.3%)
	ARVC	2 (0.5%)
	Sarcoidosis	2 (0.5%)
	HCM	4 (1.0%)
Mitral valve prolapse		7 (1.8%)

SWMA: segmental wall motion abnormality, **RV:** right ventricle, **EDD:** end diastolic diameter, **ESD:** end systolic diameter, **TAPSE:** tricuspid annular plane systolic excursion, **IVSD:** interventricular systolic diameter, **EF:** ejection fraction, **ARVC:** arrhythmogenic right ventricular cardiomyopathy, **EF:** ejection fraction, **HCM:** hypertrophic cardiomyopathy, **IHD:** ischaemic heart disease.

Laboratory findings showed positive troponin in 86 patients (21.5%). Elevated digoxin was documented in 6 patients (2.4-4.8 ng/mL; mean 3.47±0.84). Creatinine ranged 0.70-7.50 mg/dL (mean 1.22±0.76; median 1.10 [0.90-1.20]) and urea 20.0-132.0 mg/dL (mean 39.92±12.18; median 40.0 [33.0-44.0]). Electrolytes: Na 135-145 mEq/L (mean 139.79±2.86; median 140.0 [137.0-142.0]), K 2.40-6.40 mmol/L (mean 4.13±0.65; median 4.0 [3.80-4.40]), Ca 6.0-10.20 mg/dL (mean 9.32±0.59; median 9.30 [8.85-9.85]), and Mg 1.4-2.2 mg/dL (mean 1.92±0.33; median 1.90 [1.80-2.0]). Thyroid dysfunction was present in 28 patients, comprising hypothyroidism in 20 (5%) and hyperthyroidism in 8 (8%). Table 4

Table 4: Descriptive analysis of the studied cases according to laboratory investigations data.

	M(n = 400)
Serum digoxin level (n = 6)	3.47 - 0.84
Urea (mg/dL)	39.92 - 12.18
Creatinine (mg/dL	1.22 - 0.76
K^+ (mmol/dL)	4.13 - 0.65
Na^+ (mEq/L)	139.79 - 2.86
Ca^{+2} (mg/dL)	9.32 - 0.59
Mg^{+2}	1.92 - 0.33
Hemoglobin (gm/dl)	12.78 - 1.54
Platelets (number/mm3)	249.61 - 62.42
Hypothyroidism	20 (5.0%)
Hyperthyroidism	8 (92.0%)

Eighteen patients missed their follow-up visits (4.5%). Cardiac mortality had been occurred in 28 patients (7%) (17 patients had IHD, 9 patients had NICM with reduced EF, 1 had mildly reduced EF and 1 patient had restrictive cardiomyopathy). Recurrence of VA was observed in 62 patients(26 patients had monomorphic VT, 3 patients had pleomorphic PVCs, one patient had LVOT VT, 21 patients had RVOT PVCs, 2 patients had RVOT VT and 2 patients had fascicular VT) (6 patients had IHD, 29 patients had NICM with reduced EF, 1 had HCM and 26 patients had normal cardiac structure). Heart failure (HF) hospitalization occurred in 47 patients (11.7%) (8 patients had IHD, 35 had NICM with reduced EF and 4 patients had mildly reduced EF). Stroke occurred in 11 patients (2.8%) (one patient had HCM, 5 patients had IHD, 4 patients had NICM with reduced EF and 1 patient had mildly reduced EF).

The univariate and multivariate analysis were performed to investigate the possible predictive factors for cardiac mortality. In the univariate analysis: there were several factors which were good predictors of cardiac mortality: cardiogenic shock (P= 0.001), monomorphic VT (p<0.001), VF (P <0.001), EF≤40% (p<0.001), IHD (P=0.011) and NICM (P=0.049). In the multivariate analysis, the presence of the monomorphic VT and the VF had significant values for the cardiac mortality with P value (0.004 and 0.003) respectively. Table 5

Table 5: Univariate and multivariate logistic regression analysis for predictors of cardiac mortality (n= 28).

	Univariate		#Multivariate	
	P	**OR (LL - UL 95%C.I)**	**P**	**OR (LL - UL 95%C.I)**
Sex (female vs male)	0.546	1.268(0.587 - 2.742)		
Age	0.087	1.026(0.996 - 1.056)		
HTN	0.624	1.215(0.558 - 2.642)		
DM	0.943	0.971(0.427 - 2.209)		
Smokers	0.165	1.727(0.799 - 3.734)		
Dyslipidemia	0.328	1.470(0.679 - 3.180)		
Renal impairment	0.893	0.918(0.266 - 3.170)		
Prior PCI	0.835	1.073(0.551 - 2.090)		
Cardiogenic shock	0.001*	4.045(1.712 - 9.555)	0.292	1.694 (0.636 - 4.509)
Monomorphic VT	<0.001*	5.640(2.512 - 12.661)	0.004*	4.157 (1.565 - 11.040)
LBBB like PVCs (RVOT PVCs)	0.996	NA		
PVCs another morphology	0.152	0.344(0.080 - 1.484)		
VF	<0.001*	6.571(2.778 - 15.543)	0.003*	5.127 (1.724 - 15.246)
EF≤40%	<0.001*	0.920(0.885 - 0.956)	0.203	0.967 (0.919 - 1.018)
Normal structure	0.995	NA		
IHD	0.011*	2.891(1.274 - 6.560)		
Non Ischaemic heart disease	0.049*	2.485(0.999 - 6.182)	0.857	1.113 (0.349 - 3.550)
Type of AAD				
Class II B blocker	0.785	1.086(0.599-1.970)		
Class amiodarone	0.759	1.136(0.503-2.564)		

AAD: antiarrhythmic drugs, **DM:** diabetes mellitus, **EF:** ejection fraction, **HTN:** hypertension, **IHD:** ischaemic heart disease, **LBBB:** left bundle branch block, **PCI:** percutaneous coronary intervention, PVCs premature ventricular contractions, **RVOT:** right ventricular outflow tract, **VF:** ventricular fibrillation.

The univariate regression analysis for the predictors of VA recurrence, the following parameters were good predictors: monomorphic VT (P=0.025), RVOT PVC (P=0.006), EF≤40% (P=0.014), the normal structure (P=0.010), NICM (P=0.014) and the burden of the PVCs above 10% (*P*<0.001). In the multivariate analysis, the high PVCs burden above 10% was a significant predictor for the recurrence of the VA (P value<0.001). Table 6

Table 6: Univariate and multivariate logistic regression analysis for predictors of recurrence of the ventricular arrhythmia (n= 62).

	Univariate		#Multivariate	
	P	**OR (LL - UL 95%C.I)**	**P**	**OR (LL - UL 95%C.I)**
Sex (female vs male)	0.497	0.824(0.471 - 1.441)		
Age	0.842	0.998(0.980 - 1.016)		
HTN	0.375	1.282(0.741 - 2.217)		
DM	0.928	0.974(0.546 - 1.737)		
Smokers	0.497	0.824(0.471 - 1.441)		
Dyslipidemia	0.494	0.820(0.464 - 1.449)		
Renal impairment	0.217	1.613(0.755 - 3.449)		
Prior PCI	0.476	1.236(0.690 - 2.215)		
Cardiogenic shock	0.153	0.461(0.159 - 1.332)		
Monomorphic VT	0.025*	1.910(1.083 - 3.368)	0.998	NA
LBBB like PVCs (RVOT PVCs)	0.006*	2.267(1.262 - 4.071)	0.547	1.451(0.432 - 4.873)
PVCs another morphology	0.303	0.659(0.299 - 1.456)		
VF	0.988	NA		
EF≤40%	0.014*	1.027(1.005 - 1.049)	0.784	1.013(0.926 - 1.107)
Normal structure	0.009*	2.068(1.197 - 3.573)	0.998	NA
IHD	0.083	0.605(0.343 - 1.067)		
Non-Ischaemic heart disease	0.014*	2.373(1.195 - 4.713)	0.999	NA
QTC interval	0.447	1.668(0.446 - 6.241)		
PVCs burden (>10%)	<0.001*	1.255(1.153 - 1.366)	<0.001*	1.264(1.150 - 1.389)
Type of AAD				
Class IV Ca channel blocker	0.340	1.656(0.588 - 4.668)		
Class III Amiodarone	0.759	1.136(0.503 - 2.564)		
Class II B blocker	0.785	1.086(0.599 - 1.970)		
Class IC propafenone	0.291	1.874(0.584 - 6.010)		
Class IB lidocaine	0.929	0.907(0.107 - 7.668)		
ICD	0.899	1.061(0.423 - 2.661)		
Catheter ablation	0.083	0.519(0.247 - 1.089)		

AAD: antiarrhythmic drugs, **DM:** diabetes mellitus, **EF:** ejection fraction, **HTN:** hypertension, **ICD:** implantable cardioverter defibrillator, **IHD:** ischaemic heart disease, **LBBB:** left bundle branch block, **PCI:** percutaneous coronary intervention, PVCs premature ventricular contractions, **RVOT:** right ventricular outflow tract, **VF:** ventricular fibrillation.

The univariate regression analysis for the predictors of HF hospitalization, the following parameters were good predictors: previous renal impairment (P=0.029), cardiogenic shock (P<0.001), Monomorphic VT (P=0.002), the reduction of the EF≤40% (*P*<0.001), NICM (*P*<0.001), NYHA class (III-IV) (P=0.049) and the non LBBB (*P*<0.001). In the multivariate analysis, with advancement of the age (P value=0.020), in the presence of the cardiogenic shock (P value=0.045) and the presence of the non LBBB (0.011), all had significant values for HF hospitalization at follow up. Table 7

Table 7: Univariate and multivariate logistic regression analysis for predictors of heart failure (n= 47).

	Univariate		#Multivariate	
	P	**OR (LL - UL 95%C.I)**	**P**	**OR (LL - UL 95%C.I)**
Sex (female vs male)	0.474	0.794(0.422 - 1.493)		
Age	0.009*	1.033(1.008 - 1.058)	0.020*	1.044 (1.007 - 1.083)
HTN	0.325	0.722(0.377 - 1.382)		
DM	0.235	1.460(0.782 - 2.726)		
Smokers	0.474	0.794(0.422 - 1.493)		
Dyslipidemia	0.233	0.669(0.345 - 1.295)		
Renal impairment	0.029*	2.380(1.092 - 5.186)	0.308	1.656 (0.628 - 4.369)
Prior PCI	0.835	1.073(0.551 - 2.090)		
Cardiogenic shock	<0.001*	4.545(2.234 - 9.248)	0.045*	2.451 (1.021 - 5.883)
Monomorphic VT	0.002*	2.732(1.466 - 5.091)	0.787	1.113 (0.512 - 2.419)
LBBB like PVCs (RVOT PVCs)	0.996	NA		
PVCs another morphology	0.366	0.661(0.269 - 1.623)		
VF	0.079	2.131(0.915 - 4.962)		
EF≤40%	<0.001*	0.956(0.932 - 0.980)	0.862	1.004 (0.957 - 1.054)
Normal structure	<0.001*	0.135(0.047 - 0.384)	1.000	NA
IHD	0.179	1.520(0.826 - 2.799)		
Non-Ischaemic heart disease	<0.001*	4.128(2.042 - 8.341)	0.070	2.149 (0.938 - 4.922)
NYHA class (III+ IV)	0.049*	2.411(1.001 - 5.804)	0.144	2.094 (0.776 - 5.652)
Recurrence of the ventricular arrhythmia	0.331	0.618(0.234 - 1.630)		
ICD	0.899	1.061(0.423 - 2.661)		
CRT (de no vo or upgrading)	0.078	2.131(0.915 - 4.962)		
LBBB	0.727	1.194(0.441 - 3.233)		
Non LBBB	<0.001*	4.272(2.031 - 8.985)	0.011*	3.240 (1.311 - 8.007)

CRT: cardiac resynchronization therapy, **DM:** diabetes mellitus, **EF:** ejection fraction, **HTN:** hypertension, **ICD:** implantable cardioverter defibrillator, **IHD:** ischaemic heart disease, **LBBB:** left bundle branch block, **NYHA class:** New York heart association, **PCI:** percutaneous coronary intervention, PVCs premature ventricular contractions, RVOT: right ventricular outflow tract, **VF:** ventricular fibrillation.

Discussion

VAs, ranging from benign ectopy to fatal VF, remain a major clinical challenge [18]. So, this research enrolled 400 consecutive patients with VAs at Tanta University Hospitals to provide insight into their incidence, risk factors, and outcomes in a real-world tertiary care setting.

The mean age of the research population was 55.0 (45.0-66.0) years, with advancing age significantly associated with increased HF symptoms during follow-up in both univariate (P=0.009) and multivariate analyses (P=0.020). This may relate to the higher prevalence of cardiomyopathy and subsequent HF hospitalizations in older patients, consistent with Uchmanowicz and co-authors [19], who reported a mean age of 72.1 ± 7.9 years in 330 HF patients where frailty predicted rehospitalizations. Among 400 patients, 263 were male and 164 females, with no significant differences between genders regarding cardiac mortality, HF hospitalization, or VA recurrence (P=0.546, 0.474, and 0.497, respectively). This aligns with Darma and co-authors [20], who studied 309 patients post-VT ablation (271 males, 38 females) over 34 ± 28 months and found similar outcomes.

In the current research, hypertension, diabetes, dyslipidemia, and smoking showed no significant impact on cardiac mortality, HF, or VA recurrence, aligning with Darma and co-authors [20], who also found no correlation between risk factors and mortality (HTN P=0.728, DM P=0.523). Conversely, prior renal impairment significantly affected HF hospitalization in univariate analysis (P=0.029) but lost significance in multivariate analysis (P=0.308), likely due to the small number of CKD cases (n=46). Renal impairment was not associated with VA recurrence (P=0.217), which contrasts with Weidner and co-authors [21], who reported higher VA recurrence in CKD patients with ICDs (P=0.009; VT P=0.026, VF P=0.030). The discrepancy may be attributed to differences in sample size and CKD prevalence.

In the present research, the number of patients who had family history of sudden cardiac death or coronary artery disease had no significant value in the outcomes. This came in agreement with Alenazy and co-authors [22]. In This registry that enrolled 2610 HF patients during the 14 months recruitment period between October 2009 and December 2010, they found that total number of patients who had family history of SCD or CAD (23 and 88 patients) respectively and these had no significant values in the presence of the VA between the patients (P value:0.246, 0.389) respectively.

In the current research, 6 patients presented with digoxin toxicity manifested by elevated serum digoxin levels and VAs: 3 with bidirectional VT, 2 with RVOT PVCs in bigeminy form, and 1 with pleomorphic VT with a very short coupling interval (R-on-T phenomenon). All cases were associated with significant hypokalemia, which aggravated the arrhythmias. These findings align with reports by Aung and co-authors [23] and Pfirman and co-authors [24], who described the link between digoxin toxicity and bidirectional VT. The proposed mechanisms include

enhanced vagal tone at the AV node causing slow conduction and Na/Ca pump inhibition leading to intracellular calcium overload, which promotes delayed afterdepolarizations and triggered activity, predisposing to polymorphic VT.

In the present research, advanced NYHA class (III-IV) significantly affected outcomes in univariate analysis (P=0.049) but not in multivariate analysis (P=0.144). Presentation with cardiogenic shock, however, was a strong predictor of both mortality and HF hospitalization in univariate (*P*<0.001) and multivariate analyses (P=0.045). These findings align with Sundermeyer and co-authors [25], who studied 1,030 patients with HF-related cardiogenic shock across 16 tertiary centers and found higher severity and mortality in acute-on-chronic HF-CS compared to de novo cases (mortality 55.9% vs. 45.5%, adjusted HR=1.38, 95% CI: 1.10-1.72, P=0.005).

In the current research, non-LBBB QRS morphology was a significant predictor of HF hospitalization during follow-up, both in univariate (P<0.001) and multivariate analyses (P=0.011). This finding agrees with Pellicori and co-authors [26], who studied 877 patients (36% with QRS ≥120 ms) and reported lower RV ejection fraction in RBBB compared to LBBB [46% (37-57) vs. 52% (42-61), P=0.014] and higher mortality with RBBB [HR 1.98, 95% CI: 1.37-2.86, *P*<0.001]. Similarly, Kristensen and co-authors [27], in a large cohort of 11,861 patients followed for 2.5 years, found higher risks of cardiovascular death or HF hospitalization with wide QRS irrespective of morphology: LBBB HR 1.36 (1.23-1.50), RBBB HR 1.54 (1.31-1.79), and IVCD HR 1.65 (1.40-1.94), all *P*<0.001. The stronger predictive value of non-LBBB in the current research may relate to the larger number of non-LBBB cases and the beneficial impact of CRT implantation in LBBB patients, unlike Kristensen *et al*., who excluded device recipients.

In the current research, QTc prolongation was not a significant predictor of VA recurrence in univariate analysis (P=0.447). This aligns with Fernandes and co-authors [28], who retrospectively analyzed 6,280 ECGs from 5,056 patients and identified 387 with prolonged QTc (mean 502.14±32.2 ms; prevalence 7.95%). The cohort, predominantly male (53.1%) with mean age 73.6±14.7 years, showed that only 20% were symptomatic mainly syncope (50%) and no VAs were observed.

In the current research population, the EF ranged from 25.0 - 70.0%, with a mean of 49.53±13.32%. The reduction of EF% below 40% was a predictor of cardiac mortality, HF hospitalization and recurrence of VA in the follow up (P value<0.001, 0.014 and<0.001 respectively). This was in agreement with Rusnak et. [29], they concluded a large retrospective registry, including all consecutive ICD recipients with episodes of VT or fibrillation (VF) from 2002 to 2016. A total of 528 consecutive ICD recipients were included (51% with LVEF ≥35% and 49% with LVEF <35%). EF<35% has significant affection in the recurrence of the VA (P value= 0.067) and affecting hospitalization and all-cause mortality.

In the present research, abnormal cardiac structure predicted VA recurrence, with non-ischemic heart disease particularly reduced EF showing significance in univariate analysis compared to IHD (P=0.014). This aligns with Darma and co-authors [20], who found no effect of structural type on VT recurrence (P=0.480) or long-term mortality (P=0.687), likely due to differences in catheter ablation rates and revascularization. Both ischemic and NICM significantly predicted cardiac mortality in univariate (P=0.011) and multivariate (P=0.049) analyses, which contrasts with Narins and co-authors [30], who reported ischemic patients had over a twofold increased risk of all-cause, cardiac, and non-cardiac mortality compared to NICM. Furthermore, NICM was associated with higher HF hospitalization during follow-up (P<0.001), possibly explained by myocardial revascularization preserving EF and survival in ischemic cases, as well as variation in device implantation between groups.

This research had several limitations. First, the sample size was relatively small, and the follow-up period was short. Second, the lack of some facilities and interventional procedures in the tertiary center may have influenced the management and outcomes. Finally, being a single-center research, the results may not be fully generalizable to other populations or settings.

Conclusions

The incidence of VAs over the past two years was 14% among patients presenting to Tanta University Hospitals. Long-term outcomes including cardiac mortality, VA recurrence, and HF hospitalization were significantly influenced by several predictors: NYHA class III/IV, cardiogenic shock, monomorphic VT, LVEF <40%, and both ischemic and non-ischemic etiologies, all of which were associated with major adverse cardiac events.

References

1. Haïssaguerre M, Vigmond E, Stuyvers B, Hocini M, Bernus O. Ventricular arrhythmias and the His-Purkinje system. Nat Rev Cardiol. 2016;13:155-66.
2. Katritsis DG, Zareba W, Camm AJ. Nonsustained ventricular tachycardia. J Am Coll Cardiol. 2012;60:1993-2004.
3. Khan MT, Irfan G, Ansari S, Mumtaz Z, Qadir F, Shafquat A. Incidence of rapid rate non-sustained and sustained ventricular tachycardia in implantable cardioverter-defibrillator recipients and its correlation with heart failure guideline-directed medical therapy compliance. J Arrhythm. 2025;41:e70156.
4. Ozawa K, Funabashi N, Takaoka H, Uehara M, Kobayashi Y. Specific organized substrates of ventricular fibrillation: comparison of 320-slice CT heart images in non-ischemic ventricular fibrillation subjects with non-ischemic sustained and non-sustained ventricular tachycardia subjects. Int J Cardiol. 2013;168:1472-8.
5. Benito B, Josephson ME. Ventricular tachycardia in coronary artery disease. Rev Esp Cardiol (Engl Ed). 2012;65:939-55.
6. Laws JL, Lancaster MC, Shoemaker MB, Stevenson WG, Hung RR, Wells Q, *et al*. Arrhythmias as presentation of genetic cardiomyopathy. Circ Res. 2022;130:1698-722.
7. Cronin EM, Bogun FM, Maury P, Peichl P, Chen M, Namboodiri N, *et al*. 2019 HRS/EHRA/APHRS/LAHRS expert consensus statement on catheter ablation of ventricular arrhythmias. Heart Rhythm. 2020;17:e2-154.
8. Stanciulescu LA, Dorobantu M, Vatasescu R. Targeting ventricular arrhythmias in non-ischemic patients:

advances in diagnosis and treatment. Diagnostics (Basel). 2025;15:1-12.
9. Mirza M, Strunets A, Shen WK, Jahangir A. Mechanisms of arrhythmias and conduction disorders in older adults. Clin Geriatr Med. 2012;28:555-73.
10. Serhiyenko VA, Serhiyenko AA. Cardiac autonomic neuropathy: risk factors, diagnosis and treatment. World J Diabetes. 2018;9:1-24.
11. Zeppenfeld K, Tfelt-Hansen J, de Riva M, Winkel BG, Behr ER, Blom NA, *et al*. 2022 ESC guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. Eur Heart J. 2022;43:3997-4126.
12. Pedersen LR, Kristensen AMD, Petersen SS, Vaduganathan M, Bhatt DL, Juel J, *et al*. Prognostic implications of left ventricular hypertrophy diagnosed on electrocardiogram vs echocardiography. J Clin Hypertens (Greenwich). 2020;22:1647-58.
13. Prystowsky EN, Padanilam BJ, Joshi S, Fogel RI. Ventricular arrhythmias in the absence of structural heart disease. J Am Coll Cardiol. 2012;59:1733-44.
14. Mitchell C, Rahko PS, Blauwet LA, Canaday B, Finstuen JA, Foster MC, *et al*. Guidelines for performing a comprehensive transthoracic echocardiographic examination in adults: recommendations from the American Society of Echocardiography. J Am Soc Echocardiogr. 2019;32:1-64.
15. Bauersachs J, Soltani S. [Guidelines of the ESC 2021 on heart failure]. Herz. 2022;47:12-8.
16. Sanjeevi G, Gopalakrishnan U, Pathinarupothi RK, Madathil T. Automatic diagnostic tool for detection of regional wall motion abnormality from echocardiogram. J Med Syst. 2023;47:13-21.
17. Duanmu Y, Goldsmith AJ, Henwood PC, Platz E, Hoyler JE, Kimberly HH. Visual estimation of tricuspid annular plane systolic excursion by emergency medicine clinicians. West J Emerg Med. 2020;21:1022-8.
18. Batul SA, Olshansky B, Fisher JD, Gopinathannair R. Recent advances in the management of ventricular tachyarrhythmias. F1000Res. 2017;6:1027-35.
19. Uchmanowicz I, Kuśnierz M, Wleklik M, Jankowska-Polańska B, Jaroch J, Łoboz-Grudzień K. Frailty syndrome and rehospitalizations in elderly heart failure patients. Aging Clin Exp Res. 2018;30:617-23.
20. Darma A, Bertagnolli L, Dinov B, Torri F, Shamloo AS, Lurz JA, *et al*. Predictors of long-term mortality after catheter ablation of ventricular tachycardia in a contemporary cohort of patients with structural heart disease. Europace. 2020;22:1672-9.
21. Weidner K, Behnes M, Weiß C, Nienaber C, Reiser L, Bollow A, *et al*. Impact of chronic kidney disease on recurrent ventricular tachyarrhythmias in ICD recipients. Heart Vessels. 2019;34:1811-22.
22. Alenazy B, Tharkar S, Kashour T, Alhabib KF, Alfaleh H, Hersi A. In-hospital ventricular arrhythmia in heart failure patients: seven-year follow-up of the multi-centric HEARTS registry. ESC Heart Fail. 2019;6:1283-90.
23. Aung TPP, Buddhavarapu S, Park WJ, Ayala-Rodriguez C, Oo ZT, Kyaw H. A visual resolution of cardiotoxicity: a case report of digoxin-induced bidirectional ventricular tachycardia. Cureus. 2021;13:1-5.
24. Pfirman KS, Huffman TR, Singh A. Digoxin-induced bidirectional ventricular tachycardia in a patient with hypokalemia. JAMA Intern Med. 2021;181:850-2.
25. Sundermeyer J, Kellner C, Beer BN, Besch L, Dettling A, Bertoldi LF, *et al*. Clinical presentation, shock severity and mortality in patients with de novo versus acute-on-chronic heart failure-related cardiogenic shock. Eur J Heart Fail. 2024;26:432-44.
26. Pellicori P, Joseph AC, Zhang J, Lukaschuk E, Sherwi N, Bourantas CV, *et al*. The relationship of QRS morphology with cardiac structure and function in patients with heart failure. Clin Res Cardiol. 2015;104:935-45.
27. Kristensen SL, Castagno D, Shen L, Jhund PS, Docherty KF, Rørth R, *et al*. Prevalence and incidence of intra-ventricular conduction delays and outcomes in patients with heart failure and reduced ejection fraction: insights from PARADIGM-HF and ATMOSPHERE. Eur J Heart Fail. 2020;22:2370-9.
28. Fernandes DA, Camões GF, Ferreira D, Queijo C, Fontes-Ribeiro C, Gonçalves L, *et al*. Prevalence and risk factors for acquired long QT syndrome in the emergency department: a retrospective observational study. World J Emerg Med. 2023;14:454-61.
29. Rusnak J, Behnes M, Weiß C, Nienaber C, Reiser L, Schupp T, *et al*. Impact of left ventricular ejection fraction on recurrent ventricular tachyarrhythmias in recipients of implantable cardioverter defibrillators. Cardiology. 2020;145:359-69.
30. Narins CR, Aktas MK, Chen AY, McNitt S, Ling FS, Younis A, *et al*. Arrhythmic and mortality outcomes among ischemic versus nonischemic cardiomyopathy patients receiving primary ICD therapy. Clin Electrophysiol. 2022;8:1-11.

How to Cite This Article
Ahmed NH, Badr SF, Salama MM, Elshedody SA, Khalfallah M. Registry of patients with ventricular arrhythmia in Tanta university hospitals incidence, risk factors and outcomes. International Journal of Cardiology Sciences 2025; 7(2): 97-104.