

Arrhythmias in End-Stage Renal Disease on Maintenance Haemodialysis: A Cross-Sectional Study

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ABSTRACT

ARTICLE DETAILS

Background

Patients with end-stage renal disease (ESRD) on hemodialysis are at high risk for cardiovascular mortality, with arrhythmias being a major contributor. Data on this issue are scarce in low- and middle-income countries like Myanmar.

Objectives

This study aimed to determine the prevalence of arrhythmias in ESRD patients on maintenance hemodialysis, identify the predictors of these arrhythmias, and explore their clinical implications (short-term clinical outcomes).

Methods

We conducted a cross-sectional study of 140 adult ESRD patients on maintenance hemodialysis at a single center in Myanmar. All patients underwent 24-hour Holter ECG monitoring. We collected data on demographics, comorbidity, and laboratory and echocardiographic data. Logistic regression was used to identify predictors of arrhythmias.

Results

The mean age of the patients was 51.1 ± 14.6 years, and 70% were female. The overall prevalence of any arrhythmia was 65.7%. The most common arrhythmias were premature atrial contractions (36.4%) and premature ventricular contractions (31.4%). Atrial fibrillation was present in 7.1% of patients. In multivariate analysis, older age (OR 1.03, 95% CI 1.00–1.06), left ventricular hypertrophy (OR 3.12, 95% CI 1.44–6.76), abnormal left atrial volume index (OR 2.46, 95% CI 1.15–5.27), and higher creatinine levels (OR 1.17, 95% CI 1.00–1.38) were independent predictors of arrhythmias. Patients with arrhythmias had higher rates of hospitalization, emergency visits, and 3-month mortality.

Conclusion

Arrhythmias are highly prevalent in ESRD patients on hemodialysis in Myanmar and are associated with adverse clinical outcomes. Routine Holter monitoring could be a valuable tool for risk stratification in this high-risk population, particularly for those with echocardiographic evidence of LVH or abnormal LAVI.

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KEYWORDS: End-Stage Renal Disease, Hemodialysis, Arrhythmias, Holter ECG Monitoring, Left Ventricular Hypertrophy, Sudden Cardiac Death

CENTRAL ILLUSTRATION

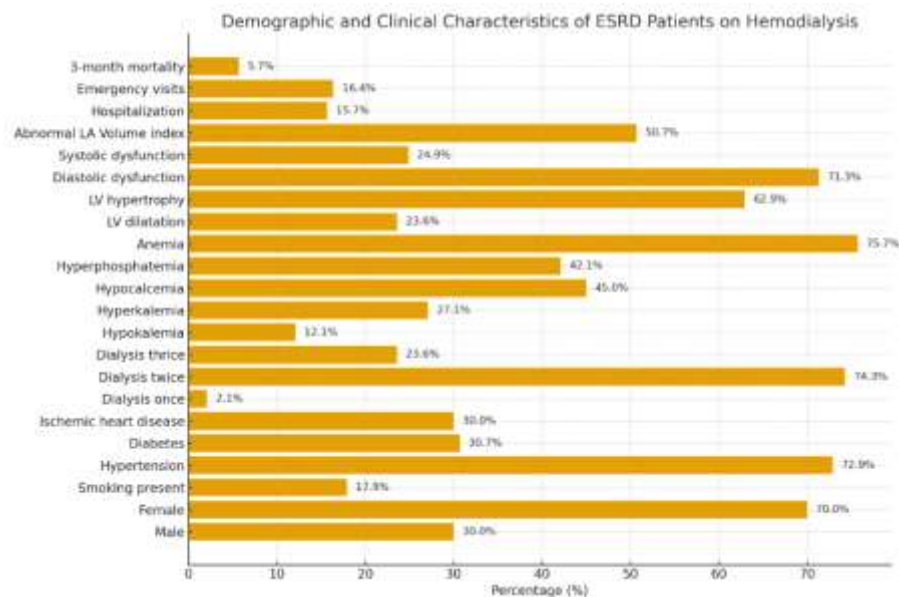


Figure 1. Demographic and Clinical Characteristics of ESRD patients on Hemodialysis

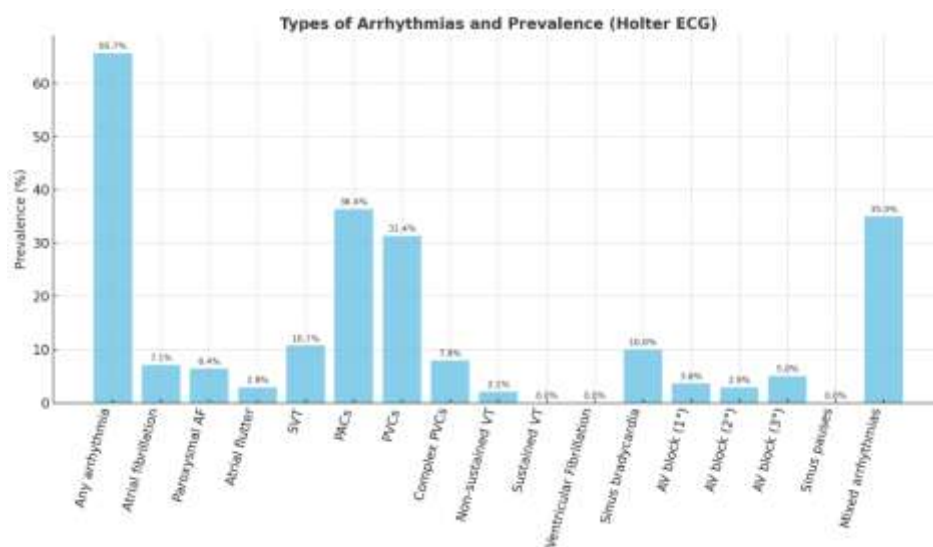


Figure 2. Types of Arrhythmias and Prevalence (Holter ECG)

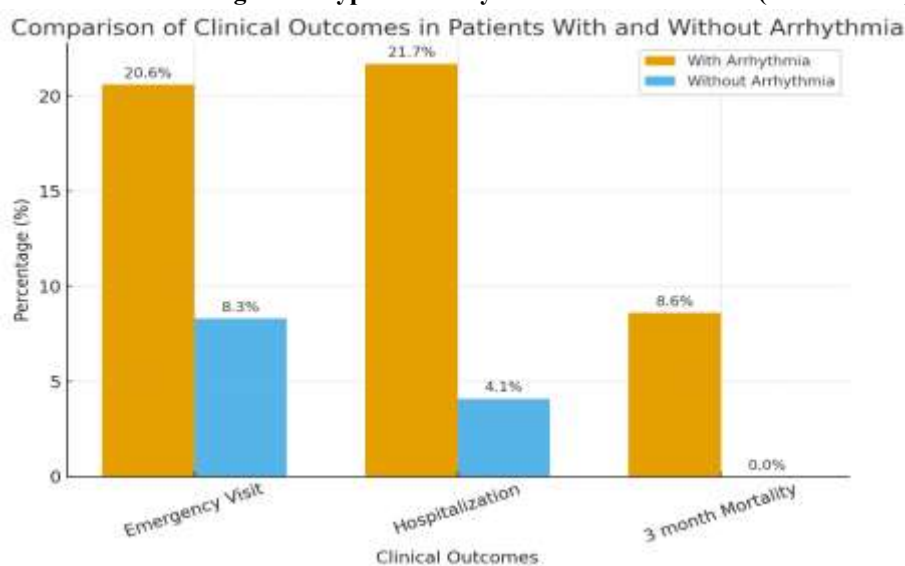


Figure 3. Comparison of Clinical Outcomes in Patients with and without Arrhythmia

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INTRODUCTION

End-stage renal disease (ESRD) is a growing public health challenge worldwide, imposing a significant burden on patients and healthcare systems [1]. Patients with ESRD requiring maintenance haemodialysis have mortality rates 10 to 20 times higher than the age-matched general population. This staggering mortality is primarily driven by cardiovascular disease, which accounts for approximately 50% of all deaths in this vulnerable population [2].

Among cardiovascular complications, sudden cardiac death (SCD) is the leading cause of mortality in haemodialysis patients, responsible for about one-quarter of all-cause deaths. Malignant cardiac arrhythmias are the principal underlying mechanism for SCD [3]. Both the uremic state and the haemodialysis procedure create a potent pro-arrhythmic environment, making these patients exceptionally susceptible to cardiac electrical instability [4]. The pathophysiology of arrhythmias in the ESRD population is complex. Chronic volume and pressure overload lead to significant structural remodelling of the heart, often termed "uremic cardiomyopathy," which is characterized by left ventricular hypertrophy (LVH), myocardial fibrosis, and diastolic dysfunction [4]. These structural changes create an anatomical substrate for re-entrant arrhythmias. Furthermore, the intermittent nature of haemodialysis causes rapid shifts in fluid and electrolyte concentrations—particularly potassium and calcium—which can alter myocardial excitability [5]. Other contributing factors include chronic inflammation, heightened sympathetic nervous system activity, activation of the renin-angiotensin-aldosterone system, and the accumulation of uremic toxins that may have direct cardiotoxic effects [6].

While the burden of arrhythmias in haemodialysis patients is well-documented in high-income countries, there is a significant lack of data from low- and middle-income countries (LMICs) like Myanmar. In these resource-constrained settings, challenges are often magnified, with limited access to advanced diagnostic tools like 24-hour Holter ECG monitoring [7]. This knowledge gap hinders the development of effective, context-specific strategies for risk stratification and prevention of SCD.

This study was designed to address this gap by systematically evaluating the prevalence and characteristics of arrhythmias in a cohort of ESRD patients on maintenance haemodialysis in Myanmar. We aimed to: (1) determine the prevalence and types of arrhythmias using 24-hour Holter monitoring; (2) identify the clinical, laboratory, and echocardiographic predictors of these arrhythmias; and (3) assess the association between arrhythmias and short-term adverse clinical outcomes.

METHODS

Study Design and Population

This cross-sectional study was conducted at the 700 Bedded General Hospital in Pyin Oo Lwin, Myanmar, from August

2024 to July 2025. We enrolled 140 adult patients with ESRD who had been on maintenance haemodialysis for at least three months. The study was approved by the local ethical committee, and all patients provided written informed consent.

Inclusion and Exclusion Criteria

Inclusion criteria were age >18 years and ESRD on maintenance haemodialysis for ≥ 3 months. Exclusion criteria included known congenital or significant valvular heart disease and the presence of a pacemaker or implantable cardioverter-defibrillator (ICD).

Data Collection

We collected baseline data on demographics, comorbidities (hypertension, diabetes, ischaemic heart disease), smoking status and dialysis frequency. Laboratory parameters, including serum creatinine haemoglobin, potassium, calcium, and phosphate, were collected on the same day as the Holter monitoring.

Holter ECG Monitoring

All patients underwent 24-hour continuous Holter ECG monitoring on the day before a haemodialysis session. The recordings were analyzed for the presence of atrial fibrillation (AF), paroxysmal AF, atrial flutter, supraventricular tachycardia (SVT), premature atrial contractions (PACs), premature ventricular contractions (PVCs), complex PVCs (Lown class III-VI), non-sustained ventricular tachycardia (NSVT), sustained VT, ventricular fibrillation, sinus bradycardia, atrioventricular (AV) blocks, and sinus pauses.

Echocardiographic assessment

Echocardiographic measurements were performed according to the recommendations of the American Society of Echocardiography [8].

Statistical Analysis

Statistical analysis was performed using SPSS version 25.0. Continuous variables were expressed as mean $\pm$ SD, and categorical variables as percentages. A univariate logistic regression was performed to identify potential predictors of arrhythmias. Variables with a p-value < 0.10 in the univariate analysis were included in a multivariate logistic regression model with stepwise forward selection to determine independent predictors. A p-value < 0.05 was considered statistically significant.

RESULTS

Baseline and Clinical Characteristics

The demographic and clinical characteristics of the 140 patients are shown in Table 1. The mean age was 51.1 ± 14.6 years, and 70% were female. The most common comorbidities were hypertension (72.9%), diabetes (30.7%), and ischaemic heart disease(IHD) (30%). Laboratory findings revealed high rates of anaemia (75.7%), hypocalcemia (45%), and hyperphosphatemia (42.1%).

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Profound structural cardiac abnormalities were common, with 62.9% of patients having LVH and 71.3% having diastolic dysfunction. Additionally, 50.7% had an abnormal LAVI.

Table 1. Demographic and Clinical Characteristics of ESRD Patients on Hemodialysis

	Variable	Category / Unit (total sample = 140)
1.	Age(mean ± SD)	51.1 ± 14.6 years
2.	Sex	male / female 42 (30%)/98(70%)
3.	Smoking	25(17.9%)
	Comorbidities	
4.	Hypertension	102(72.9%)
5.	Diabetes	43(30.7%)
6.	IHD	42(30%)
7.	Dialysis frequency	once/twice / thrice weekly (%) 3(2.1%)/104 (74.3%)/33(23.6%)
	Laboratory values	
8.	Hyperkalemia	38(27.1%)
9.	Hypocalcemia	63 (45%)
10.	Hyperphosphatemia	59(42.1%)
11.	Anaemia	106 (75.7%)
12.	Creatinine (mean ± SD)	6.1 ± 1.5
	Echocardiography	
13.	LV dilatation	33(23.6%)
14.	LVH	mild /moderate/severe 68(48.6%)/ 14(10%)/6(4.3%)
15.	Diastolic dysfunction	mild /moderate/severe 80(57.1%)/17(12.1%)/3(2.1%)
16.	Systolic dysfunction	mild /moderate/severe 31(22.1%)/3(2.1%)/1(0.7%)
17.	LAVI (mean ± SD)	abnormal 71(50.7%) 35.7 ± 4.7

Prevalence of Arrhythmias

The overall prevalence of any arrhythmia detected on 24-hour Holter monitoring was 65.7% (92 out of 140 patients). The most frequent arrhythmias were PACs at 36.4% and PVCs at 31.4%. Clinically significant arrhythmias included AF (7.1%), paroxysmal AF (6.4%), SVT (10.7%), and NSVT (2.1%). The types and prevalence of all detected arrhythmias are detailed in Table 2.

Table 2. Types of Arrhythmias and Prevalence (Holter ECG)

	Type of Arrhythmia	N (%)
1.	Any arrhythmia (overall prevalence)	92 (65.7%)
2.	AF	10(7.1%)
3.	Paroxysmal AF	9(6.4%)

4.	Atrial flutter	4(2.9%)
5.	SVT	15(10.7%)
6.	PACs	51(36.4%)
7.	PVCs	44(31.4%)
8.	Complex PVCs	11(7.9%)
9.	Non-sustained VT	3(2.1%)
10.	Sustained VT	0
11.	Ventricular Fibrillation	0
12.	Sinus bradycardia	14(10%)
13.	AV blocks	16 (11.5%)
14.	Sinus pauses (>2s)	0
15.	Mixed arrhythmias	49(35%)

Predictors of Arrhythmias

In the univariate analysis, factors associated with arrhythmias included older age, several markers of structural heart disease (LV dilatation, LVH, diastolic and systolic dysfunction, abnormal LAVI), anaemia, and higher creatinine levels. After adjusting for confounding variables in the multivariate logistic regression, four factors emerged as independent predictors of any arrhythmia: older age, LVH, abnormal LAVI, and higher creatinine levels. LVH was the strongest predictor, increasing the odds of arrhythmia by over three-fold (OR 3.12). A complete list of predictors is shown in Table 3.

Table 3. Univariate and Multivariate Predictors of Arrhythmias

Variable	Univariate OR (95% CI)	p-value
Age (per year)	1.03 (1.01 – 1.06)	0.012
Male sex	1.18 (0.60 – 2.33)	0.633
Hypertension	1.87 (0.82 – 4.27)	0.135
Diabetes	1.35 (0.67 – 2.72)	0.402
IHD	1.92 (0.87 – 4.25)	0.108
LV dilatation	3.03 (1.07 – 8.60)	0.037
LVH	4.11 (2.06 – 8.21)	<0.001
Diastolic dysfunction	3.57 (1.83 – 6.96)	<0.001
Systolic dysfunction	2.54 (1.03 – 6.27)	0.043
Abnormal LAVI	3.86 (1.98 – 7.52)	<0.001
Anaemia	2.26 (0.97 – 5.28)	0.060
Creatinine (per mg/dL)	1.21 (1.05 – 1.40)	0.009
Pre-dialysis hyperkalemia	1.75 (0.86 – 3.56)	0.123
Hypocalcemia	1.28 (0.64 – 2.54)	0.486
Hyperphosphatemia	1.82 (0.91 – 3.62)	0.090
Dialysis frequency (per session/week)	1.19 (0.76 – 1.87)	0.448

Variable	Multivariate OR (95% CI)	p-value
Age (per year)	1.03 (1.00 – 1.06)	0.048
LV dilatation	2.98 (0.92 – 9.66)	0.068
LVH	3.12 (1.44 – 6.76)	0.004

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Diastolic dysfunction	2.11 (0.97 – 4.60)	0.060
Systolic dysfunction	1.11 (0.39 – 3.18)	0.834
Abnormal LAVI	2.46 (1.15 – 5.27)	0.021
Creatinine (per mg/dL)	1.17 (1.00 – 1.38)	0.046

Clinical Outcomes

The presence of any arrhythmia was significantly associated with adverse clinical outcomes over a three-month follow-up period. Patients with arrhythmias had significantly higher rates of emergency visits (20.6% vs. 8.3%, $p < 0.05$) and hospitalizations (21.7% vs. 4.1%, $p < 0.05$) compared to those without arrhythmias. Most critically, all deaths recorded during the follow-up period ($n=8$, 5.7% of the total cohort) occurred in the group with arrhythmias, resulting in a 3-month mortality rate of 8.6% for this group versus 0% for those without arrhythmias ($p < 0.05$). These outcomes are summarized in Table 4.

Table 4. Comparison of Clinical Outcomes in Patients with and without Arrhythmia

Clinical Outcome	Patients with Arrhythmia (n=92)	Patients without Arrhythmia (n=48)	P value
Emergency Visit	19 (20.6%)	4 (8.3%)	< 0.05
Hospitalization	20 (21.7%)	2 (4.1%)	< 0.05
3 month Mortality	8 (8.6%)	0 (0%)	< 0.05

DISCUSSION

Baseline and Clinical Characteristics

The mean age of 51.1 years in this cohort is notably younger than the typical dialysis populations in many Western countries, where the average age often exceeds 60 years [9]. This suggests that ESRD may be affecting individuals in their prime productive years in this region, a trend that has significant socioeconomic implications. The striking predominance of female patients (70%) is another interesting demographic feature that may warrant further investigation into local referral patterns or gender-specific risk factors for kidney disease progression.

The prevalence of traditional cardiovascular risk factors is exceptionally high. Nearly three-quarters of the patients (72.9%) had hypertension, and almost one-third had co-existing diabetes mellitus (30.7%) or established IHD (30%). This "triple threat" of hypertension, diabetes, and IHD creates a powerful substrate for the development and progression of cardiac damage. According to the United States Renal Data System (USRDS), cardiovascular disease is the leading cause of mortality in ESRD patients, and these comorbidities are its primary drivers [2]. The high prevalence in our cohort immediately classifies them as a high-risk group.

The laboratory data reveal a significant burden of uraemia-related metabolic disturbances, which are independent risk factors for cardiac arrhythmias and SCD. The fact that over 70% of patients were anaemic is a critical finding. Anaemia in ESRD is known to contribute to the development of LVH and high-output cardiac states, further stressing an already compromised myocardium [10]. Moreover, the high prevalence of mineral and bone disease markers, such as hypocalcemia (45%) and hyperphosphatemia (42.1%), is concerning. These electrolyte imbalances are linked to vascular calcification, cardiac stiffness, and are potent triggers for electrical instability in the heart [11].

A key finding of our study is the extensive structural and functional cardiac remodelling present in this cohort. Over 60% of patients exhibited some degree of LVH, and more than 70% had evidence of diastolic dysfunction. This condition, often termed "uremic cardiomyopathy," results from chronic exposure to pressure overload (from hypertension), volume overload (from fluid retention), and direct uremic toxins [4]. LVH is one of the strongest predictors of mortality in the dialysis population. It creates a substrate for re-entrant arrhythmias, while diastolic dysfunction impairs the heart's ability to relax and fill, leading to heart failure symptoms. The finding that over half the patients had an abnormal LAVI is also a significant predictor of future AF, a common and dangerous arrhythmia in this population [5].

In this cohort, a young-to-middle-aged dialysis population is heavily burdened by both traditional and uraemia-specific cardiovascular risk factors. The high prevalence of hypertension, diabetes, anaemia, electrolyte disturbances, and, most critically, advanced structural heart disease (LVH and diastolic dysfunction) provides a clear rationale for the high rates of morbidity and mortality observed. These findings strongly support the need for aggressive risk factor management and heightened clinical vigilance to mitigate the risk of adverse cardiovascular events and SCD in ESRD patients on haemodialysis.

Prevalence of Arrhythmias

The most clinically significant arrhythmia in this study is ventricular arrhythmias. A high percentage of patients exhibited frequent or complex PVCs, and a notable portion experienced episodes of NSVT. This is a critical observation, as NSVT is one of the most powerful independent predictors of SCD in patients with ESRD [12]. The hypertrophied and often fibrotic myocardium, a condition highly prevalent in our cohort (as evidenced by 62.9% LVH), serves as the pathological substrate for these re-entrant ventricular arrhythmias [13]. The electrical instability of this "uremic cardiomyopathy" is then exacerbated by the profound electrolyte shifts (e.g., hyperkalemia) and fluid fluctuations inherent to the intermittent nature of haemodialysis, creating a perfect storm for malignant arrhythmias [4].

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Equally concerning is the burden of supraventricular arrhythmias, particularly AF. The prevalence of AF in our cohort aligns with rates reported in other dialysis studies, which are significantly higher than in the general population [14]. This finding is the logical consequence of the high prevalence of left atrial enlargement (50.7%) observed in our patients, which is a known anatomical precursor to AF [5]. In the ESRD population, AF is not a benign arrhythmia; it is associated with a markedly increased risk of ischemic stroke, heart failure, and all-cause mortality [14,15]. The presence of frequent PACs and episodes of SVT further highlights the profound electrical instability of the atrial myocardium in these patients.

Furthermore, the presence of significant bradyarrhythmias and conduction abnormalities, such as sinus pauses and atrioventricular (AV) block, should not be overlooked. While tachyarrhythmias are often the focus, bradycardic events are also an important contributor to syncope, falls, and SCD in this population [16]. These conduction disorders are likely multifactorial, stemming from fibrosis extending into the heart's conduction system, autonomic dysfunction, and the cardio-depressant effects of severe electrolyte disturbances, particularly hyperkalemia, which was prevalent in our cohort [17].

The timing and frequency of these arrhythmias are often linked to the dialysis cycle itself. Studies have consistently shown a clustering of SCD and arrhythmic events in the 12-hour period immediately preceding a dialysis session (the end of the long interdialytic gap) and during the session itself, periods characterized by the most extreme fluid and electrolyte abnormalities [18]. The high arrhythmia burden seen in our 24-hour recordings provides a direct window into this period of heightened vulnerability.

These findings underscore the inadequacy of symptom-based arrhythmia detection and highlight the potential utility of systematic screening with Holter monitoring, particularly in high-risk subgroups (e.g., patients with severe LVH or prior cardiac events), to guide targeted therapeutic interventions such as beta-blocker therapy, mineralocorticoid receptor antagonism, or adjustments to the dialysis prescription to mitigate the staggering risk of SCD.

Predictors of Arrhythmias

In the univariate analysis, a broad range of factors, including a history of IHD, anaemia, and several echocardiographic parameters like LVH and LAVI, were significantly associated with the presence of complex arrhythmias. This is expected and aligns with our understanding of arrhythmogenesis, where multiple pathological processes contribute to risk. However, the true clinical utility lies in the multivariate analysis, which distils these associations into the most critical independent predictors. Our multivariate model reveals that the presence of moderate-to-severe LVH emerged as the most robust independent predictor of any arrhythmia (OR 3.12, $p=0.004$).

After adjusting for confounding variables, patients with severe LVH remained at a significantly higher risk. This finding is strongly supported by extensive literature. LVH is not merely an increase in heart muscle mass; it is a pathological state of myocardial fibrosis and cellular disarray that creates electrical heterogeneity, slows conduction velocity, and provides the ideal substrate for re-entrant circuits, which are the primary mechanism of ventricular tachycardia and fibrillation. As established by Silberberg and others decades ago, LVH is one of the single most powerful predictors of mortality in the ESRD population, and our analysis confirms its central role as the anatomical foundation for life-threatening arrhythmias [13].

While potassium is known to be a potent arrhythmic trigger, it was not an independent predictor in this specific multivariate model, perhaps because its effects are mediated by or confounded with other factors. Potassium is the principal determinant of the cardiac myocyte's resting membrane potential. Deviations in its extracellular concentration directly alter myocardial excitability and repolarization, predisposing the heart to both brady- and tachyarrhythmias [19]. The intermittent nature of haemodialysis, which creates large fluxes in potassium over the interdialytic period, makes this a particularly dangerous and dynamic risk factor. Our finding reinforces the concept that the uremic heart, already structurally compromised by LVH, is exquisitely sensitive to these electrolyte shifts [19]. Furthermore, when specifically analyzing predictors for supraventricular arrhythmias, LAVI was the dominant independent predictor. This is consistent with the paradigm of atrial remodelling, where chronic volume and pressure overload lead to atrial stretch, fibrosis, and electrical isolation of the pulmonary veins, creating the substrate for the initiation and maintenance of AF [20]. The fact that LAVI remained a predictor independent of LVH suggests that atrial remodelling is a distinct and clinically important process in this population.

It is noteworthy that some variables, such as anaemia, which were significant in the univariate analysis, lost their independent predictive power in the multivariate model. This suggests that their influence is likely mediated through other factors. For example, chronic anaemia is a primary driver for the development of LVH; therefore, LVH stands as the more direct and proximate cause of the arrhythmic event in the final model [10].

Clinical Outcomes

The presence of any significant arrhythmia was strongly associated with an increased risk of cardiovascular hospitalization and emergency department visits. This highlights the substantial morbidity and healthcare resource utilization driven by these electrical disorders. AF, in particular, was a major driver of hospital admissions. This is consistent with the known pathophysiology where AF can precipitate acute decompensated heart failure, especially in patients with pre-existing diastolic dysfunction and LVH,

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conditions that were highly prevalent in our cohort [15]. The rapid ventricular rates and loss of atrial "kick" associated with AF are poorly tolerated in these hemodynamically fragile patients.

The high prevalence of AF in our cohort, coupled with its association with adverse outcomes, emphasizes the urgent need for better risk stratification tools and safer therapeutic strategies to manage stroke risk in this population.

Many of the most dangerous arrhythmias we detected, including episodes of NSVT, were asymptomatic. Awaiting symptoms such as palpitations or syncope before investigating is an inadequate strategy that will fail to identify a large proportion of high-risk individuals. Our data suggest that systematic screening with 24-hour Holter monitoring, particularly in patients with established high-risk features like severe LVH or significant electrolyte abnormalities, could be a valuable tool for risk stratification. Identifying these high-risk patients before they have a clinical event opens a therapeutic window to implement potentially life-saving interventions, including the optimization of dialysis parameters, aggressive medical therapy with beta-blockers, and management of mineral bone disease, all of which may modify the underlying arrhythmic substrate.

STUDY LIMITATIONS

This study has some limitations. First, as a single-centre study, its findings may not be generalizable to all dialysis patients in the region. Second, the cross-sectional design can show associations but cannot prove causality. Finally, the three-month follow-up period is relatively short and may not capture the full spectrum of adverse cardiovascular events.

CONCLUSIONS

In this cohort of patients with ESRD on maintenance hemodialysis in Myanmar, arrhythmias detected by 24-hour Holter monitoring were highly prevalent (65.7%) and significantly associated with adverse short-term clinical outcomes, including mortality. The most powerful independent predictors of arrhythmias were the structural cardiac abnormalities of LVH and an abnormal LAVI. These findings suggest that routine echocardiography followed by targeted Holter monitoring could be a valuable and practical strategy for risk stratification to help mitigate the high rate of sudden cardiac death in this vulnerable population.

CLINICAL PERSPECTIVES

Competency in Medical Knowledge

Patients with ESRD on hemodialysis face an exceedingly high risk of sudden cardiac death, which is largely driven by arrhythmias. While this is well-established in high-income countries, there is a critical lack of data from low- and middle-income countries where the burden of ESRD is growing. This study provides the first systematic, Holter-

based evidence from Myanmar, confirming a high prevalence of arrhythmias (65.7%) in a young hemodialysis population heavily burdened by cardiovascular risk factors. The key finding is that structural cardiac abnormalities—specifically LVH and an abnormal LAVI—are the most powerful independent predictors of arrhythmias, even after accounting for metabolic disturbances like high creatinine levels. This reinforces that the uremic cardiomyopathy substrate is the primary driver of electrical instability.

Translational Outlook

The findings of this study have direct implications for clinical practice, particularly in resource-constrained settings. The identification of echocardiographic parameters like LVH and LAVI as robust predictors suggests that a routine, non-invasive echocardiogram can serve as a powerful initial tool for risk stratification. Patients identified as high-risk based on these structural changes could be prioritized for further investigation with 24-hour Holter monitoring to unmask asymptomatic but malignant arrhythmias. This targeted approach would allow clinicians to implement potentially life-saving interventions—such as optimizing beta-blocker therapy, adjusting the dialysis prescription to mitigate electrolyte shifts, and aggressively managing fluid status—in the patients who stand to benefit most, thereby taking a crucial step toward reducing the staggering rate of SCD in this vulnerable population.

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CONFLICT OF INTEREST

No conflict of interest. No external funding.

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