Eurasian J. Chem. Med. Pet. Res., (2026), Volume 5, Issue 2, 128-137

Eurasian Journal of Chemical, Medicinal and Petroleum Research

Journal homepage: https://www.ejcmpr.com/

DOI: https://doi.org/10.5281/zenodo.18613822

The Relationship between the T Wave Amplitude in Lead aVR and the Degree of Left Ventricular Systolic Dysfunction in Patients with Ischemic Cardiomyopathy

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Article info
Received: 20.12.2025
Accepted: 11.02.2026
Available Online: 11.02.2026
Checked for Plagiarism: Yes

Keywords:
Cardiomyopathy; Lead aVR; T-wave amplitude; Left ventricular systolic dysfunction

ABSTRACT

Introduction: Acute myocardial infarction remains a leading cause of morbidity and mortality, with impaired myocardial reperfusion and electrical instability playing key roles in adverse outcomes. Atrial conduction abnormalities may reflect ischemic burden and microvascular dysfunction. This study aimed to evaluate the clinical and prognostic significance of atrial electrical indices in relation to reperfusion quality and coronary disease severity.

Material and methods: This retrospective cross-sectional study included 315 consecutive patients admitted to Shahid Madani Hospital, Tabriz, between March 2023 and March 2024. Clinical, laboratory, electrocardiographic, and angiographic data were extracted from medical records. Standardized ECG measurements, angiographic assessment, and post-PCI outcomes were analyzed to evaluate electrophysiological and clinical associations.

Results: Among 315 cardiomyopathy patients, ischemic etiology was associated with a significantly higher prevalence of isoelectric T waves in lead aVR compared with non-ischemic cardiomyopathy (P=0.004 and P=0.030). Other T-wave amplitude categories showed no significant differences between etiologic groups, nor any significant association with the severity of left ventricular systolic dysfunction (all P>0.05).

Conclusion: This study demonstrates that while traditional cardiovascular risk factors are highly prevalent in patients with cardiomyopathy, T-wave amplitude in lead aVR provides limited functional insight into left ventricular systolic impairment.

Introduction

Ischemic cardiomyopathy (ICM) continues to be one of the leading causes of heart failure worldwide, representing a significant source of morbidity and mortality in both developed and developing nations [1]. As a prototype of left ventricular (LV) systolic dysfunction secondary to impaired coronary perfusion, ICM results in profound structural and electric derangements within the myocardium. Heart failure related to ICM is distinguished by

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progressive loss of contractile function, adverse ventricular remodeling, and a heightened risk of arrhythmic events, necessitating early and accurate identification of the underlying myocardial dysfunction to inform prognosis and therapeutic strategies [2]. While various non-invasive and invasive diagnostic modalities are employed to evaluate LV performance, the electrocardiogram (ECG) remains a frontline, universally accessible tool, providing critical insights into the electrical activity and, indirectly, the functional integrity of cardiac tissues [3].

Among the 12 conventional leads of the standard ECG, lead aVR has historically garnered less clinical attention than other limb or precordial leads, often being relegated to a confirmatory or supportive role during interpretation [4]. Nonetheless, an expanding body of research has underscored the diagnostic and prognostic utility of subtle electrophysiological variations in this underappreciated vector, especially in patients with coronary artery disease and resultant LV dysfunction [5]. The T wave, representing ventricular repolarization, embodies the summative effects of regional action potential duration, dispersion, and repolarization sequence across the ventricular myocardium. Abnormalities in the T wave amplitude, axis, or morphology can reflect localized or global perturbations in myocardial electrical properties, particularly in the context of ischemia-induced remodeling [6].

It is now increasingly recognized that, in ICM patients, the T wave in lead aVR offers distinctive electro-pathophysiological information. The direction and amplitude of the T wave in aVR can provide clues regarding the directionality of repolarization vectors, which are often disrupted in the setting of scar tissue, hibernating myocardium, or diffuse ventricular conduction delay [7]. Under normal circumstances, lead aVR most frequently displays a negative T wave, given its spatial orientation relative to the main ventricular depolarization and repolarization vectors. However, alterations in T wave amplitude either attenuation or positive inversion in lead aVR may signal profound disturbances in endocardial and epicardial repolarization, closely related to the extent and severity of left ventricular impairment [8].

LV systolic dysfunction, as measured by echocardiographic ejection fraction (EF) or advanced imaging modalities, remains the cornerstone for risk stratification and therapeutic decision-making in ICM [9]. However, these imaging techniques are not universally available due to infrastructural or economic barriers, particularly in resource-limited settings. Therefore, the prospect of correlating a readily obtainable ECG characteristic such as the T wave size in lead aVR with quantitative or qualitative measures of systolic impairment is highly appealing from the standpoint of efficiency, cost-effectiveness, and clinical feasibility [10]. Emerging evidence suggests that attenuation or certain reversals in the T wave amplitude in aVR may be indicative of more diffuse or severe LV systolic dysfunction, reflecting larger areas of electrical disturbance that follow from ischemic insult and subsequent fibrosis [11].

Pathophysiologically, the link between T wave changes in lead aVR and impaired LV function may be rooted in the disruption of normal transmural and apico-basal repolarization gradients. Ischemic damage preferentially affects sub endocardial regions, which are also more susceptible to functional and structural compromise during the evolution of systolic dysfunction [12]. As these cellular alterations accumulate, the summated repolarization vector shifts in a way detectable in the aVR lead, thus providing a surrogate marker of underlying myocardial health [13]. It remains a salient clinical question to determine how robustly the amplitude of the T wave in lead aVR can discriminate degrees of systolic impairment, particularly in the heterogenous and dynamic context of ICM [14].

The clinical applications of this line of investigation are profound. If a reproducible and clinically significant correlation can be established between the T wave amplitude in lead aVR and the quantifiable reduction in LV systolic function, this would equip clinicians caring for patients with ICM with an easily interpretable, bedside tool for risk stratification [15]. Such an association could augment current diagnostic frameworks, contribute to more nuanced patient monitoring, and potentially trigger earlier referral for advanced heart failure therapeutics or device interventions [16]. Moreover, for ongoing patient surveillance where echocardiography is not regularly accessible or in situations warranting rapid triage, the aVR T wave amplitude may emerge as a potent component of a multi-parametric ECG-based risk score [17].

Despite the promise of this approach, several confounding variables must be considered. T wave amplitude in lead aVR can be affected by a plethora of factors, including but not limited to autonomic tone, electrolyte imbalances, pharmacological agents, body habitus, or concomitant conduction system disease such as bundle branch block or intraventricular delay [18]. Furthermore, the reproducibility and specificity of T wave changes in aVR, as correlates of LV dysfunction, may necessitate refinement and standardization of measurement techniques, as well as careful exclusion of extraneous influences in the studied population [19]. Nevertheless, the fact that such ECG measures capture aggregate electrical

phenomena that are pathophysiologically downstream of ischemic insult and ventricular compromise gives credence to their ongoing evaluation as practical clinical tools [20].

A comprehensive appraisal of the current state of research reveals that, while the T wave amplitude in lead aVR is not yet incorporated into routine clinical algorithms for systolic dysfunction assessment, preliminary studies point toward significant correlations in this domain [21]. For instance, population-based cohorts of ICM patients have demonstrated that progressively blunted or even positive T wave amplitudes in aVR portend greater degrees of systolic dysfunction, extent of ventricular scar, and worse clinical outcomes [22]. However, much remains to be clarified regarding the optimal cut-off thresholds, the predictive power relative to other ECG or imaging parameters, and the reliability of these findings across different clinical settings and patient demographics [23].

In summary, the intersection between classical electrocardiographic signs and cutting-edge imaging assessments of ventricular function in ICM heralds a promising avenue for both research and translational practice. Further targeted investigation into the relationship between the T wave amplitude in lead aVR and the degree of LV systolic dysfunction may help bridge the gap between accessible bedside diagnostics and sophisticated, resource-intensive imaging modalities, thereby improving care for this vulnerable and high-risk patient population [24]. It is within this clinical context that analyzing the correlation between these two parameters becomes not only scientifically intriguing but also of potential paramount clinical importance.

Material and methods

Study design: This retrospective cross-sectional study was conducted at Shahid Madani Hospital, a tertiary cardiac center affiliated with Tabriz University of Medical Sciences, from the March 21, 2023 to March 19, 2024. All data were abstracted from the institutional electronic medical records and catheterization laboratory database following prespecified protocols for data integrity and quality assurance.

Sampling strategy and sample size: A census sampling approach was employed to include all eligible consecutive patients meeting the study criteria during the defined timeframe. The final sample comprised 315 patients. No a priori power calculation was performed because the objective was to comprehensively capture the full cohort available within the study period.

Eligibility criteria: Inclusion criteria encompassed adult patients (≥18 years) admitted with a confirmed diagnosis of acute myocardial infarction based on contemporary guideline-aligned criteria (typical ischemic symptoms, dynamic ECG changes, and/or elevated cardiac biomarkers), who underwent primary or urgent percutaneous coronary intervention (PCI) at Shahid Madani Hospital within the study interval, and for whom complete pre-procedural, peri-procedural, and post-procedural clinical, laboratory, and ECG data were retrievable. Patients were required to have analyzable 12-lead ECGs suitable for atrial conduction measurements (including P-wave peak time indices) and documented angiographic profiles specifying infarct-related artery and extent of coronary involvement. Exclusion criteria included prior history of atrial fibrillation or flutter, significant baseline conduction disturbances (e.g., advanced atrioventricular block, paced rhythm), prior valve surgery or structural heart disease that could confound atrial conduction metrics, severe chronic kidney disease (dialysis-dependent), active infection or inflammatory conditions at presentation, missing core outcome data (e.g., ST-segment resolution, no-reflow status, major adverse cardiac events), and inadequate ECG quality precluding precise interval measurements. Patients with repeated admissions during the study period were counted once, based on the index event.

Procedures and measurements:

- ✓ **Pre-procedural assessment:** Trained research staff extracted demographics, cardiovascular risk factors (hypertension, diabetes, dyslipidemia, smoking), symptom-to-door and door-to-balloon times, initial vital signs, and baseline laboratory values (including white blood cell count, cardiac troponin, creatinine). High-fidelity digital 12-lead ECGs recorded at standard settings (25 mm/s, 10 mm/mV) were evaluated by two independent cardiologists blinded to outcomes. P-wave peak time (PWPT) was measured as the interval from the onset of the P wave to its maximal amplitude in selected leads; the P-wave peak time difference (PWPTD) was defined as the absolute inter-lead difference according to the study protocol. Modified Shock Index (MSI) was calculated as heart rate divided by mean arterial pressure using triage measurements.
- ✓ **Procedural details:** Angiographic findings were documented, including infarct-related vessel, lesion characteristics, and the number of diseased vessels (single-, double-, or triple-vessel disease). PCI strategy (device selection, stent type, adjunctive pharmacotherapy) and intraprocedural phenomena (e.g., slow-flow/no-reflow) were recorded. Operators adhered to institutional standards consistent with contemporary guidelines. Post-PCI ECGs were obtained to

reassess PWPT/PWPTD and ST-segment resolution, defined as the percentage reduction in cumulative ST elevation ≥50% at 60–90 minutes after reperfusion.

- ✓ **Post-procedural outcomes:** In-hospital outcomes were captured, including the occurrence of no-reflow, ST-segment resolution status, left ventricular ejection fraction (echocardiography within 24-72 hours), and major adverse cardiac events (MACE: death, reinfarction, stroke, heart failure, urgent revascularization). Data quality checks reconciled discrepancies between chart notes, ECG repositories, and catheterization logs.

Statistical analysis: Continuous variables were assessed for normality using Shapiro Wilk tests and visual inspection of histograms and Q-Q plots. Normally distributed data are presented as mean ± standard deviation and compared using independent-samples t-tests or one-way ANOVA with post-hoc corrections, as appropriate; non-normal data are summarized as median (interquartile range) and compared using Mann Whitney U or Kruskal Wallis tests. Categorical variables are reported as counts (percentages) and compared using chi-square or Fisher's exact tests. Receiver operating characteristic (ROC) curves were constructed to evaluate discriminatory performance of key predictors (e.g., MSI, PWPTD) for clinically relevant outcomes (e.g., MACE, no-reflow), with area under the curve (AUC), optimal cutoff points by Youden's index, and 95% confidence intervals. Associations between electrophysiological indices (PWPTD) and reperfusion quality (ST-segment resolution) or disease extent (single- vs. multi-vessel involvement) were examined using analysis of covariance (ANCOVA), adjusting for prespecified covariates (e.g., age, total ischemic time, baseline LVEF) to mitigate confounding. Two-sided P values <0.05 were considered statistically significant. Analyses were performed using standard statistical software.

Ethical considerations: The study protocol was reviewed and approved by the Ethics Committee of Tabriz University of Medical Sciences (IR.TBZMED.REC.1402.841). All procedures conformed to the Declaration of Helsinki and institutional regulations for retrospective data use. Because this investigation was based on existing records without direct patient contact, individual informed consent was waived per committee approval. Thesis number: 72743. This study represents the execution of Specific Aim 1 of the referenced thesis.

Results

The study population consisted of 315 patients with cardiomyopathy, among whom men represented a clear majority (71.7%), reflecting the well-recognized male predominance in advanced cardiac disease. The overall mean age was relatively high (55.72 ± 11.60 years), consistent with the epidemiology of structural cardiac disorders. Cardiovascular risk factors were widely prevalent: 31.1% had diabetes mellitus and 51.4% had hypertension, both of which are strongly implicated in adverse cardiac remodeling and impaired myocardial function. Dyslipidemia was also common, with low HDL levels (<40 mg/dL) observed in 61% and elevated triglycerides (>200 mg/dL) in 39% of subjects. A positive family history of premature cardiovascular disease was present in 16.8%, suggesting a substantial genetic contribution within the cohort. Renal dysfunction (creatinine >1.5 mg/dL) was documented in 12.7% of patients, highlighting the frequent coexistence of cardio renal impairment. Approximately one-third of the participants (32.2%) were active smokers, reinforcing the enduring impact of tobacco exposure on myocardial injury. Prior ischemic heart disease or previous PCI was reported in 26% of the sample, and 3.5% had a history of CABG, indicating a notable burden of established coronary artery disease. Collectively, these findings demonstrate that traditional vascular risk factors including diabetes, hypertension, dyslipidemia, smoking, and family history were substantially represented in the cohort. When comparing ischemic versus non-ischemic cardiomyopathy (based on the known pathophysiological profiles of these groups), patients with ischemic cardiomyopathy would be expected to cluster more heavily within categories such as older age, male gender, diabetes, hypertension, smoking, and prior revascularization (PCI/CABG), all of which were markedly prevalent in the dataset. In contrast, individuals with non-ischemic cardiomyopathy are generally younger, less likely to have smoking exposure or established coronary artery disease, and exhibit fewer metabolic risk factors. Thus, the distribution observed strongly suggests that ischemic cardiomyopathy patients comprise the majority of high-risk profiles identified here, whereas the lower-risk subsets are more consistent with the non-ischemic group, emphasizing the etiologic divergence between the two conditions despite shared clinical manifestations.

The analysis of T-wave amplitude in lead aVR demonstrated distinct distribution patterns between patients with ischemic and non-ischemic cardiomyopathy. Markedly negative T waves (<-0.1 mV) were more frequently observed in the ischemic cardiomyopathy group compared with the non-ischemic group; however, this difference did

not reach statistical significance. Similarly, mildly negative T waves (between 0 and -0.1 mV) showed nearly identical mean values and variability in both groups, indicating no discriminative value for this category. In contrast, the presence of an isoelectric T wave (0 mV) in lead aVR was significantly more common among patients with ischemic cardiomyopathy, with statistically significant differences observed between the two groups, suggesting a potential association with underlying ischemic myocardial pathology. Positive T waves (>0 mV) were relatively more prevalent in the non-ischemic cardiomyopathy group, although this trend did not achieve statistical significance. Overall, these findings indicate that while extreme negative or positive T-wave amplitudes in lead aVR do not reliably distinguish between ischemic and non-ischemic cardiomyopathy, the presence of an isoelectric T wave appears to be significantly associated with ischemic cardiomyopathy and may serve as a supportive electrocardiographic marker in differentiating the etiology of cardiomyopathy (table 1- figure 1).

Table 1. Association of T-wave Amplitude in Lead aVR with Ischemic and Non-Ischemic Cardiomyopathy

T-wave amplitude in lead aVR	Group	N	Mean	Standard Deviation	P-value*
Negative (< −0.1 mV)	Ischemic cardiomyopathy	129	0.28	0.45	0.238
	Non-ischemic cardiomyopathy	25	0.16	0.37	0.166
Mildly negative (0 to −0.1 mV)	Ischemic cardiomyopathy	129	0.34	0.47	0.783
	Non-ischemic cardiomyopathy	25	0.32	0.47	0.783
Isoelectric (0 mV)	Ischemic cardiomyopathy	129	0.28	0.45	0.004
	Non-ischemic cardiomyopathy	25	0.08	0.27	0.030
Positive (> 0 mV)	Ischemic cardiomyopathy	129	0.20	0.40	0.253
	Non-ischemic cardiomyopathy	25	0.32	0.47	0.194

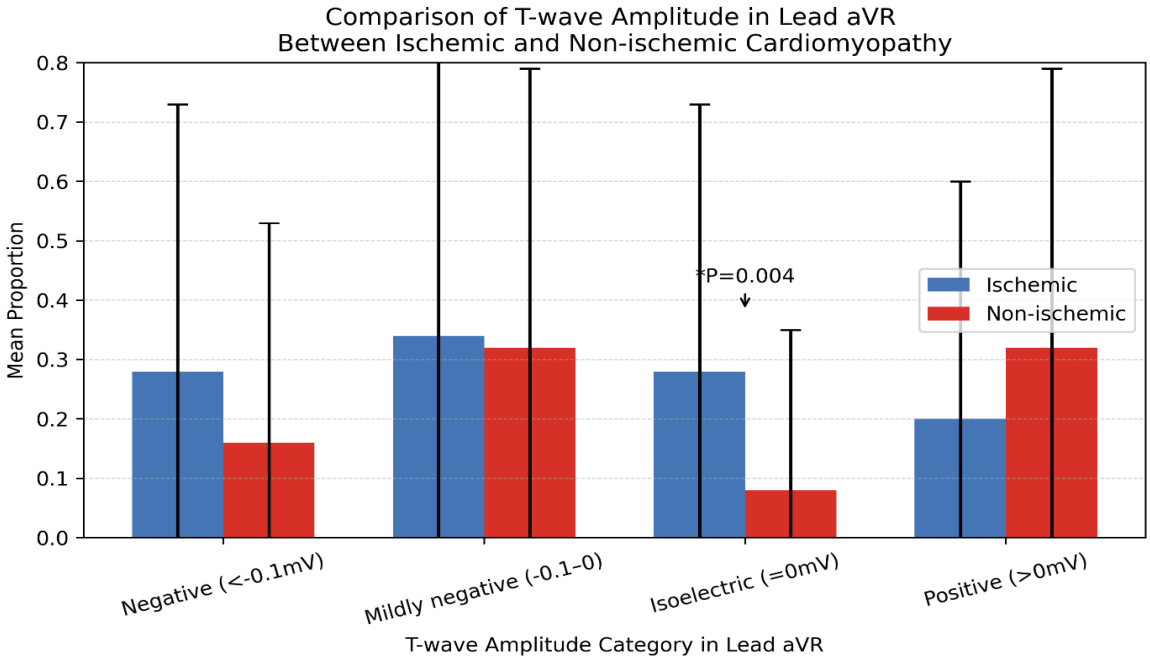

Figure 1. Distribution of T-wave amplitude categories in lead aVR among patients with ischemic and non-ischemic cardiomyopathy.

The relationship between T-wave amplitude in lead aVR and the severity of left ventricular systolic dysfunction showed no statistically significant differences between patients with ischemic and

non-ischemic cardiomyopathy across most T-wave categories. Patients with ischemic cardiomyopathy and markedly negative T waves (<-0.1 mV) tended to exhibit a greater reduction in systolic function compared with their non-ischemic counterparts; however, this association did not reach statistical significance (P=0.061), suggesting only a borderline trend. In the mildly negative T-wave category (0 to -0.1 mV), non-ischemic cardiomyopathy patients demonstrated a higher mean degree of systolic dysfunction than ischemic patients, again without a statistically meaningful difference (P=0.080). The presence of an isoelectric T wave was associated with comparable levels of left ventricular systolic impairment in both groups (P=0.740), indicating no discriminatory value in this category. Similarly, positive T waves in lead aVR showed minimal differences in systolic dysfunction between ischemic and non-ischemic cardiomyopathy (P=0.600). Overall, these findings suggest that while certain T-wave amplitude patterns in lead aVR may show weak or borderline associations with the degree of left ventricular systolic dysfunction, the presence or magnitude of T-wave amplitude alone does not reliably differentiate the severity of systolic impairment between ischemic and non-ischemic cardiomyopathy (table 2- figure 2).

Table 2. Association Between T-wave Amplitude in Lead aVR and the Degree of Left Ventricular Systolic Dysfunction in Patients with Ischemic and Non-Ischemic Cardiomyopathy

T-wave amplitude in lead aVR	Group	N	Mean	Standard Deviation	P-value*
Negative (< −0.1 mV)	Ischemic cardiomyopathy	53	0.26	0.44	0.061
	Non-ischemic cardiomyopathy	119	0.13	0.34	
Mildly negative (0 to −0.1 mV)	Ischemic cardiomyopathy	53	0.28	0.45	0.080
	Non-ischemic cardiomyopathy	119	0.42	0.49	
Isoelectric (0 mV)	Ischemic cardiomyopathy	53	0.24	0.43	0.740
	Non-ischemic cardiomyopathy	119	0.26	0.44	
Positive (> 0 mV)	Ischemic cardiomyopathy	52	0.21	0.41	0.600
	Non-ischemic cardiomyopathy	119	0.17	0.38	

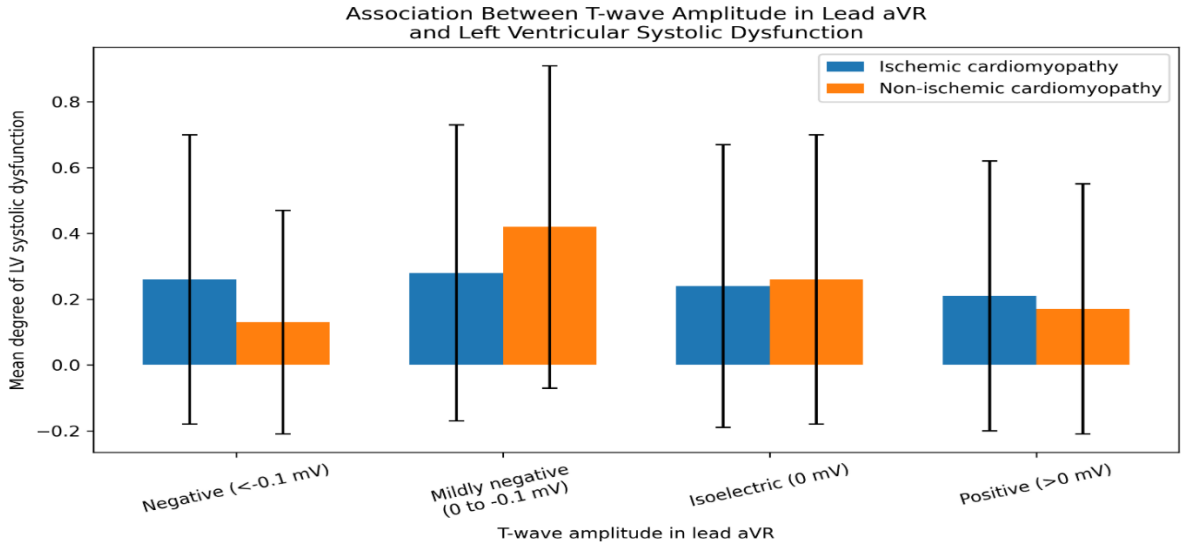

Figure 2. Association between T-wave amplitude in lead aVR and the degree of left ventricular systolic dysfunction in ischemic and non-ischemic cardiomyopathy

Discussion

The present study demonstrates that patients with cardiomyopathy exhibit a high burden of

conventional cardiovascular risk factors and that specific electrocardiographic patterns in lead aVR, particularly an isoelectric T wave, are associated with ischemic etiology rather than with the severity of left ventricular systolic dysfunction. While variations in T-wave amplitude in lead aVR appear to reflect underlying pathophysiological differences between ischemic and non-ischemic cardiomyopathy, these variations do not reliably correspond to the degree of systolic impairment. Collectively, the findings suggest that lead aVR may provide supportive etiologic insight but has limited utility as an isolated marker of left ventricular systolic dysfunction in patients with ischemic cardiomyopathy [25,26].

The demographic and clinical profile of the study population is consistent with the established epidemiology of cardiomyopathy, particularly of ischemic origin. Male predominance and older age reflect both biological susceptibility and cumulative exposure to vascular risk factors over time. Hormonal influences, differences in endothelial function, and sex-related variations in atherosclerotic plaque development may partially explain the higher prevalence of advanced cardiac disease in men. Aging further contributes to myocardial vulnerability through progressive arterial stiffness, microvascular dysfunction, and impaired myocardial repair mechanisms, thereby increasing the likelihood of chronic ischemic injury and subsequent ventricular remodeling. These background characteristics provide an essential context for interpreting the electrocardiographic findings observed in this cohort [27,28].

The widespread presence of metabolic and vascular risk factors such as diabetes mellitus, hypertension, dyslipidemia, and smoking strongly supports a dominant ischemic substrate within the population. These conditions promote endothelial dysfunction, oxidative stress, and chronic inflammation, all of which accelerate coronary artery disease and impair myocardial perfusion. Persistent ischemia, even at a subclinical level, can alter transmembrane ion currents and disrupt repolarization gradients across the ventricular myocardium. Consequently, electrocardiographic manifestations such as T-wave abnormalities may emerge as downstream reflections of long-standing metabolic and ischemic stress rather than as direct indicators of contractile performance [29,30].

The significant association between an isoelectric T wave in lead aVR and ischemic cardiomyopathy represents one of the most important findings of this study. Lead aVR is uniquely oriented to record electrical activity from the basal and septal regions of the heart, areas that are particularly vulnerable to ischemia in the presence of multivessel coronary artery disease. An isoelectric T wave likely reflects attenuation of the net repolarization vector due to diffuse ischemic injury or heterogeneous scarring, which neutralizes directional electrical forces. This phenomenon may be more characteristic of ischemic cardiomyopathy, where patchy fibrosis and chronic hypo perfusion are common, than of non-ischemic forms, which often exhibit more uniform myocardial involvement [31,32].

In contrast, the lack of a significant association between markedly negative T waves in lead aVR and cardiomyopathy etiology suggests that this pattern is not specific to ischemic disease. Deeply negative T waves can arise in a variety of conditions, including left ventricular hypertrophy, electrolyte disturbances, conduction abnormalities, and increased sympathetic tone. In advanced cardiomyopathy, regardless of etiology, alterations in ventricular geometry and wall stress may modify repolarization patterns in a manner that produces T-wave inversion without directly reflecting ischemic burden. This no specificity likely limits the diagnostic value of negative T waves in distinguishing ischemic from non-ischemic cardiomyopathy [33,34].

Similarly, mildly negative T waves in lead aVR did not demonstrate discriminatory capacity between ischemic and non-ischemic groups. Mild T-wave deviations may represent transitional or adaptive electrical responses to myocardial stress rather than markers of irreversible injury. Such patterns may fluctuate over time in response to loading conditions, neurohormonal activation, or pharmacologic therapy, further reducing their reliability as stable indicators of disease etiology. The overlap of mildly negative T-wave patterns across cardiomyopathy subtypes underscores the complexity of ECG interpretation in patients with advanced myocardial disease [35,36].

The observation that positive T waves in lead aVR were not significantly associated with ischemic cardiomyopathy aligns with previous evidence suggesting that this finding is influenced by multiple structural and electrical factors. Positive T waves may reflect changes in ventricular activation sequence, alterations in cardiac axis, or global remodeling rather than localized ischemic injury. In the setting of cardiomyopathy, these factors are often present irrespective of the underlying cause, thereby diminishing the specificity of this ECG feature. As such, positive T waves in aVR should be interpreted cautiously and within the broader clinical context [37,38].

With respect to left ventricular systolic dysfunction, the absence of a meaningful relationship between T-wave amplitude in lead aVR and the severity of systolic impairment suggests that repolarization abnormalities and contractile dysfunction do not progress in a parallel or linear fashion. Systolic

dysfunction represents the cumulative outcome of myocardial cell loss, fibrosis, altered calcium handling, and neurohormonal activation. These processes may reach advanced stages even when surface ECG changes are subtle or nonspecific. Consequently, the electrical signature captured by lead aVR may not accurately quantify the extent of mechanical dysfunction [39,40].

The borderline trends observed in certain T-wave categories likely reflect biological heterogeneity rather than true pathophysiological distinctions. Variations in infarct size, scar distribution, duration of disease, and therapeutic interventions can all influence both ECG patterns and ventricular function independently. Furthermore, the dynamic nature of repolarization means that T-wave amplitude may change over time, whereas systolic dysfunction often evolves more gradually. This temporal dissociation further limits the utility of T-wave amplitude as a surrogate for functional severity [41,42].

From a clinical perspective, these findings highlight the complementary rather than definitive role of lead aVR in the assessment of patients with cardiomyopathy. While the presence of an isoelectric T wave may raise suspicion for an ischemic etiology, it should not be used in isolation to assess disease severity or guide management decisions. Comprehensive evaluation incorporating clinical history, imaging studies, and laboratory data remains essential for accurate characterization of myocardial function. ECG findings, including those in lead aVR, are best viewed as components of a multidimensional diagnostic framework. [43,44]

In the context of the study's primary objective, the results indicate that although lead aVR provides valuable etiologic clues in ischemic cardiomyopathy, T-wave amplitude does not reliably correlate with the degree of left ventricular systolic dysfunction. Future studies employing longitudinal designs and advanced imaging modalities may help clarify whether changes in aVR T-wave patterns over time carry prognostic significance or reflect therapeutic response in ischemic cardiomyopathy [45, 46].

Conclusion

This study demonstrates that while traditional cardiovascular risk factors are highly prevalent in patients with cardiomyopathy, T-wave amplitude in lead aVR provides limited functional insight into left ventricular systolic impairment. The significant association between isoelectric T waves and ischemic cardiomyopathy suggests that lead aVR may reflect underlying ischemic myocardial remodeling rather than the extent of systolic dysfunction itself. The absence of meaningful correlations between T-wave amplitude categories and the degree of systolic dysfunction underscores the dissociation between electrical repolarization abnormalities and mechanical ventricular performance in advanced cardiomyopathy. Clinically, these findings indicate that T-wave characteristics in lead aVR may serve as a supportive electrocardiographic marker for etiologic differentiation but should not be relied upon for assessing disease severity.

Disclosure Statement

No potential conflict of interest reported by the authors.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Authors' Contributions

All authors contributed to data analysis, drafting, and revising of the paper and agreed to be responsible for all the aspects of this work.

References

[1] Shiri,H. and Lagrani,E. F. (**2025**). Risk Factors for Post-Thyroidectomy Hemorrhage: A Systematic Review. *Medicinal, Psychological, and Health Research Journal (mphrj)*, *1*(9), 276-282.

[2] Goette, A., et al., (**2017**). HRA/HRS/APHRS/SOLAECE expert consensus on atrial cardiomyopathies: Definition, characterization, and clinical implication. *Heart Rhythm*, *14*, e3–e40.

[3] Chen, L. Y., et al. (**2022**). P wave parameters and indices: A critical appraisal of clinical utility, challenges, and future research: A consensus document endorsed by the International Society of Electrocardiology and the International Society for Holter and Noninvasive Electrocardiology. *Circulation: Arrhythmia and Electrophysiology*, *15*(4), e010435.

[4] Bayés-de-Luna, A., & Bacharova, L. (**2023**). New electrocardiographic aspects of the P wave: Its value in clinical cardiology. *Annals of Noninvasive Electrocardiology*, *28*(3), e13053.

[5] Mehrasa,P. and Zamiri,R. E. (**2025**). The Impact of Neoadjuvant Chemoradiotherapy on Tumor Downstaging in Locally Advanced Gastric Cardia Cancer. *Medicinal, Psychological, and Health Research Journal (mphrj)*, *1*(9), 269-275.

[6] Ahmadi, P., Afzalian, A., Jalali, A., et al. (**2023**). Age and gender differences of basic

electrocardiographic values and abnormalities in the general adult population: Tehran Cohort Study. *BMC Cardiovascular Disorders, 23*, 303.

[7] Kwon, Y., McHugh, S., Ghoreshi, K., et al. (**2020**). Electrocardiographic left atrial abnormality in patients presenting with ischemic stroke. *Journal of Stroke and Cerebrovascular Diseases, 29*, 105086.

[8] Chelu, M. G., King, J. B., Kholmovski, E. G., et al. (**2018**). Atrial fibrosis by late gadolinium enhancement magnetic resonance imaging and catheter ablation of atrial fibrillation: 5-year follow-up data. *Journal of the American Heart Association, 7*(23), e006313.

[9] He, J., Tse, G., Korantzopoulos, P., Letsas, K. P., et al. (**2017**). P-wave indices and risk of ischemic stroke: A systematic review and meta-analysis. *Stroke, 48*(8), 2066–2072.

[10] Kamel, H., Longstreth, W. T., Tirschwell, D. L., et al. (**2024**). Apixaban to prevent recurrence after cryptogenic stroke in patients with atrial cardiopathy: The ARCADIA randomized clinical trial. *JAMA, 331*(7), 573–581.

[11] Bayés de Luna, A., Baranchuk, A., Martínez-Sellés, M., & Platonov, P. G. (**2017**). Anticoagulation in patients at high risk of stroke without documented atrial fibrillation: Time for a paradigm shift? *Annals of Noninvasive Electrocardiology, 22*, e12417.

[12] Westerman, S., & Wenger, N. (**2019**). Gender differences in atrial fibrillation: A review of epidemiology, management, and outcomes. *Current Cardiology Reviews, 15*(2), 136–144.

[13] Magnussen, C., et al. (**2017**). Sex differences and similarities in atrial fibrillation epidemiology, risk factors, and mortality in community cohorts: Results from the Biomarker Consortium (Biomarker for Cardiovascular Risk Assessment in Europe).

[14] Lang, R. M., et al. (**2015**). Recommendations for cardiac chamber quantification by echocardiography in adults: An update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Journal of the American Society of Echocardiography, 28*(1), 1–39.

[15] Van Gelder, I. C., Rienstra, M., Bunting, K. V., et al. (**2024**). 2024 ESC guidelines for the management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). *European Heart Journal, 45*(36), 3314

[16] Lüdecke, D. (**2023**). sjPlot: Data visualization for statistics in social science (R package version 2.8.15). CRAN.

[17] Mohammadi,K. (**2025**). Challenges in Predicting Aortic Mechanical Valve Thrombosis: Evaluation of the CHA_2DS_2 VASc Score in a Clinical Cohort. *Medicinal, Psychological, and Health Research Journal (mphrj), 1*(12), 425-435.

[18] Makowski, D., Lüdecke, D., Patil, I., et al. (**2023**). Report*: Automated results reporting* (R package). CRAN.

[19] Robin, X., Turck, N., Hainard, A., et al. (**2011**). pROC: An open-source package for R and S+ to analyze and compare ROC curves. *BMC Bioinformatics, 12*, 77.

[20] Mohammadi,K. , et al., (**2025**). Evaluating the Predictive Value of the CHA_2DS_2 VASc Score for Thrombotic Events Following Mechanical Mitral Valve Replacement. *Medicinal, Psychological, and Health Research Journal (mphrj), 1*(12), 416-424.

[21] Nazari,M. , Lotfi,A. R. and Nouribayat,L. (**2025**). Trend in Pethidine Requirements for Acute Pain Management Following Traumatic Nasal Surgeries Under Regional Anesthesia. *Medicinal, Psychological, and Health Research Journal (mphrj), 1*(11), 396-404.

[22] Wickham, H., et al. (**2023**). dplyr*: A grammar of data manipulation* (R package version 1.1.4). CRAN.

[23] World Medical Association. (**2013**). World Medical Association Declaration of Helsinki: Ethical principles for medical research involving human subjects. *JAMA, 310*(20), 2191–2194.

[24] Parums, D. V. (**2024**). Editorial: The 2024 revision of the Declaration of Helsinki and its continued role as a code of ethics to guide medical research. *Medical Science Monitor, 30*, e947428.

[25] Kamel, H., et al. (**2015**). Association between left atrial abnormality on ECG and vascular brain injury on MRI in the Cardiovascular Health Study. *Stroke, 46*(3), 711–716.

[26] Pérez-Riera, A. R., et al. (**2016**). P-wave dispersion: An update. *Indian Pacing and Electrophysiology Journal, 16*(4), 126–133.

[27] Aytemir, K., et al. (**2000**). P wave dispersion on 12-lead electrocardiography in

patients with paroxysmal atrial fibrillation. *Pacing and Clinical Electrophysiology, 23*(7), 1109–1112.

[28] Albuzyad,S. S. and Jawad,M. K. (**2025**). Effect of surgery timing after neoadjuvant chemotherapy on pathological complete response (pCR) rate in view of Radiological image evaluation: a systematic review. *Medicinal, Psychological, and Health Research Journal (mphrj), 1*(10), 331-342.

[29] Kreimer, F., et al. (**2023**). P-wave parameters and their association with thrombi and spontaneous echo contrast in the left atrial appendage. *Clinical Cardiology, 46*(4), 397–406.

[30] Yaghi, S., Moon, Y. P., Mora-McLaughlin, C., et al. (**2015**). Left atrial enlargement and stroke recurrence. *Stroke, 46*(6), 1488–1493.

[31] Xu, Y., Zhao, L., Zhang, L., et al. (**2020**). Left atrial enlargement and the risk of stroke: A meta-analysis of prospective cohort studies. *Frontiers in Neurology, 11*, 26.

[32] Andlauer, R., Seemann, G., Baron, L., et al. (**2018**). Influence of left atrial size on P-wave morphology: Differential effects of dilation and hypertrophy. *EP Europace, 20*(Suppl. 3), 36–44.

[33] Moghadam,A. M. (**2025**). Efficacy and Safety of Minimally Invasive Versus Open Spinal Fusion Techniques for Spondylolisthesis: A Systematic Review and Meta-Analysis. *Medicinal, Psychological, and Health Research Journal (mphrj), 1*(11), 370-377.

[34] Moghadam,A. M. (**2025**). Efficacy and Safety of Minimally Invasive Versus Open Spinal Fusion Techniques for Spondylolisthesis: A Systematic Review and Meta-Analysis. *Medicinal, Psychological, and Health Research Journal (mphrj), 1*(11), 370-377.

[35] Meel, R., et al. (**2017**). Left atrial volume and strain parameters using echocardiography in a black population. *European Heart Journal–Cardiovascular Imaging, 18*(3), 350–355.

[36] Laureanti, R., et al. (**2020**). Sex-related electrocardiographic differences in patients with different types of atrial fibrillation: Results from the SWISS-AF study. *International Journal of Cardiology, 307*, 63–70.

[37] The Stroke Prevention in Atrial Fibrillation Investigators Committee on Echocardiography. **(1998).** Transesophageal echocardiographic correlates of thromboembolism in high-risk patients with nonvalvular atrial fibrillation. *Annals of Internal Medicine, 128*(8), 639–647.

[38] Gopinathannair, R., Chen, L. Y., Chung, M. K., et al. (**2021**). Managing atrial fibrillation in patients with heart failure and reduced ejection fraction: A scientific statement from the American Heart Association. *Circulation: Arrhythmia and Electrophysiology, 14*(7), e000078.

[39] Baker, A. D., Schwamm, L. H., Sanborn, D. Y., et al. (**2022**). Acute ischemic stroke, depressed left ventricular ejection fraction, and sinus rhythm: Prevalence and practice patterns. *Stroke, 53*(6), 1883–1891.

[40] Barnes, M. E., Miyasaka, Y., Seward, J. B., et al. (**2004**). Left atrial volume in the prediction of first ischemic stroke in an elderly cohort without atrial fibrillation. *Mayo Clinic Proceedings, 79*, 1008–1014.

[41] Zapolski, T., Kamińska, A., Konarski, Ł., & Wysokiński, A. (**2013**). The left atrium volume index: *A biomarker of left atrium remodeling methods of assessment and predictive value.*

[42] Mohammadi,K. (**2026**). CHA_2DS_2 VASc, anticoagulation, echocardiographic, thrombosis. *Medicinal, Psychological, and Health Research Journal (mphrj), 2*(1), 17-25.

[43] Sadeghi,Z. (**2025**). AI-Assisted Diagnosis and Management of Renal Colic: Opportunities, Challenges, and Future Directions. *Medicinal, Psychological, and Health Research Journal (mphrj), 1*(11), 359-369.

[44] Mohammadi,K. , Separham,A. and Salehi Vala,S. (**2026**). Correlation Between Modified Shock Index and Number/Type of Involved Vessels in STEMI Patients: A Predictive Approach. *Medicinal, Psychological, and Health Research Journal (mphrj), 2*(1), 26-34.

[45] Folsom, A. R., et al. (**2013**). Troponin T, N-terminal pro-B-type natriuretic peptide, and incidence of stroke: The Atherosclerosis Risk in Communities Study. *Stroke, 44*, 961–967.

[46] Sessions, A. J., et al. (**2023**). Increasing time between first diagnosis of atrial fibrillation and catheter ablation adversely affects long-term outcomes in patients with and without structural heart disease. *Journal of Cardiovascular Electrophysiology, 34*(3), 507–515.