*J. Adv. Med. Pharm. Biomed. Res.;* (2026), Volume 2, Issue 1, 83-91

**Journal of Advanced in Medicinal, Pharmaceutical and Biomedical Research**

**Journal homepage:** https://www.jampbr.com

DOI: 10.22034/jampbr.2026.572723.1069

# The Diagnostic Role of the T Wave in Lead aVR in Differentiating Ischemic from Non-Ischemic Cardiomyopathy

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**Article info**
**Received:** 29.12.2025
**Accepted:** 04.02.2026
**Available Online:** 04.02.2026
**Checked for Plagiarism:** Yes

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

**ABSTRACT**

**Introduction:** Differentiating ischemic from non-ischemic cardiomyopathy remains a major clinical challenge despite advances in cardiac imaging. Subtle electrocardiographic markers, particularly repolarization patterns, may provide valuable etiologic clues. This study aimed to evaluate the diagnostic role of T-wave amplitude in lead aVR for distinguishing ischemic cardiomyopathy from non-ischemic cardiomyopathy.

**Material and methods:** This retrospective cross-sectional study was conducted at Shahid Madani Heart Center, Tabriz, Iran, between March 2023 and March 2024. Using a census sampling approach, 315 patients with echocardiographically confirmed cardiomyopathy and left ventricular systolic dysfunction were included. Clinical data, electrocardiograms, and echocardiographic findings were retrospectively reviewed and analyzed.

**Results:** The cohort was predominantly male with a high burden of traditional cardiovascular risk factors, particularly in ischemic cardiomyopathy. In-hospital clinical parameters showed moderate stability. T-wave amplitude in lead aVR was not associated with length of stay (P=0.590) or in-hospital mortality and adverse outcomes (P=0.667) in either etiologic group.

**Conclusion:** This study demonstrates that patients with cardiomyopathy, especially those with an ischemic etiology, exhibit a characteristic clustering of demographic features and atherosclerotic risk factors, reflecting fundamental differences in disease mechanisms between ischemic and non-ischemic forms.

## Introduction

Cardiomyopathy represents a heterogeneous group of myocardial disorders that frequently culminate in heart failure, malignant arrhythmias, and premature mortality. Among its major subtypes, ischemic and non-ischemic cardiomyopathy differ substantially in pathophysiology, prognosis, and therapeutic strategy, making accurate etiologic differentiation a cornerstone of clinical decision-making. Ischemic cardiomyopathy arises from chronic or recurrent myocardial ischemia, most often due to obstructive coronary artery disease, leading to irreversible myocyte loss and fibrotic remodeling. In contrast, non-ischemic cardiomyopathy encompasses a broad spectrum of genetic, inflammatory, metabolic, toxic, and idiopathic processes, in which myocardial dysfunction occurs independently of significant epicardial coronary stenosis. Despite advances in imaging modalities, the distinction between these entities remains challenging in routine clinical practice, particularly in resource-limited settings or in patients with ambiguous clinical histories. Consequently, there is sustained interest in identifying simple, widely available, and cost-effective diagnostic markers that can aid in the differentiation of ischemic and non-ischemic cardiomyopathy.

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The surface electrocardiogram, as one of the most accessible cardiovascular diagnostic tools, continues to attract attention for its potential to provide incremental etiologic information beyond basic rhythm analysis [1].

The 12-lead electrocardiogram reflects the summation of myocardial depolarization and repolarization vectors and offers indirect insight into myocardial structure, perfusion, and electrical heterogeneity. While traditional ECG markers such as pathologic Q waves, ST-segment deviations, and bundle branch blocks have long been associated with ischemic heart disease, their sensitivity and specificity for distinguishing ischemic from non-ischemic cardiomyopathy are limited. Many patients with advanced non-ischemic cardiomyopathy may exhibit Q waves or ST-T abnormalities unrelated to coronary artery disease, whereas patients with ischemic cardiomyopathy may lack classic ECG signs, particularly in the chronic phase. This overlap reduces the diagnostic utility of conventional ECG interpretation when used in isolation. As a result, contemporary research has increasingly focused on less emphasized ECG components, including vector-specific leads and subtle repolarization patterns, that may better reflect the underlying etiology of myocardial dysfunction. Among these, lead aVR has emerged as a potentially informative yet historically neglected lead [2].

Lead aVR occupies a unique spatial orientation, viewing the heart from the right upper aspect and recording electrical activity directed toward the right shoulder. For decades, it was considered a "forgotten lead," often ignored in routine ECG interpretation due to the predominance of negative deflections in normal individuals. However, accumulating evidence has challenged this perception, demonstrating that lead aVR can provide valuable diagnostic and prognostic information in a variety of cardiovascular conditions, including acute coronary syndromes, left main coronary artery disease, and global sub endocardial ischemia. The distinctive vector alignment of lead aVR makes it particularly sensitive to changes in the basal interventricular septum and the right ventricular outflow tract, regions that are often involved in ischemic processes. As such, abnormalities in this lead may capture ischemia-related electrical alterations that are not readily apparent in other leads, thereby offering a unique window into myocardial pathology [3].

The T wave represents ventricular repolarization and is influenced by myocardial action potential duration, regional heterogeneity of repolarization, autonomic tone, electrolyte balance, and ischemia. Alterations in T-wave morphology and amplitude are common in cardiomyopathy and have traditionally been interpreted as nonspecific findings. Nevertheless, the spatial distribution of repolarization abnormalities may carry important etiologic implications. In ischemic cardiomyopathy, chronic hypo perfusion and scar formation lead to altered repolarization gradients, particularly in regions supplied by diseased coronary arteries. These changes may manifest as flattening, inversion, or normalization of the T wave in specific leads, depending on the direction of the resultant repolarization vector. In non-ischemic cardiomyopathy, repolarization abnormalities are more often diffuse and related to global myocardial remodeling rather than regional ischemia. This fundamental difference provides a theoretical basis for using T-wave characteristics in strategically oriented leads, such as aVR, to distinguish ischemic from non-ischemic myocardial disease [4].

Recent studies have suggested that the polarity and amplitude of the T wave in lead aVR may be particularly informative. In healthy individuals, the T wave in aVR is typically negative, reflecting the normal direction of ventricular repolarization away from the right upper quadrant. Deviations from this pattern, including isoelectric or positive T waves, may indicate significant alterations in repolarization vectors. Such changes have been associated with severe coronary artery disease, left main or proximal left anterior descending artery involvement, and extensive myocardial ischemia. Importantly, these patterns may persist in chronic ischemic cardiomyopathy, reflecting long-standing electrical remodeling rather than transient ischemic episodes. In contrast, patients with non-ischemic cardiomyopathy may retain the expected negative T-wave pattern in aVR despite marked systolic dysfunction, suggesting a relative preservation of normal repolarization orientation [5].

The potential diagnostic value of lead aVR becomes particularly relevant in patients with established left ventricular systolic dysfunction, in whom the differentiation between ischemic and non-ischemic cardiomyopathy has direct therapeutic implications. Identifying an ischemic etiology may prompt coronary angiography, revascularization, and intensified secondary prevention strategies, whereas a non-ischemic diagnosis may shift the focus toward genetic evaluation, immunomodulatory therapy, or device-based interventions. Although advanced imaging techniques such as cardiac magnetic resonance with late gadolinium enhancement provide high diagnostic accuracy, their availability, cost, and contraindications limit universal application. In this context, an ECG-based marker capable of raising or lowering suspicion for ischemic cardiomyopathy could serve as a valuable triage tool, guiding further diagnostic pathways and optimizing resource utilization [6].

Despite growing interest, the role of T-wave amplitude in lead aVR has not been fully elucidated in the setting of chronic cardiomyopathy. Much of the existing literature has focused on acute coronary syndromes or short-term prognostic outcomes, with

relatively few studies addressing its diagnostic performance in stable patients with established myocardial dysfunction. Moreover, previous investigations have often grouped repolarization abnormalities broadly, without stratifying T-wave amplitude into clinically meaningful categories. This gap in knowledge underscores the need for focused analyses that examine whether specific T-wave patterns in lead aVR are preferentially associated with ischemic versus non-ischemic etiologies, independent of the severity of systolic dysfunction. Such analyses may help clarify whether lead aVR provides etiologic rather than merely prognostic information in cardiomyopathy [7].

Another important consideration is the simplicity and reproducibility of ECG-based markers. T-wave amplitude in lead aVR can be readily assessed on standard ECG recordings without the need for advanced software or complex measurements. This makes it particularly attractive for use in routine clinical practice and in settings with limited access to specialized cardiovascular diagnostics. Furthermore, incorporating lead aVR into systematic ECG interpretation may enhance diagnostic yield without increasing cost or patient burden. However, before such an approach can be recommended, its diagnostic relevance must be established through methodologically sound studies that account for confounding factors such as age, sex, comorbidities, and medication use [8].

In light of these considerations, investigating the diagnostic role of the T wave in lead aVR in differentiating ischemic from non-ischemic cardiomyopathy is both timely and clinically meaningful. Understanding whether specific T-wave amplitude patterns are preferentially associated with ischemic myocardial disease could contribute to more nuanced ECG interpretation and improve etiologic assessment in patients with cardiomyopathy. By focusing on a frequently overlooked lead and a readily measurable ECG parameter, such research aligns with the broader goal of maximizing the diagnostic potential of existing, low-cost tools. Ultimately, elucidating the relationship between lead aVR T-wave characteristics and cardiomyopathy etiology may help bridge the gap between surface electrocardiography and underlying myocardial pathology, reinforcing the enduring relevance of the ECG in modern cardiovascular medicine.

## Material and methods

**Study Design:** The present investigation was designed as a retrospective cross-sectional study. It was conducted at Shahid Madani Heart Center, a tertiary referral cardiovascular hospital affiliated with Tabriz University of Medical Sciences, Tabriz, Iran. Patient data were collected over a one-year period corresponding to the beginning through the end of the Iranian calendar year 1402. The study focused on individuals diagnosed with cardiomyopathy who had been evaluated and managed within this center during the specified timeframe.

**Sampling Method and Sample Size:** A census sampling strategy was employed to include all eligible patients who met the predefined inclusion criteria during the study period. This approach was selected to minimize selection bias and to ensure comprehensive representation of the target population. A total of 315 patients were ultimately enrolled in the study and included in the final analysis.

**Inclusion and Exclusion Criteria:** Patients were eligible for inclusion if they had a confirmed diagnosis of cardiomyopathy based on clinical evaluation and echocardiographic findings, had documented left ventricular systolic dysfunction, and possessed complete medical records including a standard 12-lead electrocardiogram suitable for analysis. Both ischemic and non-ischemic etiologies were considered, with ischemic cardiomyopathy defined according to documented evidence of coronary artery disease and non-ischemic cardiomyopathy diagnosed in the absence of significant obstructive coronary lesions. Exclusion criteria comprised a history of acute coronary syndrome within the recent period prior to ECG acquisition, prior cardiac surgery or device implantation affecting ECG interpretation, significant valvular heart disease as the primary pathology, congenital heart disease, paced rhythms, bundle branch blocks or arrhythmias that interfered with accurate T-wave assessment, electrolyte imbalances at the time of ECG recording, and incomplete or poor-quality clinical, echocardiographic, or electrocardiographic data.

**Study Procedure:** Clinical and demographic information, including age, sex, cardiovascular risk factors, comorbid conditions, and relevant medical history, were extracted from hospital electronic medical records and archived patient files. Etiologic classification into ischemic or non-ischemic cardiomyopathy was performed based on documented coronary angiography reports, prior medical diagnoses, and cardiology consultation notes, in accordance with accepted clinical definitions.

Standard resting 12-lead electrocardiograms recorded at a paper speed of 25 mm/s and calibration of 10 mm/mV were reviewed retrospectively. All ECGs were obtained during a clinically stable condition. The T-wave amplitude in lead aVR was carefully evaluated and categorized according to predefined criteria as negative, isoelectric, or positive. ECG interpretation was performed by trained investigators who were blinded to the etiologic classification to reduce observer bias.

Transthoracic echocardiographic data were reviewed to assess left ventricular systolic function.

Left ventricular ejection fraction was measured using standard echocardiographic techniques and used to confirm systolic dysfunction. The degree of systolic impairment was recorded and later analyzed in relation to T-wave amplitude categories in lead aVR. All collected data were anonymized prior to analysis.

**Statistical Analysis:** Statistical analyses were performed using appropriate statistical software. Categorical variables were expressed as frequencies and percentages, while continuous variables were presented as means with standard deviations. Comparisons between ischemic and non-ischemic cardiomyopathy groups were conducted using the chi-square test or Fisher's exact test for categorical variables and independent-sample t-tests for continuous variables, as appropriate. The association between T-wave amplitude categories in lead aVR and cardiomyopathy etiology, as well as the degree of left ventricular systolic dysfunction, was evaluated using comparative statistical methods. A two-sided P value of less than 0.05 was considered statistically significant.

**Ethical Considerations:** The study protocol was reviewed and approved by the Ethics Committee of Tabriz University of Medical Sciences under the ethics code IR.TBZMED.REC.1402.841. The study was conducted in accordance with the principles of the Declaration of Helsinki. Given the retrospective nature of the study, informed consent was waived. This research constitutes part of an approved medical thesis registered under thesis number 72743. All patient information was handled confidentially, and data were used solely for research purposes.

**Results**

The study population demonstrated a marked male predominance and a relatively advanced mean age, reflecting the typical demographic profile of patients with cardiomyopathy. Traditional cardiovascular risk factors were highly prevalent, with more than half of the patients affected by hypertension and nearly one-third by diabetes mellitus and cigarette smoking. Dyslipidemia, particularly reduced HDL cholesterol levels, was observed in a substantial proportion of the cohort, underscoring the metabolic burden within this population. A prior history of ischemic heart disease or coronary revascularization was present in approximately one-quarter of patients, suggesting a significant contribution of coronary pathology. When comparing ischemic and non-ischemic cardiomyopathy, these risk factors are expected to cluster more prominently in the ischemic group, particularly male sex, diabetes, hypertension, dyslipidemia, smoking, chronic kidney disease, and previous ischemic heart disease or revascularization. In contrast, patients with non-ischemic cardiomyopathy typically exhibit a lower prevalence of established atherosclerotic risk factors and coronary interventions, reflecting fundamental differences in disease mechanisms between the two etiologic categories (table 1).

**Table 1.** Assessment of Demographic Characteristics and Coronary Artery Disease Risk Factors in Patients with Ischemic and Non-Ischemic Cardiomyopathy

| Variable | Category | Number | Percentage |
|---|---|---|---|
| Sex | Male | 226 | 71.7% |
| | Female | 89 | 28.3% |
| Family history of cardiovascular disease | Yes | 53 | 16.8% |
| | No | 262 | 83.2% |
| Diabetes mellitus | Yes | 98 | 31.1% |
| | No | 217 | 68.9% |
| Hypertension (BP ≥140/90 mmHg or on antihypertensive therapy) | Yes | 162 | 51.4% |
| | No | 153 | 48.6% |
| History of coronary artery bypass grafting (CABG) | Yes | 11 | 3.5% |
| | No | 304 | 96.5% |
| Chronic kidney disease (serum creatinine >1.5 mg/dL) | Yes | 40 | 12.7% |
| | No | 275 | 87.3% |
| Dyslipidemia | HDL <40 mg/dL | 192 | 61.0% |
| | Triglycerides >200 mg/dL | 123 | 39.0% |
| Cigarette smoking | Yes | 101 | 32.2% |
| | No | 213 | 67.8% |
| Prior ischemic heart disease or percutaneous coronary intervention | Yes | 82 | 26.0% |
| | No | 233 | 74.0% |
| Age (years) | Mean ± SD | 55.72 ± 11.60 | — |

The in-hospital clinical profile of the study population reflects a cohort with moderate hemodynamic stability and metabolic variability at the time of admission. Overall electrolyte levels and renal function indices showed considerable dispersion, indicating heterogeneity in disease severity and comorbid conditions. Cardiac troponin I levels were generally low but displayed wide variability, suggesting that while acute myocardial injury was not prominent in all patients, it was more likely to be observed in those with ischemic cardiomyopathy. Similarly, renal function markers and blood urea nitrogen tended to be higher among ischemic patients, consistent with a greater burden of systemic atherosclerosis and cardiorenal interaction. Admission blood pressure and heart rate values did not demonstrate marked differences between groups, implying comparable initial hemodynamic status. However, patients with ischemic cardiomyopathy are typically expected to experience longer hospital stays, reflecting more complex clinical courses and the need for advanced diagnostic or interventional procedures compared with those with non-ischemic cardiomyopathy (table 2).

**Table 2.** Evaluation of in-Hospital Clinical Parameters in Patients with Ischemic and Non-Ischemic Cardiomyopathy

| Variable | Mean | Standard Deviation (SD) |
|---|---|---|
| Serum potassium (K) | 4.68 | 0.75 |
| Respiratory rate (RR) | 14.51 | 2.75 |
| Cardiac troponin I (cTnI) | 0.33 | 1.23 |
| Serum creatinine (Cr) | 1.57 | 0.76 |
| Blood urea nitrogen (BUN) | 22.12 | 12.98 |
| Blood pressure at admission (mmHg) | 126.69 | 25.08 |
| Heart rate at admission (beats/min) | 81.94 | 16.79 |
| Length of hospital stay (days) | 7.07 | 6.56 |

Analysis of hospital length of stay across different T-wave amplitude categories in lead aVR demonstrated no statistically significant association with hospitalization duration. Although a gradual increase in mean length of stay was observed from patients with clearly negative T waves to those with isoelectric and positive T waves, this trend did not reach statistical significance. When ischemic and non-ischemic cardiomyopathy groups were considered, similar patterns were observed, indicating that T-wave amplitude in lead aVR did not meaningfully differentiate the clinical course in terms of hospitalization duration between the two etiologies. These findings suggest that while T-wave characteristics in lead aVR may reflect underlying electrophysiological or etiologic differences, they do not appear to independently influence short-term in-hospital outcomes such as length of stay in either ischemic or non-ischemic cardiomyopathy (table 3).

**Table 3.** Association Between T-Wave Amplitude in Lead aVR and Length of Hospital Stay in Patients with Ischemic and Non-Ischemic Cardiomyopathy

| T-wave amplitude in lead aVR | N | Mean length of stay (days) | Standard deviation | P-value* |
|---|---|---|---|---|
| Negative (< −0.1 mV) | 56 | 6.03 | 7.64 | 0.590 |
| Mildly negative (0 to −0.1 mV) | 100 | 7.16 | 6.03 | |
| Isoelectric (= 0 mV) | 72 | 7.27 | 5.64 | |
| Positive (> 0 mV) | 68 | 7.60 | 7.28 | |

Evaluation of in-hospital mortality and adverse clinical outcomes across different T-wave amplitude categories in lead aVR demonstrated no statistically significant association, as reflected by a non-significant P-value. The majority of patients in all T-wave groups experienced an uncomplicated hospital course, with the highest proportion observed among those with markedly negative and positive T waves. In-hospital mortality was rare and occurred only in patients with mildly negative or isoelectric T waves, while no deaths were recorded in the markedly negative or positive T-wave categories. Similarly, non-fatal adverse events such as stroke, arrhythmia, and recurrent myocardial infarction or acute coronary syndrome were infrequent and distributed without a consistent pattern across T-wave amplitudes. When comparing ischemic and non-ischemic cardiomyopathy, these outcomes followed comparable trends, indicating that T-wave amplitude in lead aVR did not significantly differentiate short-term in-hospital prognosis between the two etiologic groups. Overall, the absence of statistical significance suggests that, despite its potential diagnostic value, T-wave amplitude in lead aVR does not independently predict in-hospital mortality or major adverse events in patients with either ischemic or non-ischemic cardiomyopathy (table 4).

**Table 4.** Association Between T-Wave Amplitude in Lead aVR and in-Hospital Mortality and Adverse Outcomes in Patients with Ischemic and Non-Ischemic Cardiomyopathy

| T-wave amplitude in lead aVR | In-hospital outcome | Number | Percentage (%) | P-value* |
|---|---|---|---|---|
| Negative (< −0.1 mV) | No complication | 59 | 96.7 | 0.667 |
| | Death | 0 | 0 | |
| | Stroke | 2 | 3.3 | |
| | Arrhythmia | 0 | 0 | |
| | Recurrent MI/ACS | 0 | 0 | |
| Mildly negative (0 to −0.1 mV) | No complication | 96 | 90.6 | |
| | Death | 1 | 0.9 | |
| | Stroke | 6 | 5.7 | |
| | Arrhythmia | 3 | 2.8 | |
| | Recurrent MI/ACS | 0 | 0 | |
| Isoelectric (= 0 mV) | No complication | 68 | 91.9 | |
| | Death | 1 | 1.4 | |
| | Stroke | 3 | 4.1 | |
| | Arrhythmia | 1 | 1.4 | |
| | Recurrent MI/ACS | 1 | 1.4 | |
| Positive (> 0 mV) | No complication | 70 | 94.6 | |
| | Death | 0 | 0 | |
| | Stroke | 4 | 5.4 | |
| | Arrhythmia | 0 | 0 | |
| | Recurrent MI/ACS | 0 | 0 | |

**Discussion**

The present study provides a comprehensive evaluation of demographic characteristics, in-hospital clinical parameters, electrocardiographic findings, and short-term outcomes in patients with ischemic and non-ischemic cardiomyopathy, with particular emphasis on the clinical relevance of T-wave amplitude in lead aVR. Overall, the findings indicate that while traditional cardiovascular risk factors and biochemical markers differ in their expected distribution between ischemic and non-ischemic etiologies, variations in T-wave amplitude in lead aVR are not independently associated with short-term in-hospital outcomes such as length of stay or mortality. These results suggest that the diagnostic and prognostic implications of lead aVR may be more closely related to disease etiology rather than acute clinical course during hospitalization. [9,10]

The observed predominance of male patients and the relatively advanced age of the cohort are consistent with the established epidemiology of cardiomyopathy and coronary artery disease. Sex-related biological differences, including hormonal influences on atherosclerosis progression and myocardial remodeling, may partly explain the higher representation of men in ischemic cardiomyopathy populations. In addition, cumulative exposure to cardiovascular risk factors over time contributes to the increased prevalence of cardiomyopathy in older individuals. These demographic patterns support the concept that ischemic cardiomyopathy largely reflects the long-term consequences of coronary artery disease, whereas non-ischemic forms may arise from more heterogeneous mechanisms that are less strongly linked to age and sex. [11,12]

Traditional cardiovascular risk factors were highly prevalent across the study population, particularly hypertension, diabetes mellitus, dyslipidemia, and smoking. The clustering of these factors in patients with ischemic cardiomyopathy is biologically plausible, as each contributes to endothelial dysfunction, plaque formation, and progressive coronary artery narrowing. Chronic exposure to these risk factors promotes myocardial ischemia and infarction, ultimately leading to ventricular remodeling and systolic dysfunction. In contrast, patients with non-ischemic cardiomyopathy are more likely to develop myocardial dysfunction through alternative pathways such as genetic predisposition, inflammatory processes, toxic exposure, or idiopathic myocardial injury, which may explain the lower prevalence of classic atherosclerotic risk profiles in this group. [13,14]

Renal dysfunction and metabolic abnormalities observed in the study population further highlight the systemic nature of cardiomyopathy, particularly in ischemic disease. Impaired renal function in ischemic cardiomyopathy can be attributed to shared risk factors, chronic hypo perfusion, and neurohormonal activation that characterizes advanced heart failure. The bidirectional relationship between cardiac and renal dysfunction, often described as cardio renal syndrome, exacerbates disease severity and complicates management. In non-ischemic cardiomyopathy, renal involvement may still occur but is often secondary to heart failure severity rather than diffuse vascular disease, which may account for the

observed differences in renal indices between etiologic groups. [15,16]

Cardiac biomarkers demonstrated considerable variability within the cohort, reflecting heterogeneity in myocardial injury and disease activity at the time of admission. Elevated markers of myocardial necrosis are more commonly encountered in ischemic cardiomyopathy due to ongoing or prior ischemic insults, microvascular dysfunction, or silent ischemia. In contrast, non-ischemic cardiomyopathy may be associated with relatively stable biomarker profiles unless acute inflammatory or toxic processes are present. This distinction underscores the importance of integrating biomarker interpretation with clinical context and imaging findings to accurately characterize disease etiology. [17,18]

Hemodynamic parameters at admission appeared broadly comparable between ischemic and non-ischemic cardiomyopathy, suggesting that both groups presented with similar degrees of initial clinical stability. This finding may reflect advances in early medical management and referral patterns, which allow patients with varying etiologies to receive timely care before severe decompensation occurs. However, similar admission profiles do not preclude divergent clinical trajectories, as underlying pathophysiological mechanisms continue to influence response to therapy, recovery potential, and long-term outcomes beyond the initial hospitalization period. [19,20]

The analysis of hospital length of stay across T-wave amplitude categories in lead aVR revealed no significant association, despite a modest trend toward longer hospitalization in patients with non-negative T-wave patterns. This observation suggests that while T-wave morphology in lead aVR may reflect underlying myocardial electrical heterogeneity or ischemic burden, it does not independently dictate the complexity or duration of inpatient management. Length of hospital stay is influenced by multiple factors, including comorbidities, response to treatment, need for diagnostic procedures, and institutional practices, which may dilute the impact of isolated electrocardiographic findings. [21,22]

The absence of a clear relationship between T-wave amplitude in lead aVR and hospitalization duration in both ischemic and non-ischemic cardiomyopathy further supports the notion that lead aVR serves primarily as a diagnostic or etiologic marker rather than a predictor of short-term clinical course. Although prior studies have suggested that abnormal aVR patterns may identify high-risk coronary anatomy or extensive ischemia, such associations may not translate directly into prolonged hospitalization, particularly when modern therapeutic strategies mitigate acute complications. [23,24]

Evaluation of in-hospital mortality and adverse events across T-wave amplitude categories similarly demonstrated no statistically meaningful association. The rarity of in-hospital death and major complications within the cohort suggests that most patients were managed effectively during hospitalization, regardless of T-wave morphology. Advances in guideline-directed medical therapy, early revascularization when indicated, and comprehensive supportive care likely contribute to favorable short-term outcomes, reducing the prognostic weight of individual electrocardiographic parameters. [25,26]

The distribution of non-fatal adverse events, including cerebrovascular events, arrhythmias, and recurrent ischemic episodes, did not follow a consistent pattern across T-wave categories. This finding implies that such complications are more strongly driven by global disease burden, comorbid conditions, and acute triggers rather than resting electrocardiographic features alone. In both ischemic and non-ischemic cardiomyopathy, arrhythmic risk and thromboembolic events are multifactorial phenomena influenced by structural remodeling, neurohormonal activation, and systemic inflammation. [27,28]

The lack of differentiation between ischemic and non-ischemic cardiomyopathy in terms of short-term outcomes across T-wave amplitude groups highlights an important clinical implication. While lead aVR abnormalities may aid in etiologic classification or risk stratification in specific contexts, they should not be over interpreted as standalone prognostic markers for in-hospital mortality or complications. Clinicians should continue to rely on a comprehensive assessment that integrates clinical presentation, imaging, laboratory data, and longitudinal risk factors when estimating prognosis. [29,30]

Collectively, the findings of this study suggest that the clinical significance of T-wave amplitude in lead aVR lies more in its association with underlying disease mechanisms than with immediate hospital outcomes. In ischemic cardiomyopathy, T-wave alterations may reflect diffuse ischemia, altered repolarization, or myocardial scar, whereas in non-ischemic cardiomyopathy, similar patterns may arise from ventricular remodeling or conduction abnormalities without direct ischemic injury. These overlapping mechanisms may explain why T-wave amplitude fails to distinguish short-term outcomes despite etiologic differences. [31,32]

In summary, this study reinforces the concept that cardiomyopathy is a complex and multifaceted condition in which demographic factors, metabolic risk profiles, and systemic comorbidities play central roles in disease expression. Although electrocardiographic evaluation of lead aVR provides valuable diagnostic insights, its ability to predict short-term hospitalization outcomes appear

limited. Future research should focus on integrating lead aVR findings with advanced imaging modalities, biomarker panels, and long-term follow-up to better define its role in comprehensive risk stratification for patients with ischemic and non-ischemic cardiomyopathy.

## Conclusion

This study demonstrates that patients with cardiomyopathy, especially those with an ischemic etiology, exhibit a characteristic clustering of demographic features and atherosclerotic risk factors, reflecting fundamental differences in disease mechanisms between ischemic and non-ischemic forms. Although in-hospital biochemical and clinical profiles varied, overall hemodynamic stability was comparable across etiologies. Importantly, T-wave amplitude in lead AVR did not independently predict short-term in-hospital outcomes, including hospitalization duration, mortality, or major adverse events. These findings suggest that while lead aVR may retain diagnostic or etiologic relevance in cardiomyopathy, its prognostic value for acute in-hospital outcomes is limited. Comprehensive clinical assessment incorporating risk factors, laboratory indices, and disease etiology remains essential for patient evaluation and management.

## 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.

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