CARDIOLOGIE TUNISIENNE

ARTICLE ORIGINAL

Apport de l'ECG dans l'hypertrophie ventriculaire gauche du rétrécissement aortique serré

Diagnostic usefulness of ECG for hypertrophy in aortic valve stenosis

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Summary

Background: Left ventricular hypertrophy (LVH) in aortic stenosis (AS) is an adaptive response of the myocardium to chronic pressure overload. Although electrocardiography (ECG) was used to be a traditional method to detect LVH, nowadays, echocardiography has emerged as a routine technique to diagnose LVH. Various ECG criteria for LVH have been proposed, but their association with the severity of aortic stenosis is unknown.

Aim : To evaluate the prevalence of the electrocardiographic criteria for LVH in patients with clinically significant AS and to evaluate the relationship between the ECG criteria for LVH and echocardiographic parameters.

Methods : The clinical data of 132 patients diagnosed in the cardiology department of Charles Nicolle hospital-Tunisia with moderate to severe AS were retrospectively analyzed. Nine ECG criteria for identification of LVH were used and compared to the results of transthoracic echocardiography (TTE).

Results : The population was composed 77 men and 55 women with a mean age 68,2 ± 10,1 years. In 62% of patients, at least one of the ECG criteria for LVH was found and the prevalence of LVH at echography was 81 %. These patients had a greater left ventricular mass index (LVMI) (158.8 ± 35.6 vs. 128.1 ± 25.5 g/m2, p = 0.03) and peak aortic jet velocity (4.5 ± 0.5 vs. 3.9 ± 0.9 m/s, p = 0.028) along with smaller aortic valve area (0.71± 0.13 vs 0.91 ± 0.32 cm2, p = 0.03) compared to patients with a negative ECG for LVH. **T**he ECG parameters had a low sensitivity (10%–36.4%) with a specificity of up to 100%.

There was a positive correlation between the majority of ECG parameters and the LVMI as well as the peak aortic jet velocity however the strength of those correlations still weak. The highest correlation with the LVMI and with peak aortic jet velocity was for the Cornell Voltage (r = 0.351, p = 0.016 and r= 0.329, p = 0.002 respectively).

Conclusion: The ECG criteria for LVH in patients with moderate or severe AS have a poor sensitivity in identifying LVH confirmed by TTE. Cornell Voltage was the best-performing criterion which the best correlates with the LVMI and with peak aortic jet velocity.

Keywords

Aortic valve stenosis - Left ventricular hypertrophy – Electrocardiography - Echocardiography - Cornell voltage

Résumé

Introduction : L' hypertrophie ventriculaire gauche (HVG) dans le rétrécissement aortique (RA) est une réponse adaptative du myocarde à une surcharge de pression chronique. Bien que l'électrocardiographie (ECG) ait été la méthode traditionnelle de détection de l'HVG, l'échocardiographie est actuellement le gold standard pour son diagnostic. Divers critères ECG pour l'HVG ont été proposés, mais leur association avec la sévérité de la sténose aortique reste méconnue.

Objectif : Évaluer la prévalence de l'HVG selon des critères électrocardiographiques d'HVG chez les patients présentant un RA cliniquement significatif et évaluer la relation entre ces critères ECG et les paramètres échocardiographiques.

Méthodes : Les données cliniques de 132 patients diagnostiqués avec un RA modéré à sévère dans le service de cardiologie de l'hôpital Charles Nicolle (Tunisie) ont été analysées rétrospectivement. Neuf critères ECG d'identification de l'HVG ont été utilisés et comparés aux résultats de l'échocardiographie transthoracique (ETT).

Résultats : Notre étude, incluant 132 patients (âge moyen 68,2 ±10,1 ans ; 77 hommes) , a révélé une prévalence de HVG de 81 % à l'échocardiographie contre 62 % via les critères électrocardiographiques (ECG). Les patients présentant un ECG positif pour l'HVG aivaient un profil hémodynamique significativement plus sévère, caractérisé par un indice de masse ventriculaire gauche (IMVG) plus élevé (158,8 ±35,6 vs 128,1 ±25,5 g/m², p = 0,03) , une vitesse maximale du jet aortique supérieure (4,5 ±0,5 vs 3,9 ± 0,9 m/s, p = 0,028) et une surface valvulaire aortique plus réduite (0,71 ±0,13 vs 0,91 ±0,32 cm², p = 0,03). Malgré une spécificité atteignant 100 %, les paramètres ECG ont montré une faible sensibilité (10 % à 36,4 %) et des corrélations globalement faibles avec les données échocardiographiques , bien que l'indice de Cornell se soit distingué par la corrélation la plus élevée tant pour l'IMVG (r = 0,351, p = 0,016) que pour la vitesse maximale du jet (r = 0,329, p = 0,002).

Conclusion : Les critères ECG d'HVG chez les patients souffrant de RA modéré ou sévère présentent une faible sensibilité pour identifier l'HVG confirmée par ETT. Les valeurs des critères ECG sélectionnés ne sont que faiblement corrélées aux indices échographiques d'HVG ainsi qu'aux marqueurs de sévérité du RA.

Mots-clés

Rétrécissement aortique - Hypertrophie ventriculaire - Électrocardiographie -Échocardiographie - Indice de Cornell

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INTRODUCTION

Aortic valve stenosis (AS) is a valvular heart disease entity in which the aortic valve becomes stenotic and thereby increases the afterload to the left ventricle [1]. The frequency of aortic valve stenosis (AS) is on the rise in the overall population due to longer life expectancy and the aging of the population [2]. So it is expected that AS will become an increasingly common health problem. Left ventricular hypertrophy (LVH) is an adaptive response of the myocardium to chronic pressure overload[3]. The increasing afterload seen at the LV over a period of time leads to a steep rise in the prevalence and severity of LVH in patients with aortic stenosis. Therefore, it is logical to infer that ECG criteria used as a primary screening tool, which reflect LVH will be useful indicators of the severity of stenosis and the future clinical problems of patients with AS [4]. However, although those electrocardiographic parameters related to left ventricular hypertrophy (LVH) exist, their accuracy in AS patients remains inadequately assessed [5].

Through this study we aimed to evaluate the prevalence of the electrocardiographic criteria for LVH in patients with clinically significant AS and to evaluate the relationship between the ECG criteria for LVH and echocardiographic parameters.

METHODS

This was a descriptive retrospective, mono centric, study. We enrolled all patients diagnosed consecutively with moderate to severe AS between January 2017 and October 2022 in the in the cardiology department of Charles Nicolle academic hospital Tunis-Tunisia. All of the clinical, electrocardiographic, and echocardiographic data were analyzed. Patients were excluded if their EKG present with QRS duration of more than 120 ms, complete and incomplete bundle branch blocks, nonspecific intraventricular conduction disturbances, paced rhythms, and those with history of previous aortic valve replacement. For each ECG performed and, we used those following often used criteria to assess LVH:

- The amplitude of the R wave in aVL > 1.1 mV [6]
- Sokolow-Lyon index: the sum of the S wave in V1 and the R wave in V5 or V6 > 3.5 mV [7]
- Sokolow Product : the sum of QRS duration times the corresponding Sokolow-Lyon voltage ≥3,340 mm × ms [8]
- Cornell Voltage: the sum of the R wave in aVL and the S wave in V3 > 2.0 mV for women and >2.8 mV for men [9]
- Cornell Product : QRS duration times the corresponding Cornell voltage ≥2,440 mm × ms [10]
- Lewis Index [11]
- Peguero–Lo Presti Index : the sum of the amplitude of the deepest S wave in any lead and the S wave in lead V4 ≥ 2.8 mV in men and ≥2.3 mV in women [12]
- Framingham-adjusted Cornell voltage [13]
- Romhilt Estes Score [14]

An ECG was classified as positive for LVH if at least one of the LVH criteria was found.

All of the echocardiographic examinations were performed by experienced cardiologist with General Electric Vivid E9. The measurements of the intraventricular septum diameter (IVSd), left ventricular internal diameter (LVIDd), and posterior wall thickness diameter (PWTd) were used to calculate the left ventricular mass (LVM) according to the formula: $0.8 \times 1.4 \times [(IVSd + LV\ IDd + PWTd)^3 - LVIDd^3] + 0.6g$ as recommended by the guidelines of the American Society of Echocardiography [15].

The presence of LVH in the TTE was determined using the left ventricular mass index (LVMI), which is defined as LVM/ body surface area (g/m2). The upper normal range for the LVMI was 95 g/m2 in women and 115 g/m2 in men. Higher values indicate LVH. [15]

The peak aortic jet velocity and the aortic mean gradient were measured using continuous-wave Doppler and the multiple acoustic windows approach. The continuity equation was used to calculate the aortic valve area (AVA) [16] The severity of the aortic stenosis was assessed according to ESC guidelines on the management of valvular heart [16]

STATISTICAL ANALYSIS

Sensitivity and specificity, of electrocardiographic LVH criteria were calculated based on results of TTE. Sensitivity was computed using formula: number of true positives/ (number of true positives + number of false negatives). Specificity: number of true negatives/(number of true negatives + number of false positives). Data were expressed as mean ± standard deviation or median (interquartile range) for continuous variables or as counts and percentages for categorical variables an independent group t student test was carried out for comparisons of means between two independent normally distributed groups. while the non-normal data were compared using the Mann–Whitney U test. Comparisons of percentages on independent groups

were performed using the Pearson Chi-squared ($\chi 2$) test and when this test was not applicable, the Fisher's exact test two-tailed was used. The Spearman's rank correlation coefficient was used for testing the strength of the correlation between two variables A p value of < 0.05 was considered to be statistically significant.

RESULTS

The population was composed 77 men and 55 women with a mean age 68,2 ± 10,1 years.

In 62.12% (n=81) of patients, at least one of the ECG criteria for LVH was found and the prevalence of LVH at echography was 81% (n=107).

These patients had a greater LVMI (158.8 ± 35.6 vs. 128.1 ± 25.5 g/m^2, p = 0.03) and peak aortic jet velocity (4.5 ± 0.5 vs. 3.9 ± 0.9 m/s, p = 0.028) along with smaller aortic valve area (0.71± 0.13 vs 0.91 ± 0.32 cm^2, p = 0.03) compared to patients with a negative ECG for LVH

Table 1. Echocardiographic findings of the study population

	Positive ECG for LVH (n = 82)	Negative ECG for LVH (n = 50)	P
Left ventricular ejection fraction (%)	56.7 ± 8.4	53.9 ± 9.8	0.12
Left ventricular mass index (g/m^2)	158.8 ± 35.6	128.1 ± 25.5	0.03*
Peak aortic jet velocity	4.5 ± 0.5	3.9 ± 0.9	0.028*
Aortic mean gradient	42.1 ± 10.8	37.7 ± 17.8	0.06
Aortic valve area	0.71± 0.13	0.91 ± 0.32 cm^2	0.03*
LVH in TTE	70 (85%)	37 (74%)	0.09
Severe AS	74(90%)	29 (58%)	0.01*

*Statistically significant (p < 0.05)

The most frequent positive criteria were the Cornell Voltage index criteria (39 patients, 36.4%), the Cornell Voltage product (37 patients, 34.6%), and the Romhilt Estes Score (35 patients, 32.9%). The ECG criteria had a low sensitivity, which ranged from 10.2 to 36.4% with a specificity of up to 100%.

Table 2. Characteristics of the ECG criteria for LVH compared to echocardiographic-based LVH

	Sensitivity (%)	Specificity (%)
R aVL (mm)	10.2	100
Sokolow Index(mm)	29.2	80
Sokolow Product (mm.ms)	24.4	76
Cornell Voltage (mm)	36.4	68
Cornell Product (mm.ms)	34.5	64
Lewis Index (mm)	12.6	88
Peguero Lo Presti Index (mm)	26.2	76
Framingham-adjusted Cornell voltage (mm)	17.6	84
Romhilt Estes Score ≥ 4 (points)	32.9	96

Patients who met at least one electrocardiographic criterion for LVH exhibited a significantly more advanced hemodynamic profile compared to those with a negative ECG. Specifically, the ECG-positive group demonstrated a significantly higher peak aortic jet velocity (4.5±0.5m/s vs. 3.9 ± 0.9 m/s, p = 0.028). Furthermore, this group had a significantly smaller aortic valve area (0.71± 0.13cm^2 vs 0.91± 0.32cm^2, p = 0.03) and a higher prevalence of severe AS (90% vs 58%, p = 0.01).

There was a positive correlation between the majority of ECG criteria and the LVMI as well as the peak aortic jet velocity however the strength of those correlations still weak. The highest correlation with the LVMI and with peak aortic jet velocity was for the Cornell Voltage (r = 0.351, p = 0.016 and r= 0.329, p = 0.002 respectively).

Table 3. Correlation between ECG criteria for LVH and the echocardiographic parameters

		LVM	LVM i	AV Area	Peak aortic jet velocity	Mean aortic gradient
R aVL (mm)	r	0.108	0.045	0.07	0.124	0.099
	p	0.321	0.681	0.946	0.251	0.363
Sokolow Index(mm)	r	0.235	0.023	-0.1	0.188	0.156
	p	0.028	0.038	0.926	0.042	0.148
Sokolow Product (mm.ms)	r	0.229	0.196	-0.031	0.220	0.167
	p	0.033	0.049	0.772	0.041	0.123
Cornell Index (mm)		0.257	0.251	-0.090	0.329	0.301
	p	0.016	0.019	0.407	0.002	0.005
Cornell Product (mm.ms)		0.241	0.213	-0.041	0.254	0.183
	p	0.025	0.048	0.708	0.018	0.09
Lewis Index (mm)	r	0.039	-0.024	-0.016	0.120	0.075
	p	0.721	0.827	0.883	0.268	0.489
Peguero–Lo Presti Index (mm)	r	0.256	0.256	-0.209	0.314	0.373
	p	0.017	0.017	0.042	0.003	0.00001
Framingham-adjusted Cornell (mm)	r	0.365	0.337	-0.225	0.385	0.361
	p	0.001	0.001	0.036	0.0001	0.01
Romhilt Estes Score ≥ 4 (points)	r	0.172	0.132	-0.112	0.124	0.146
	p	0.112	0.223	0.300	0.253	0.178

DISCUSSION

The chronic pressure overload imposed by a stenotic aortic valve triggers LVH as a fundamental adaptive response. While initially compensatory to maintain cardiac output, this process ultimately leads to adverse clinical outcomes, including heart failure and increased mortality[17,18]. Electrocardiography (ECG), by virtue of its low cost, universal availability, and ability to detect the electrical manifestations of increased myocardial mass, has long been employed as

an initial contemporary LVH ECG criteria specifically in the context of AS remains incompletely characterized [19]. Our study highlights a critical diagnostic gap: while anatomical LVH was prevalent in 81% of our cohort, surface ECG failed to detect it in a significant proportion of patients.

The Paradox of Low Sensitivity and High Specificity

Our findings demonstrate that traditional ECG criteria exhibit consistently poor sensitivity in patients with moderate to severe AS, ranging from 10.2% to 36.4%. This observation aligns with previous work by Bula et al., who reported a similar sensitivity range (6% to 36.4%) in significant AS. Conversely, the specificity in our study was remarkably high, reaching 100% for the R wave in aVL > 1.1mVand 96% for the Romhilt-Estes score[19]. More recently, Chyrchel et al. found that classical ECG criteria achieved sensitivities of only 40.8% at best in 74 patients with severe AS, with no incremental diagnostic value observed when stratifying by coronary artery disease burden [20]. The consistency of these findings across different cohorts underscores the inherent limitations of surface ECG in detecting anatomical hypertrophy within the pressure-overloaded left ventricle. Conversely, specificity in the present study was notably high, reaching 100% for the Cornell Voltage criterion and 96% for the Romhilt-Estes score. Similar values have been reported by Bula et al., who documented specificities of up to 100% for several criteria[19]. This high specificity indicates that while a positive ECG finding is strongly predictive of echocardiographic LVH, a negative study cannot reliably exclude its presence.

Pathophysiological Mechanisms of Electrical-Anatomical Discordance

Despite the high prevalence of echocardiographic LVH in the present cohort (81%), the majority of ECG criteria failed to identify a substantial proportion of patients with anatomically confirmed hypertrophy.

The dissociation between electrical and anatomical findings reflects the complex myocardial response to chronic pressure overload. Several factors contribute to this discordance:

- Myocardial Fibrosis: Persistent afterload leads to cardiomyocyte apoptosis and progressive fibrosis. [17,21] Fibrotic tissue is electrically inert and disrupts the propagation of depolarization. Consequently, a ventricle with high myocardial mass may generate attenuated QRS voltages if viable myocardium has been replaced by collagen. [17]
- Geometric Remodeling: Heterogeneity in remodeling patterns—ranging from concentric to eccentric hypertrophy—alters the direction and magnitude of electrical forces. Concentric remodeling, typical of AS, may not project electrical forces as effectively toward surface electrodes as eccentric forms. [22,23]
- Confounding Variables: Factors such as obesity increase the distance between the heart and electrodes, attenuating precordial voltages. This particularly impacts the Sokolow-Lyon index, which we found to be less reliable than Cornell-based criteria.

Superiority of Cornell Voltage Criteria

The Cornell Voltage and product emerged as our best-performing indices, with the highest sensitivity (36.4%) and strongest correlations with the LVMI ($r = 0.351$, $p = 0.016$). The superiority of Cornell criteria likely stems from their incorporation of both limb (aVL) and precordial (V3) leads. This allows for the capture of electrical activity across two orthogonal planes, providing a more comprehensive assessment of the hypertrophied ventricle than single-plane indices [5,19] . Conversely, the Sokolow-Lyon index demonstrated inferior performance (sensitivity 29.2%), consistent with regression analysis showing its susceptibility to body mass index and hemodynamic load, whereas Cornell voltage remains predominantly determined by left ventricular mass [24]. The Romhilt-Estes score achieved excellent specificity (96%), and the Peguero-Lo Presti index demonstrated significant correlations with both LVMI and peak aortic jet velocity, warranting further investigation [19].

While some criteria are nearly 100% specific, the Cornell Voltage sacrifices some specificity to achieve its superior sensitivity. Its superiority is the most actionable clinical takeaway of this study. In a clinical setting, while the overall sensitivity of ECG is low (10.2% to 36.4%), a positive Cornell Voltage provides the strongest «rule-in» evidence for LVH, correlating significantly with both anatomical mass and hemodynamic severity.

The Link Between Electrical Manifestations and Hemodynamic Severity

A critical finding of this study is the significant, though weak, association between a positive ECG for LVH and the severity of the valvular obstruction. Specifically, the Cornell voltage ($r = 0.329$) and Framingham-adjusted Cornell ($r = 0.385$) showed associations with hemodynamic markers. However, correlations with the AVA were generally absent, echoing previous findings [19, 20, 24]. The significantly higher peak aortic jet velocity (4.5 m/s vs 3.9m/s) in the ECG-positive group suggests that while the ECG has low sensitivity, it effectively identifies a cohort with a higher hemodynamic burden. This relationship can be interpreted through several physiological lenses:

- The «Threshold» Effect: The increased afterload from a higher jet velocity (> 4m/s) likely drives the myocardial mass beyond a certain «electrical threshold» where it finally becomes detectable on a surface ECG, despite the presence of confounding factors like fibrosis.
- Prognostic Stratification: Since patients with positive ECG criteria are more likely to have severe AS (90% in our cohort), the ECG serves as a low-cost «red flag» for advanced disease that requires urgent echocardiographic confirmation.
- Electrical-Hemodynamic Coupling: While correlations with AVA were absent, the correlation with peak velocity suggests that the ECG may be more sensitive to the pressure-gradient-driven response of the myocardium than to the anatomical size of the valve orifice itself.

These divergent observations underscore the complexity of the relationship between electrical hypertrophy and hemodynamic burden. Several interrelated factors explain this discordance. First, myocardial fibrosis renders myocardial tissue electrically inert, attenuating QRS voltages despite increased left ventricular mass [3]. Second, obesity attenuates QRS amplitudes, particularly in precordial leads, due to increased distance between heart and electrodes [12]. Bula et al demonstrated that body mass index independently impacts Sokolow-Lyon voltage, whereas Cornell voltage remains predominantly determined by left ventricular mass [5]. Third, age-related changes and sex-specific differences in ventricular geometry further modulate electrocardiographic expression of hypertrophy[25,26] . Fourth, heterogeneity in left ventricular remodeling patterns ranging from concentric to eccentric hypertrophy alters the direction and magnitude of electrical forces[27] . Finally, subtle intraventricular conduction delays, despite exclusion of overt bundle branch blocks, may affect voltage summation. Collectively, these factors explain why the relationship between electrical hypertrophy and hemodynamic burden is neither linear nor consistent across all criteria, reflecting the multifaceted nature of the myocardial response to chronic pressure overload.

Clinical Implications and Future Directions

The poor sensitivity of ECG precludes its use as a standalone screening tool for LVH in AS. However, it retains value as a complementary tool for risk stratification. Patients with positive ECG criteria may represent a specific phenotype with less advanced fibrosis and a higher capacity for reverse remodeling following valve intervention. [17]

Future research integrating artificial intelligence and advanced multimodality imaging (such as Cardiac MRI for fibrosis quantification) is necessary to further refine the role of the ECG in the modern management of aortic stenosis.

Strengths and Limitations

The present study offers several strengths, including comprehensive evaluation of nine ECG-LVH criteria in a well-characterized AS cohort and strict exclusion of conduction abnormalities to ensure voltage-based assessments were not confounded. However, limitations must be acknowledged. The retrospective, single-center design introduces potential selection bias, and the moderate sample size precluded robust subgroup analyses. Echocardiography as the reference standard is inherently less accurate than cardiac magnetic resonance for mass quantification. The absence of direct fibrosis measures renders explanations for ECG-echocardiography discordance speculative. Additionally, the study was not designed to assess prognostic implications of ECG criteria. Prospective multicenter validation with multimodality imaging is required.

CONCLUSION

This study demonstrates that electrocardiographic criteria for left ventricular hypertrophy in patients with moderate to severe aortic stenosis exhibit limited sensitivity but high specificity when referenced against echocardiographic standards. The Cornell voltage criteria emerged as the best-performing index, correlating most strongly with left ventricular mass index and markers of hemodynamic severity. The discordance between electrical and anatomical hypertrophy is multifactorial, with myocardial fibrosis, obesity, age, sex, and ventricular geometry all contributing to QRS voltage attenuation. While ECG cannot replace echocardiography for the diagnosis and quantification of LVH in AS, it retains clinical value as a complementary tool for risk stratification and monitoring. Further research integrating advanced imaging and artificial intelligence may refine the role of electrocardiography in the modern management of aortic stenosis.

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