Original Article

Detection and Location of Myocardial Infarction Using Electrocardiogram: Validation by Cardiovascular Magnetic Resonance Imaging

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Abstract

Background: In the assessment of ischemic heart disease, cardiac magnetic resonance (CMR) is considered the gold standard for detecting and locating myocardial infarction (MI), but electrocardiogram (ECG) is less expensive and more widely available. Recognition of MI on ECG outside the acute phase is challenging; Q waves are absent in a significant proportion of patients and may reduce or disappear over time. Although ECG is widely used in the initial assessment of previous infarction, studies to validate ECG using CMR as a reference in the context of chronic coronary disease are limited.

Objectives: To evaluate the diagnostic performance of ECG in detecting and locating CMR-defined MI.

Methods: This study included 352 individuals who underwent CMR and ECG, 241 patients with previous MI confirmed by CMR and 111 controls with normal CMR. Their ECG tracings were analyzed by 2 observers, who were blinded to the CMR, for detection and location of MI following to the Fourth Brazilian Society of Cardiology Guidelines on the Analysis and Issuance of Electrocardiographic Reports. The significance level adopted was 5% ($p < 0.05$).

Results: ECG showed good performance for detecting previous MI, with sensitivity of 69.3% (64.5% to 74.1%), specificity of 99.1% (98.1% to 100%), and accuracy of 78.7% (74.4% to 83.0%). However, in locating MI in accordance with CMR, its accuracy was unsatisfactory.

Conclusions: When compared to CMR, ECG was shown to be a method with good accuracy for detecting previous MI, but not for defining its location.

Keywords: Myocardial Infarction; Coronary Artery Disease; Electrocardiography; Magnetic Resonance Imaging.

Introduction

Coronary artery disease (CAD) continues to be one of the main diseases of the twenty-first century due to its high morbidity and mortality,[1] and it is the leading cause of heart failure.[2]

Late gadolinium enhancement cardiovascular magnetic resonance imaging (LGE-CMR) has emerged as an important tool in the assessment of ischemic heart disease,[3,4] and it is able to provide diagnostic and prognostic information that is superior to other methods in different contexts.[5-8] With high accuracy, LGE-CMR identifies even small infarctions,[7,9] in addition to several alternative diagnoses,[10,11] but its availability is limited, especially in the Brazilian context, in smaller municipalities, and within the public health system.

Myocardial infarction (MI) generally results in a series of electrocardiogram (ECG) changes involving the ST segment and the T wave and, in many cases, abnormalities in the initial portion of the QRS complex, known as pathological Q waves.[12] Recognition of old MI is difficult.[13] In a series of 100 patients with documented MI more than 7 days prior to undergoing CMR, Q waves were present in 28% of subendocardial MI cases and absent in 29% of transmural MI cases.[13] One study demonstrated Q wave regression after

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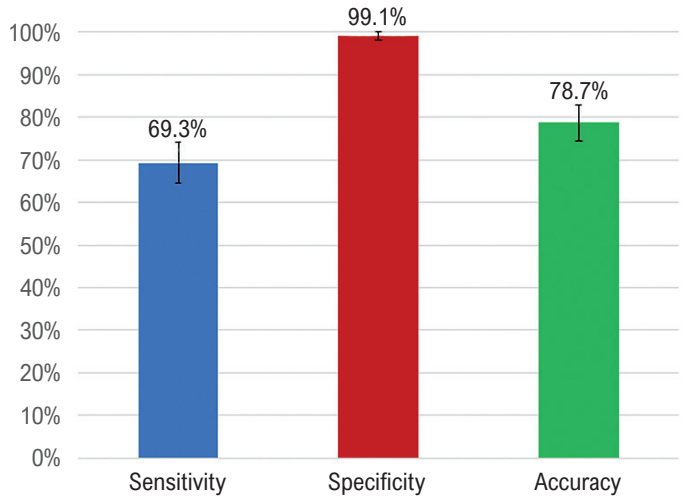
Central Illustration: Detection and Location of Myocardial Infarction Using Electrocardiogram: Validation by Cardiovascular Magnetic Resonance Imaging



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Performance of electrocardiogram in detecting myocardial infarction. Source: Produced by the author (2024).

MI in a substantial proportion of cases (42% and 13% of 127 men had total and partial regression, respectively).[14]

The classic study by Myers et al. has, for the past 50 years, provided the basis for the widely accepted association between Q waves and the presence of myocardial scarring.[15] In addition to the limited sample size, these autopsy studies have a major limitation for locating infarction; during autopsy, the heart is evaluated outside the thorax, in a position different from its usual orientation within the thorax.[16]

ECG is a low-cost tool, and it is widely available in clinical practice for detecting patients with prior MI.[17] Despite its widespread clinical use, some studies published to date comparing ECG findings with the presence of ischemic myocardial necrosis on CMR have included a limited number of patients, and the majority of them have not shown satisfactory accuracy.[13,18] This study aimed to evaluate the diagnostic performance of ECG for detecting and locating CMR-defined MI in a sample from the Brazilian population.

Methods

This was a retrospective, observational, case-control study. The project was approved by the Research Ethics Committee of both Instituto do Coração da Faculdade de Medicina da Universidade de São Paulo (InCor) and Hospital do Coração de São Paulo (HCOR), who waived the requirement for participants to sign a consent form, because there would be no intervention, but only a study of data from medical records.

The sample of cases with previous infarction was obtained through retrospective analysis of electronic medical records of patients who were treated at Instituto do Coração da Universidade de São Paulo, from June 2014 to December 2019. The first analysis included all patients whose medical records reported diagnosis of acute MI (ICD I.21) and who underwent ECG and LGE-CMR after the date of the acute coronary syndrome (ACS), with an interval of up to 1 year between the exams.

The control sample (individuals without previous infarction) was obtained from another study by this group, entitled "Avaliação de disfunção microcirculatória a partir de biomarcadores metabólicos séricos em pacientes com diabetes mellitus" – ENDOCRINE (SDC UNEX 012/22/002),[19] conducted at one of the participating centers (HCOR) between 2018 and 2020. This was an observational study in a national cohort that included 51 healthy individuals and 258 diabetic patients, 108 of whom had had a previous acute myocardial infarction and the others had no clinical history suggestive of infarction. Follow-up was 6 months

From the ENDOCRINE[19] study sample, we included individuals who underwent ECG and LGE-CMR on the same day and who did not have fibrosis suggestive of previous infarction or any other abnormality on CMR, except mild left ventricular (LV) hypertrophy up to 13 mm. The reference value for normal wall thickness vary in different studies between 8.3 ± 1.0 cm and 10.2 ± 1.1 cm for males and 6.8 ± 0.9 cm and 9.2 ± 0.9 cm for females.[20-22]

This study excluded individuals with CMR results that did not have satisfactory image quality or with a diagnosis other than infarction (other cardiomyopathies, for example, hypertrophic cardiomyopathy, Chagas disease, myocarditis, amyloidosis, etc.) and patients with a record of a new ACS

between the ECG and the CMR. This study also excluded patients whose ECGs were only available during the acute phase of the infarction (up to 7 days between the MI and the ECG) and patients with pacemaker rhythm.

The presence of risk factors for coronary disease was defined by their description in the electronic medical records, either in outpatient medical assessments or during hospitalizations.

Digital images of the 12-lead ECG performed at rest with a tracing speed of 25 mm/sec were extracted from electronic medical records of the cases and controls. ECGs were analyzed manually by 2 cardiologists specialized in electrocardiography with more than 20 years of experience, both of whom were blinded to the CMR results. When there was disagreement between the observers, the ECG was evaluated by a third specialist.

Infarction was detected in accordance with the Fourth Brazilian Society of Cardiology Guidelines on the Analysis and Issuance of Electrocardiographic Reports.[23] Presence of infarction was defined as the existence of pathological Q waves in 2 contiguous leads, with a duration equal to or greater than 40 ms, whether or not it was associated with amplitude > 25% of the entire QRS.[12,23]

All LGE-CMR examinations were performed on 1.5-T equipment specifically designed for cardiovascular applications (Supplemental Material). Two pulse sequences were used:

a) Cine imaging with balanced steady-state free precession (b-SSFP), currently considered the "state of the art" for assessing ventricular function, volumes, and mass.[24,25] This sequence allows acquisition of dynamic images with a temporal resolution of 50 ms or less, with excellent contrast between the cardiac chambers and the blood.

   Images were acquired in long and short axes, covering both ventricles. Short-axis slices were acquired in numbers from 8 to 14, aiming to cover the entire extension of the LV. Long-axis slices were planned from short-axis images as follows: 2 in 2-chamber view, 1 in 4-chamber-view, and 1 in 3-chamber view, evaluating the LV outflow tract.

b) Late gadolinium enhancement (LGE): This technique allows the identification of areas of fibrosis (old scar) or necrosis (acute and irreversible myocyte injury) within the LV myocardium with great precision, resolution, and anatomical detail.[9]

   Images were acquired during respiratory pauses and coupled to the ECG, in 4 chambers, LV short and long axes, in the same locations as the cine sequences. This allowed a better comparison between regional cardiac function and myocardial structure.

CMR was analyzed by a cardiovascular imaging specialist with more than 4 years of experience in the area, who was blinded to the ECG. Doubtful cases were evaluated by a second senior evaluator with more than 25 years of experience in CMR in order to obtain measurement results by consensus.

Total fibrosis mass and its percentage in relation to LV mass were assessed. Planimetry of the LGE area and the total area of the segment was performed in each of the 17 segments, following the American Heart Association (AHA) myocardial segmentation model,[26] obtaining the percentage of fibrosis in each of them, which was categorized according to extent in relation to the area of the segment as follows: 0 (absent), 1 (1% to 25%), 2 (26% to 50%), 3 (51% to 75%), and 4% (76% to 100%).

The myocardial fibrosis mass of each segment and the total mass were quantified using the full width at half maximum (FWHM). Transmural extent was defined as myocardial fibrosis greater than 50% of the area of the LV myocardial segment[27] in at least 1 of the 17 segments standardized according to the AHA model.[26] Postprocessing of the images, with morphofunctional analysis and myocardial viability, was performed using CVi 42 commercial software, which has been validated by the Brazilian Health Regulatory Agency (ANVISA, acronym in Portuguese). Additionally, the AHA global fibrosis score was calculated by adding the value of the LGE extent category (0 to 4) of each of the 17 segments divided by 68 (number corresponding to the maximum score of 4 for all 17 segments), resulting in the value of fibrosis mass as a percentage of LV mass.

In order to compare the ECG with CMR (gold standard) regarding ability to correctly locate the infarction, it was necessary to establish simplified locations in 3 categories that correspond to usual coronary territories (Table 1). When the infarction was extensive anterior, the main simplified location was defined as anterior and/or septal and/or apical and the secondary as lateral.

The sum of the LGE scores of each segment that makes up the respective wall was obtained in order to define the main, secondary, and, eventually, tertiary locations of the infarction, in decreasing order of the result of this sum. When there was a tie in the sum of the LGE scores in 2 walls, the main location of the infarction was arbitrarily defined in the following order: anterior and/or septal and/or apical, followed by inferior, and, finally, lateral. When there was only 1 point in a single segment of a given location, the presence of infarction in that location was not considered, except when it was the only fibrosis detected on CMR.

Statistical analysis

Frequency distribution for categorical variables was used to characterize the cases of MI diagnosed by CMR; for continuous variables, the Kolmogorov-Smirnov normality test was applied. For variables with normal distribution, the means and respective standard deviations were calculated; for variables with non-normal distribution, the medians and interquartile ranges were shown.

To compare the groups of patients with MI and controls regarding clinical and demographic characteristics, habits, comorbidities, and ECG and CMRI parameters, Student's t test for independent samples was applied to compare means, and the Mann-Whitney test was used to compare medians. Pearson's chi-square test was used to compare frequencies. When the expected frequency of any of the categories was below 5, Fisher's exact test was used.

The main response variable for CMR was LGE, and its extent in relation to the area of the segment was analyzed by categories (0 to 4). Considering diagnosis of MI by CMR, an ECG validation analysis was performed, estimating sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV), as well as the overall accuracy of the tests. For all measurements, 95% confidence intervals (CI) were presented.

ECG validation was also analyzed with stratification by fibrosis mass and number of segments with transmural LGE. Receiver operating characteristic curves were constructed to assess ECG accuracy.

To analyze interobserver agreement for detection and location of MI by ECG, the kappa coefficient was estimated with the respective 95% CI. The significance level adopted was 5% ($p < 0.05$), and the CI were set at 95%. STATA statistical software, version 14 was used for all analyses.

Results

This study assessed 2,784 patients with previous infarction and 258 individuals without infarction for eligibility. After applying the exclusion criteria, 241 cases and 111 controls were included. Figures 1 and 2 display flowcharts of case and control selection. In the patients for whom CMR revealed another diagnosis, the most frequent among cases were myocarditis (n = 17), hypertrophic cardiomyopathy (n = 8), and Chagas disease (n = 6). The characteristics of the sample are detailed in Table 2.

The event preceding the analyzed CMR and ECG was ST-segment elevation MI in 52.7% of cases and non-ST-segment elevation MI in 14.5% of cases; in 32.8% of the sample, there was no clear clinical presentation of ACS, or it was not registered in the medical records. The median fibrosis mass was 22.6% of the LV mass, and the number of segments with transmural LGE was 4 (2 to 6). The main location of the infarction was anterior and/or septal and/or apical in 67.2%, inferior in 21.0%, and lateral in 11.5% of cases. The majority of cases presented involvement of 2 or more simplified locations on CMR (85.1%).

Infarction detected by ECG

ECG showed good performance for detecting MI, with sensitivity of 69.3% (64.5 - 74.1), specificity of 99.1% (98.1 - 100), and accuracy of 78.7% (74.4 - 83.0), with no significant difference between observers (Central Illustration). In the population studied, the PPV was 99.4% (98.6 - 100), and the NPV was 59.8% (54.7 - 64.9). The sensitivity of ECG for detecting MI varied according to size, assessed by tertiles of fibrosis mass and number of segments with transmural LGE on CMR (Figure 3).

When stratifying the fibrosis mass by the median (22.6%), the sensitivity of ECG in detecting MI was 55.5% (46.5 - 64.4) and 82.8% (76.1 - 89.5) for fibrosis masses up to the median and above ($p < 0.001$). For the median number of segments with transmural LGE, the difference in ECG sensitivity was also significant, namely, 54.8% (46.1 - 63.6) versus 84.6% (78.1 - 91.2) for the strata up to the median and above ($p < 0.001$).

Table 1 – Simplified location of myocardial infarction on ECG and CMR

Simplified location	
ECG	
Anterior and/or septal e/ou apical	Q in V1, V2, V3, V4
Lateral	Q in V5, V6, D1, AVL
Inferior	Q in D2, D3 e AVF
LGE-CMR	
Anterior and/or septal e/ou apical	Segments 1, 2, 7, 8, 13, 14, 15, 16, and 17
Lateral	Segments 5, 6, 11, and 12
Inferior	Segments 3, 4, 9, and 10

ECG: electrocardiogram; CMR: cardiovascular magnetic resonance; LGE: late gadolinium enhancement. Source: Produced by the author (2024).

The association between MI size on CMR and accuracy of ECG in detecting MI was strong (Figure 4). The fibrosis mass with the best sensitivity-to-specificity ratio was 15.5% of the LV mass, with a sensitivity of 83.2% (76,7 - 88,2) and specificity of 83.7% (77.6 - 88.4).

ECG's performance in detecting MI in any wall was also evaluated according to the MI groups on CMR (anterior and/or septal and/or apical, lateral, and inferior) (Figure 5). Figure 6 shows an example in which the ECG correctly detected and located an inferior infarction. Figure 7 shows a false negative, in which there were no pathological Q waves (> 40 ms) in at least 2 ECG leads, and CMR revealed an inferior and lateral MI and large fibrosis mass (30.3%).

ECG's performance in detecting MI was analyzed according to the time between the date of the MI and the date of the ECG, stratified by the median (150 days), and there was no difference in sensitivity between the periods: 73.9% (65.7 - 82.0) for MI less than 150 days before the ECG and 65.4% (57.2 - 73.6) for MI 150 days or more ($p = 0.154$).

Infarction location on ECG

ECG had limited performance in correctly identifying the main location of the MI, considering CMR as a reference, especially for lateral infarctions followed by inferior infarctions (Table 3).

Considering that 85.1% of the cases had LGE involving more than one simplified MI location and that the sum of the LGE scores between the main and secondary locations were similar in many cases, the performance of ECG in locating MI was evaluated by considering it correct whenever the main location on the ECG was the same as the main or secondary location on the CMR (Table 3).

The differences in sensitivity of ECG for locating MI were statistically significant for comparisons with the lateral wall.

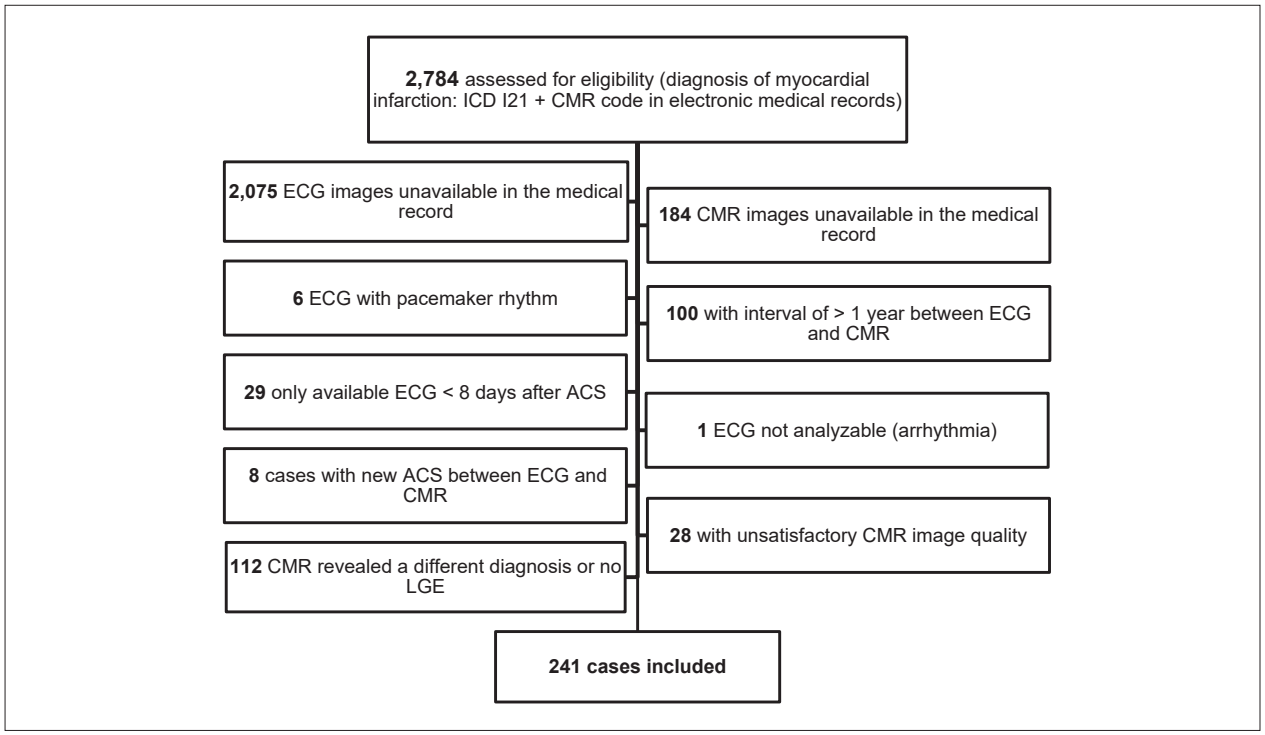

Figure 1 – *Inclusion flowchart (cases). ACS: acute coronary syndrome; CMR: cardiovascular magnetic resonance; ECG: electrocardiogram; LGE: late gadolinium enhancement. Source: Produced by the author (2024).*

Interobserver agreement analyses

Interobserver agreement in detecting MI was good, with a kappa coefficient of 0.75 (95% CI: 0.64 - 0.85). Interobserver agreement for the main location of the infarction was only fair, with a kappa of 0.39 (0.32 - 0.45).

The agreement between ECG and CMR regarding the main location of MI was minimal, with a kappa coefficient of 0.24 (95% CI: 0.18 - 0.29).

Discussion

The original characteristics of our study were evaluation of the accuracy of ECG in detecting and locating MI outside the acute phase, including controls without infarction in a sample of the Brazilian population, using LGE-CMR as a reference. One of the advantages of LGE-CMR is its ability to assess the presence of subendocardial infarction and delineate the transmural extent of MI with high spatial resolution, which allows the detection of small infarctions that are often not identified by other methods.[7,9]

It is important to highlight that, for the purposes of statistical analysis, ECGs were classified according to the presence or absence of infarctions by observers blinded to the CMR results and clinical data of the cases. However, in clinical practice, there is a wide range of doubtful cases, and physicians always have additional information to assist in decision-making, for example, previous ECGs, information from other complementary exams, the presence of risk factors, and, especially, detailed clinical history and physical examination.

The sample of cases consisted mostly of relatively extensive infarctions (median fibrosis mass of 22.6%), whose locations were compatible with involvement of the left anterior descending artery, and slightly more than half of the cases had LV ejection fraction below 40%.

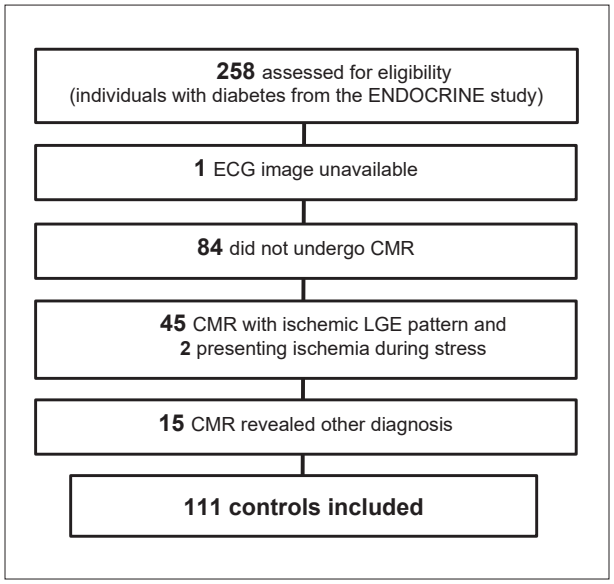

Figure 2 – *Inclusion flowchart (controls). CMR: cardiovascular magnetic resonance; ECG: electrocardiogram; LGE: late gadolinium enhancement. Source: Produced by the author (2024).*

Table 2 – Sample characteristics

Characteristics	Cases (n = 241)	Controls (n = 111)	p value
Age (mean ± SD)	59.2 ± 10.9	54.1 ± 15.9	<0.001
Sex			
Female	69 (28.6%)	73 (65.8%)	<0.001
Male	172 (71.4%)	38 (34.2%)	<0.001
BMI (mean ± SD)	28.0 ± 5.4	29.2 ± 5.7	0.060
Obesity (BMI ≥ 30 kg/m²)	72 (30.0%)	43 (38.7%)	0.105
Diabetes mellitus	85 (35.3%)	111 (100%)	<0.001
Hypertension	165 (68.5%)	75 (67.6%)	0.825
Prior or active smoking	139 (57.7%)	49 (44.1%)	0.001
LVEF	37 (27 – 47)*	67 (62 – 72)*	<0.001
LVEF < 40%	141 (58.5%)	0 (0%)	<0.001
LV mass (g/m²)*	68.8 (60.2 – 79.8)*	57.2 (48.9 – 66.1)*	<0.001
Time between CMR and ECG (in days)	42 (7 – 118)	0 (0 – 0)	<0.001

Asterisks indicate values expressed as median (interquartile range). BMI: body mass index; CMR: cardiovascular magnetic resonance; ECG: electrocardiogram; LV: left ventricular; LVEF: left ventricular ejection fraction; SD: standard deviation. Source: Produced by the author (2024).

Jaarsma et al. compared the findings of ischemic LGE-CMR with ECG scores in 78 patients, 3 months after myocardial infarction with ST-segment elevation, and 36 controls without any structural heart disease on CMR.[28] The sensitivity of the third universal definition of infarction proposed by the ESC/ACC/AHA/World Heart Foundation in 2012, the Minnesota ECG code, Selvester score, and subjective assessment by cardiologists for detection of previous MI was 33%, 79%, 90%, and 67%, and accuracy was 54%, 77%, 71%, and 74%, respectively. The authors concluded that caution should be exercised when ruling out previous MI based solely on ECG findings, which also seems important. In our study, with a larger sample, ECG showed similar accuracy.

We found a strong association between MI size on CMR and the accuracy of ECG in detecting it, both in the assessment by percentage of fibrosis mass and fibrosis score on CMR (area under the curve of 0.9 for both). The greatest limitation of ECG was for smaller infarctions, where the sensitivity of ECG was less than 60%. These data reflect the limitations of tools for detecting infarctions on ECG based on the identification of pathological Q waves.

Although previous studies have associated a higher risk of recurrent ischemia and reinfarction with infarction without Q waves,[29,30] Phibbs et al. highlighted biases in these studies, especially due to the combined inclusion of patients with first infarction and subsequent infarctions, who present different mortality and morbidity. In an extensive review of well-conducted studies, these authors demonstrated similar prognoses for infarctions with and without the appearance of pathological Q waves.[31]

Although Q waves are frequently associated with transmural MI, pathology studies have shown that their presence on ECG was not able to differentiate transmural

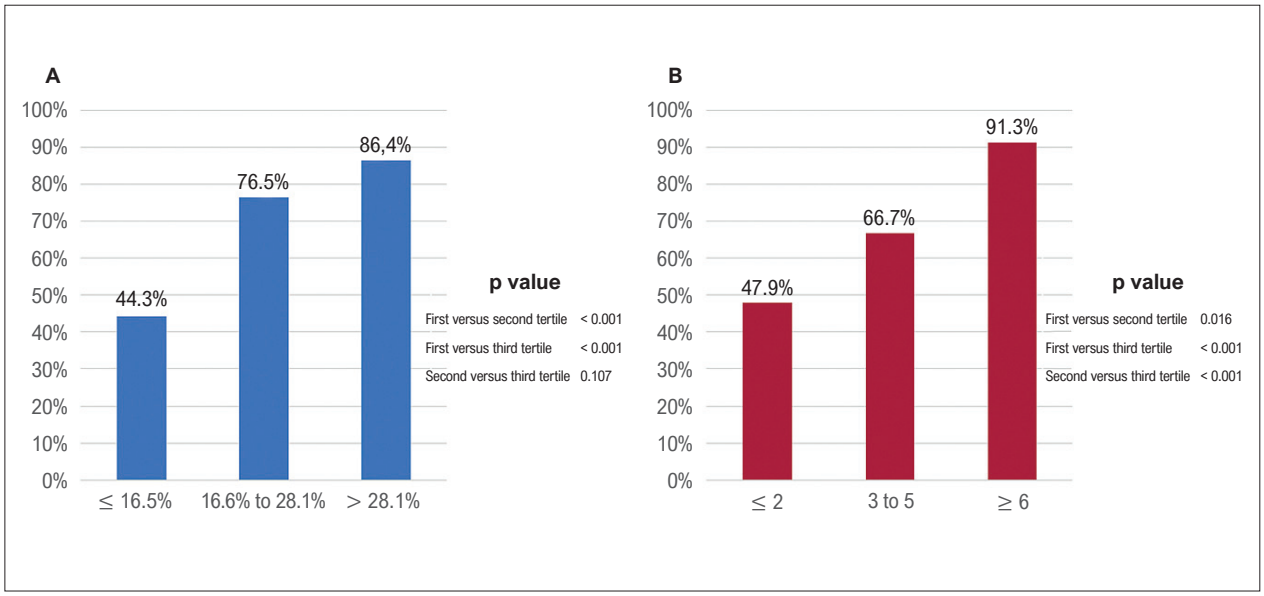

Figure 3 – *Sensitivity of electrocardiogram in detecting myocardial infarction according to fibrosis mass percentage tertiles (A) and number of segments with transmural late gadolinium enhancement (B) on cardiovascular magnetic resonance. Source: Produced by the author (2024).*

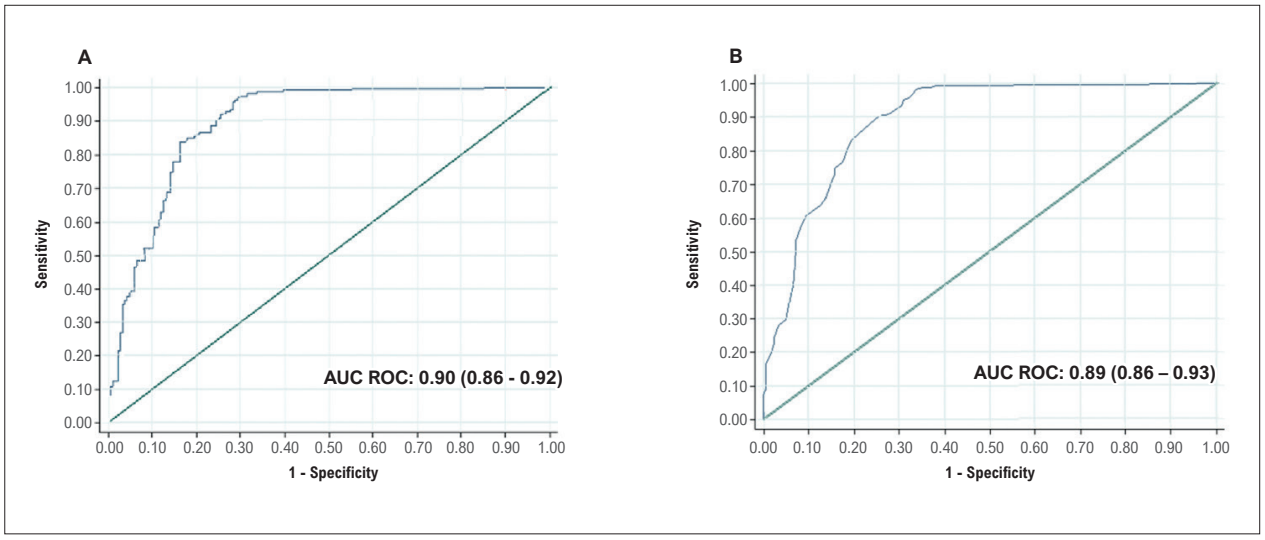

Figure 4 – *Accuracy of electrocardiogram methods in detecting myocardial infarction according to fibrosis mass percentage (A) and global fibrosis score (B). AUC ROC: area under the receiver operating characteristic curve; ECG: electrocardiogram. Source: Produced by the author (2024).*

from nontransmural scars.[32,33] Another study compared the results of resting and stress rubidium-82 perfusion positron emission tomography and F-18 fluorodeoxyglucose metabolic positron emission tomography imaging in identifying infarction with and without Q waves in patients with LV dysfunction, demonstrating a significantly greater total amount of ischemic viable myocardium in those without Q waves (6.5 ± 5.2 versus 2.9 ± 2.8 segments, $p < 0.001$), with no significant differences in LV ejection fraction between the groups.[34]

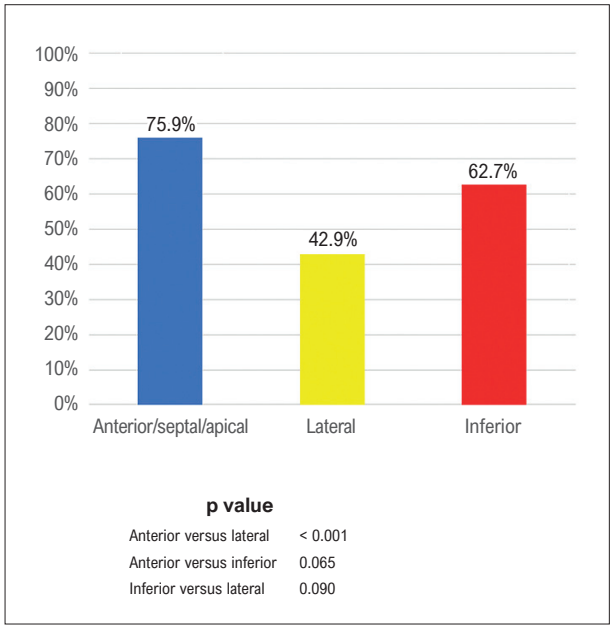

Figure 5 – *Sensitivity of electrocardiogram in detecting myocardial infarction defined by cardiovascular magnetic resonance. Source: Produced by the author (2024).*

More recently, the performance of the fourth universal definition of infarction criteria for detecting MI was tested in a single-center study from the Netherlands that evaluated ECGs of 974 patients undergoing CMR (205 with ischemic LGE pattern and 769 without LGE). The authors found low sensitivity of 38% (95% CI: 31.6 - 44.8) and specificity of 86.9% (95% CI: 84.4 - 89.1).[35] In the analysis of cases stratified into 2 locations, the anterior location had much higher sensitivity than the inferior location, which included the inferior, inferolateral, inferoseptal, and lateral segments (63.3% versus 20.2%). The increase in size, assessed by the number of segments involved, or in the transmural extent of the infarction was not associated with improved ECG sensitivity, contrary to what we observed in our study.

On the other hand, in our study, which included a greater number of cases but a smaller number of controls, the sensitivity and specificity of ECG were higher. The authors of the study conducted in the Netherlands[35] emphasized that the inclusion of all patients undergoing CMR during the selection period for various indications resulted in heterogeneity that may justify differences compared to the results of other populations, for example, patients with suspected CAD, such as ours, where cases underwent CMR after a clinical diagnosis of MI. Moreover, in the aforementioned article, the fibrosis mass among patients with infarction was not described, which may also have impacted the difference in sensitivity found. In our analysis, the lateral and inferior walls were assessed separately, and sensitivity was low for the lateral wall, but the values for the inferior location were close to the sensitivity of the anterior wall.

The performance of ECG in locating MI compared to CMR was limited, especially for the lateral wall. Previous publications that studied the location of prior infarction by means of leads where pathological Q waves are found had limited samples and low statistical power to assess the accuracy of ECG in locating MI.[15,16,36] Larger studies that

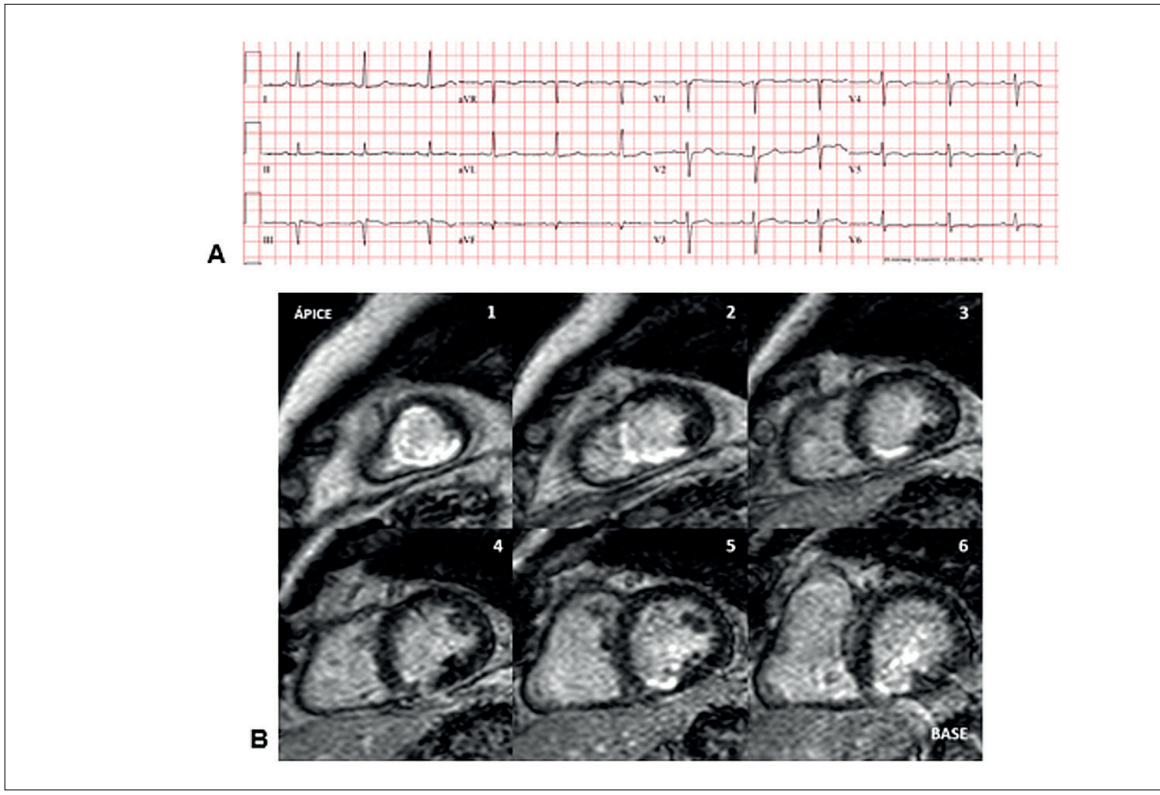

Figure 6 – *Example of a case in which the electrocardiogram (A) correctly detected and located an inferior myocardial infarction. Cardiovascular magnetic resonance (B) estimated the myocardial fibrosis mass at 17.2%. Source: Produced by the author (2024).*

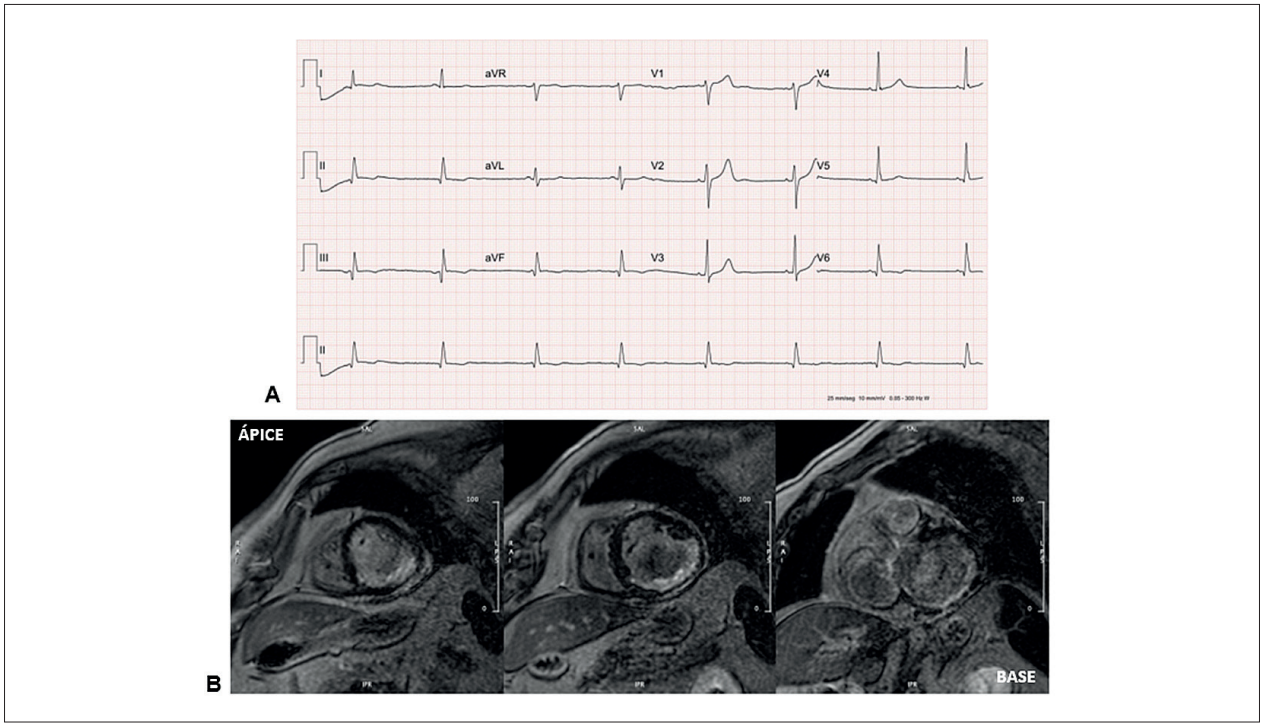

Figure 7 – *Example of a case in which the electrocardiogram (A) did not meet the criteria for detecting myocardial infarction, and cardiovascular magnetic resonance (B) revealed inferior and lateral myocardial infarction and fibrosis mass of 30.3%. Source: Produced by the author (2024).*

Table 3 – Sensitivity of ECG in locating CMR-defined MI (cases)

Location of MI on CMR	If primary location of MI on ECG coincided with primary location	If primary location of MI on ECG coincided with primary or secondary location on CMR
	(95% CI)	(95% CI)
Anterior and/or septal and/or apical	53.8% (46.7% – 60.8%)*	61.5% (53.7% – 68.7%)*
Lateral	0%	0%
Inferior	40.0% (22.0% – 61.2%)*	45.1% (31.7% – 59.2%)*
p value		
Anterior versus lateral	<0.001	<0.001
Anterior versus inferior	0.193	0.039
Inferior versus lateral	0.010	<0.001

Asterisks indicate values expressed as median (interquartile range). CI: confidence interval; CMR: cardiovascular magnetic resonance; ECG: electrocardiogram; MI: myocardial infarction. Source: Produced by the author (2024).

analyzed the performance of ECG in diagnosing MI using CMR as a reference did not describe its accuracy in locating the myocardial scar, due to the lack of a control group.[37,38]

Silent infarctions are not rare, and they have a negative impact on prognosis.[39,40] A prospective cohort study from the Netherlands performed serial ECGs on more than 6,000 individuals over 55 years of age. In a mean follow-up of 13.2 years, the prevalence of silent infarctions, defined by the appearance of new Q waves on the ECG without a clinical history compatible with ACS, was 5.8% in men and 4.5% in women.[39] In another study, silent infarctions accounted for 9.4% of events, with a cardiovascular mortality rate of 10.7% in 3 years.[40] In another study, in type 2 diabetics, 36.8% of infarctions were silent.[41] In almost one third of the cases in our sample, there was no clear clinical presentation of ACS, or it was not registered in the medical records, with the diagnosis of previous infarction being made by CMR during the etiological investigation of heart failure.

Limitations

The retrospective study design has multiple limitations, including variability in the quality of clinical data. Despite the widespread use of CMR for assessment after MI in the service where the cases originated, in a very high number of patients assessed for eligibility, no digital image of the ECG was available in the electronic medical records, only the written report.

It is also important to highlight the possibility of measurement bias; since this was a study of MI, the assessors may have been more likely to identify changes in the exams, which may have increased the sensitivity of the ECG.

Another limitation was the small number of MI cases with a mainly lateral location.

A difference worth highlighting was that all controls had diabetes, while the prevalence of diabetes among cases was 35.3%. Although diabetes is an important risk factor for CAD, and the occurrence of silent infarctions in this population is higher,[42] the fact that the controls underwent CMR and ECG on the same day rules out the possibility of an acute coronary event occurring between the exams. Moreover, the controls' CMR included assessment of myocardial ischemia with pharmacological stress, making it able to detect not only infarctions, but also ischemia, which also led to the exclusion of controls. We included only those who did not present any alteration in the exam, except minimal LV hypertrophy of up to 13 mm maximum parietal thickness.

Clinical data, for example, duration and intensity of symptoms, medication use, and occurrence of cardiovascular events were not evaluated; moreover, coronary anatomy and interventions performed were not analyzed. The ECG tracings were obtained at different institutions and in different sectors of both centers included, and there is no description of the ECG device used in the majority of them.

Finally, the study involved ECG and CMR data acquired in only 2 institutions, limiting the external validity of our results, which should be confirmed in a large, multicenter, population-based study.

Conclusions

- ECG was shown to be a method with good accuracy for detecting previous MI when compared to LGE-CMR: 78.7% (74.4 - 83.0).
- ECG's performance in locating the infarction showed very limited usefulness, especially in the lateral wall.
- As ECG is a low-cost tool and the most widely available in the assessment of CAD, it is important to validate its sensitivity compared to LGE-CMR in detecting MI, especially in populations with more limited access to higher-cost tests, which is the case in many Brazilian cities. The data from this study may provide a basis for further studies on the applicability of ECG in screening patients who will require additional diagnostic investigation for CAD.
- Considering the sensitivity of ECG in detecting prior MI, based on the identification of pathological Q waves (69.3%), the identification of additional ECG criteria capable of improving ECG performance in the context of chronic CAD would be of great value.

Author Contributions

Conception and design of the research: Guerra MCMD, Chalela WA, Uchida AH, Santos ECL, Cintra RA, Ramires JAF, Rochitte CE; Acquisition of data: Guerra MCMD, Fonseca RA,

Cintra RA; Analysis and interpretation of the data: Guerra MCMD, Rezende AGS, Magalhães TA, Chalela WA, Uchida AH, Fonseca RA, Heringer Filho N, Beuther J, Garcia G, Rochitte CE; Statistical analysis: Guerra MCMD, Montarroyos UR, Rochitte CE; Writing of the manuscript: Guerra MCMD, Rochitte CE; Critical revision of the manuscript for content: Magalhães TA, Rochitte CE.

Potential conflict of interest

No potential conflict of interest relevant to this article was reported.

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Study association

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Ethics approval and consent to participate

This study was approved by the Ethics Committee of the Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo – HCFM/USP and Hospital do Coração/Associação Beneficente Síria – HCOR under the protocol number 6,016,574 and 6,314,851, respectively. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013.

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*Supplemental Materials

For additional information Supplemental Material, please click here.