

Electrocardiogram and CMR to Differentiate Tachycardia-induced Cardiomyopathy From Dilated Cardiomyopathy in Patients Admitted for Heart Failure

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Abstract

Purpose

In patients admitted for heart failure (HF) with reduced left ventricular dysfunction and a concomitant supraventricular tachyarrhythmia (SVT) it is a challenge to predict LVEF recovery and differentiate tachycardia-induced cardiomyopathy (TIC) from dilated cardiomyopathy (DC). The role of cardiac magnetic resonance (CMR) and the electrocardiogram (ECG) in this acute setting remains unsettled.

Methods

Forty-three patients admitted for HF due to SVT and LVEF<50% undergoing CMR in the acute phase were retrospectively included. Those who had LVEF>50% at follow up were classified as TIC and those with LVEF<50% were classified as DC. Clinical, CMR and ECG findings were analyzed to predict LVEF recovery.

Results

Twenty-five (58%) patients were classified as TIC. Patients with DC had wider QRS (121.2 ± 26 vs 97.7 ± 17.35 ms; $p=0.003$). On CRM the TIC group presented with higher LVEF (33.4 ± 11 vs $26.9\pm 6.4\%$; $p=0.019$) whereas late gadolinium enhancement (LGE) was more frequent in DC group (61 vs 16%; $p=0.004$). On multivariate analysis, QRS duration ≥ 100 ms ($p=0.027$), LVEF<40% on CMR ($p=0.047$) and presence of LGE ($p=0.03$) were independent predictors of lack of LVEF recovery. Furthermore, during follow-up (median 60 months) DC patients were admitted more frequently for HF (44% vs 0%; $p<0.001$) than TIC patients.

Conclusion

In patients with reduced LVEF admitted for HF due to SVT, QRS ≥ 100 ms, LVEF<40% and LGE are independently associated with lack of LVEF recovery and worse clinical outcome.

Background

Supraventricular tachyarrhythmia (SVT) may trigger a reversible dilated cardiomyopathy referred as tachycardia-induced cardiomyopathy (TIC) (1). TIC is confirmed when left ventricular ejection fraction (LVEF) recovery is observed after rate control. However, since SVT is also a common finding in patients with dilated cardiomyopathy (DC) (2), a prompt differentiation of TIC from DC in patients admitted for heart failure (HF) with both high rate SVT and reduced LVEF remains problematic. The challenge is to determine whether the high-rate arrhythmia is responsible or not for the observed left ventricular dysfunction (chicken-egg dilemma) (3). Some echocardiographic and biochemical predictors of TIC have been previously described (4, 5). However, the combined role of electrocardiogram (ECG) and cardiac magnetic resonance (CMR) to differentiate these two clinical entities in the acute setting is scarce. The aim of this study is to assess the value of ECG and CMR as predictors of LVEF recovery in patients admitted for HF with reduced LVEF and SVT.

Methods

Study sample

From 2008 to 2018, we recruited all consecutive patients admitted for a first episode of HF with SVT (defined as non-sinus SVT with a resting heart rate >100 beats/min) and LVEF <50% undergoing CMR in the acute phase. Clinical, laboratory, ECG, echocardiographic and CMR data were collected at recruitment. Patients were systematically followed.

Electrocardiogram

An ECG was performed on admission in the emergency department in all patients. ECG was interpreted by cardiologists according to the guidelines (6).

Echocardiography

All patients underwent echocardiography on admission and 24 months after by cardiologists accredited on echocardiography. Assessments were performed according to the guidelines of the American Society of Echocardiography and European Association of Cardiovascular Imaging using 2-dimensional acquisitions (7, 8). Those who had -after heart rate normalization and excluding other reversible underlying causes- LVEF >50% at 24 months follow up were classified as TIC and those who had LVEF <50% were classified as DC. We compared both groups to detect lack of ejection fraction recovery predictors.

Cardiac magnetic resonance

All patients underwent CMR in the acute phase (within 3 months from index admission) after controlling the heart rate. CMR were performed on a clinical 1.5-T MRI scanner (Signa HDxt; General Electric Healthcare®, Milwaukee, WI, USA).. Four, three and two chamber cine images as well as short axis cine slices were acquired using SSFP sequences. Short axis cine slices were used to trace LV and RV endocardial and epicardial contours on the end-systolic and end-diastolic frames to quantify mass, volumes and EF. Those parameters were measured by accredited CMR experts using dedicated software. (CVI42, Circle Cardiovascular Imaging, Calgary, Alberta, Canada). LGE was obtained using a T1-weighted inversion recovery gradient echo technique in both long- and short-axis views 10-15 min after a bolus (0.2 mmol/kg to a maximum of 20 mmol) of gadolinium-diethylenetriamine pentaacetic acid (Magnevist, Bayer Schering, Berlin, Germany) to identify regional fibrosis, Inversion time was adequated for optimal nulling myocardium. LGE was quantified manually using dedicated software (CVI42, Circle Cardiovascular Imaging, Calgary, Alberta, Canada).

Outcomes:

Our primary outcome was lack of LVEF recovery at 2 years follow-up. Secondary outcome was readmission for HF.

Statistical analysis

Qualitative variables are expressed as frequency (percentage). Quantitative variables are expressed as mean \pm standard deviation. To compare qualitative variables we used Chi square or Fisher test as appropriate. Kolmogorov-Smirnov test was performed in quantitative variables to test normality. According to that, parametric or non-parametric tests were used to compare quantitative variables. Statistically significant quantitative variables were categorized using receiver-operating characteristic (ROC) curves choosing values with best sensitivity and specificity to detect lack of EF recovery. Significant variables were assessed in an univariate and multivariate binary logistic regression analysis. Incremental prognostic value of multivariate model above individual significant variables was defined as an increase in the area under the ROC (AUROC) curve. Readmission for heart failure analysis was performed using Kaplan-Meier curves. All analyses were conducted by using SPSS version 20 (IBM SPSS Statistics, IBM Corporation).

Results

Patient characteristics

A total of 43 patients were included in the study. Of these, 25 (58%) were classified as TIC. Twenty three percent of patients were female. Mean age was 61 years.

Clinical and laboratory associations with LVEF recovery

Clinical and biochemical characteristics on admission are expressed in Table 1. Patients in the DC group trended to be older (65.6 ± 11.5 years vs 58.8 ± 10.5 years; $p=0.052$). There were no differences in cardiovascular risk factors between the two groups. Likewise, there were no differences in other comorbidities such as chronic kidney disease, stroke or chronic obstructive pulmonary disease. Previous use of angiotensin-converting-enzyme inhibitors (ACEI) and betablockers (BB) were similar in DC and TIC groups. Previous history of atrial fibrillation (AF) was similar in both groups. Troponin T, NTproBNP and other biochemical markers were similar in both groups. Twenty-one patients (49%) underwent coronary angiography on admission. None of the patients had significant coronary artery disease to justify the LV systolic dysfunction. Coronary artery disease was ruled-out in the rest of patients with non-invasive techniques.

Table 1

Clinical and biochemical characteristics on admission

| | TIC (n=25) | DC (n=18) | P value |
|---|-------------|-------------|---------|
| Age (years) | 58.8± 10,6 | 65.6±11.5 | 0.052 |
| Female | 6 (24%) | 4 (22%) | 1 |
| Hypertension | 11 (44%) | 8 (44%) | 1 |
| Diabetes | 3 (12%) | 7 (39%) | 0.062 |
| Dyslipidemia | 8 (32%) | 6 (33%) | 1 |
| Previous tobacco use | 14 (56%) | 14(78%) | 0.199 |
| CKD (GFR < 60 ml/min/1.73m ²) | 2 (8%) | 0 (0%) | 0.502 |
| COPD | 1 (4%) | 1 (5.5%) | 1 |
| Previous Stroke | 0 (0%) | 1 (5.5%) | 0.419 |
| Previous AF | 8 (32%) | 7 (39%) | 0.750 |
| Previous ACEI use | 4 (16%) | 6 (33%) | 0.275 |
| Previous beta-blocker use | 2 (8%) | 5 (28%) | 0.110 |
| Previous thyroid pathology | 2 (8%) | 1 (5.5%) | 1 |
| CHA ₂ DS ₂ -VASc | 2±1 | 2,5±1.58 | 0.108 |
| Hemoglobin (g/dl) | 14.96±1.94 | 14.06±1.25 | 0.075 |
| T troponin (ng/L) | 31.54±28.97 | 26.14±9.4 | 0.636 |
| NTproBNP (pg/ml) | 3684±5320 | 4124±4319 | 0.580 |
| TSH (mU/L) | 2±1.6 | 1.58±0.85 | 0.405 |
| Sodium (mEq/L) | 141±3.14 | 140±3 | 0.623 |
| Creatinine clearance (mil/min) | 72.88±19.61 | 72.88±13.72 | 1 |
| ACEI= angiotensin-converting-enzyme inhibitors; AF=atrial fibrillation; CKD=chronic kidney disease; COPD= chronic obstructive pulmonary disease; DC=Dilated cardiomyopathy ; TIC=tachycardia induced cardiomyopathy | | | |

Echocardiographic associations with LVEF recovery

Echocardiographic findings on admission were similar in both groups including LV volume, diameters and function, left atrial (LA) volume, pulmonary systolic pressure (PSP), any degree of mitral regurgitation

(MR) or right ventricular (RV) dysfunction (Table 2).

Table 2
Echocardiography findings on admission

| | TIC (n=25) | DC (n=18) | P value |
|--|--------------|-------------|---------|
| LA diameter (mm) | 47.54±6.36 | 47.2±6.8 | 0.876 |
| LA volume (ml) | 76.5±25.91 | 98.6±28.69 | 0.061 |
| LV end-diastolic volume (ml) | 145.14±47.13 | 157.1±35.11 | 0.413 |
| LV end-diastolic volume indexed (ml/m ²) | 71.94±19.1 | 84.35±20 | 0.084 |
| Dilated LV according indexed end-diastolic volume | 9 (36%) | 12 (67%) | 0.061 |
| LV end-diastolic diameter (mm) | 56.17±6.4 | 57±8.44 | 0.750 |
| LVEF (%) | 33.76±9.2 | 29.71±6.6 | 0.126 |
| Septum wall thickness (mm) | 10.5±1.35 | 10.13±1.99 | 0.482 |
| Pulmonary systolic pressure (mmHg) | 34.45±10 | 36.47±7.52 | 0.515 |
| E/E' | 9.38±3.14 | 11.56±2.9 | 0.138 |
| Mitral regurgitation | 16 (64%) | 10 (56%) | 0.757 |
| RV dysfunction | 9 (36%) | 9 (50%) | 0.348 |
| DC=Dilated cardiomyopathy; LA=left atrium ; LV=left ventricle; LVEF=left ventricular ejection fraction; RV=right ventricle; TIC=tachycardia induced cardiomyopathy | | | |

ECG and CMR associations with LVEF recovery

ECG and CMR findings are summarized in Table 3. Median time from HF admission to CMR imaging was 9 days without differences between both groups. According to ECG findings 20 patients (80%) in the TIC group presented AF and 5 showed atrial flutter (AFT) as SVT on admission whilst 17 (94%) patients showed AF and 1(6%) patient showed AFT in the DC group although these differences did not reach statistical significance. Patients with DC had wider QRS (121.22±26.78 ms vs 98.36±17.87 ms; p=0.003). There were no differences in other ECG findings such as pathologic Q waves, left ventricular hypertrophy and bundle branch block between groups. On CMR there were no differences in LV and RV volume between groups. However, LVEF was significantly higher in patients with TIC (32.96±11% vs 26.94±6% p=0.044); and presence of mid-myocardium LGE was more frequent in the DC group (61% vs 16%; p=0.004).

Table 3
Electrocardiography and cardiac magnetic resonance findings on admission

| | TIC (n=25) | DC (n=18) | P value |
|---|------------------|-----------------|---------|
| Electrocardiography findings | | | |
| AF/AFT | 20 (80%)/5 (20%) | 17 (94%)/1 (6%) | 0.375 |
| pathological Q waves | 2 (8%) | 0 (0%) | 0.502 |
| LV hypertrophy | 2 (8%) | 3 (16%) | 0.634 |
| BBB | 4 (16%) | 8 (44%) | 0.082 |
| RBBB | 2 (8%) | 3 (16%) | 0.634 |
| LBBB | 2 (8%) | 5 (28%) | 0.110 |
| QRS (ms) | 98.36±17.87 | 121.22±26.78 | 0.003 |
| CMR findings | | | |
| CRM LV end-diastolic diameter (mm) | 60.6±5.4 | 61.83±7.47 | 0.534 |
| CMR LV end-diastolic volume (ml) | 181.83±48.74 | 198.28±57.72 | 0.323 |
| Indexed CMR end-diastolic volume (ml) | 91.36±21.08 | 105.24±32.47 | 0.115 |
| CMR end-systolic volume (ml) | 124.21±48.62 | 146.83±50 | 0.151 |
| CMR LVEF (%) | 32.96±11 | 26.94±6 | 0.044 |
| CMR Septum wall thickness (mm) | 10.47±1.38 | 10.75±2.17 | 0.775 |
| CMR RV end-diastolic volume (ml) | 141.90±33.9 | 124.78±30.63 | 0.109 |
| CMR RV end-systolic volume (ml) | 87.48±30.14 | 75.17±24 | 0.172 |
| CMR RVEF (%) | 40.09±13.35 | 42.39±11.51 | 0.568 |
| LGE | 4 (16%) | 11(61%) | 0.004 |
| Mid-myocardium LGE | 100% | 100% | |
| AF=atrial fibrillation; AFT=Atrial flutter; BBB= Bundle branch block; CMR=cardiac magnetic resonance; DC=Dilated cardiomyopathy; LBBB= Left bundle branch block; LGE=late gadolinium enhancement; LV=left ventricle; LVEF=left ventricular ejection fraction; RBBB= Right bundle branch block; RV=right ventricle; RVEF=right ventricular ejection fraction; TIC=tachycardia induced cardiomyopathy | | | |

The values with best sensitivity and specificity to detect lack of EF recovery as determined by ROC curves were QRS ≥ 100 ms on ECG and LVEF $< 40\%$ on CMR. In the uni and multivariate analysis, a QRS duration ≥ 100 ms, a LVEF $< 40\%$ and presence of LGE were independently associated with lack of LVEF recovery (OR 6.41 [95% CI 1.23-33.44] p = 0.027, OR 13.8 [95% CI 1.03-184.75] p = 0.047 and OR 6.34 [95% CI 1.19-

33.63] p = 0.03, respectively) (Table 4) (Figure 1). The multivariate model predicted lack of ejection fraction recovery with a sensitivity of 83.3%, specificity of 71%, positive predictive value of 68% and negative predictive value of 85%.

Table 4
Uni and Multivariate analysis

| Univariate Binary Logistic Regression | | |
|---|---------------------|----------------|
| Lack of LVEF recovery | | |
| | OR (95% CI) | p value |
| QRS \geq 100ms | 6.22 (1.57-24.7) | 0.009 |
| LVEF<40% | 9.56 (1.08-84.24) | 0.042 |
| LGE | 7.86 (1.38-32.89) | 0.005 |
| Multivariate Binary Logistic Regression | | |
| | OR (95% CI) | p value |
| QRS \geq 100ms | 6.41 (1.2-33.4) | 0.027 |
| LVEF <40% | 13.80 (1.03-184.75) | 0.047 |
| LGE | 6.33 (1.19-33.73) | 0.03 |
| LGE=late gadolinium enhancement LVEF=Left ventricular ejection fraction | | |

To better delineate the magnitude and incremental value of the significant associations with lack of EF recovery ROC curves were generated. QRS performed best (AUROC curve of 0.78 p=0.002), followed by LGE (AUROC curve of 0.72 p=0,015) and LVEF on CMR (AUROC curve of 0.68 p=0,045). LGE and LVEF on CMR were then sequentially added to QRS and the final AUROC was 0.86 (Figure 2).

Treatment at discharge associations with LVEF recovery

Treatment at discharge is presented in (Table 1 Supplemental material). There were no differences in ACEI, beta-blockers and mineralcorticoid receptor antagonists (MRA) treatment at discharge between both groups. There were no differences in digoxin use at discharge. There were no differences in patients discharged at sinus rhythm.

Echocardiography findings at 24 months follow up

Table 5 shows echocardiography findings at 24 months follow-up between TIC and DC groups. Patients in the DC group showed higher LV end-diastolic volume (139 \pm 41.05 ml vs 107.90 \pm 47.13 ml p=0.024) higher LV indexed end-diastolic volume (70.3 \pm 18.9 ml/m² vs 52.32 \pm 12.44 ml/m² p=0.002) and LV end-diastolic diameter (54.18 \pm 3.82 mm vs 50.83 \pm 4.34 mm p=0.016). At 24 months follow up only 2 patients

in the TIC group presented LV dilatation whilst 9 patients in the DC group presented LV dilatation (8% vs 50% p=0.002). DC patients showed more frequently any degree of MR (67% vs 32% p=0.028).

Table 5
Echocardiography findings at 24 months follow-up

| | TIC (n=25) | DC (n=18) | P value |
|---|--------------|--------------|---------|
| LA diameter (mm) | 43.40±6.76 | 47.21±8.01 | 0.143 |
| LA volume (ml) | 79.33±34.87 | 106.62±61.68 | 0.106 |
| LV end-diastolic volume (ml) | 107.90±47.13 | 139±41.05 | 0.024 |
| LV end-diastolic volume indexed (ml/m ²) | 52.32±12.44 | 70.3±18.9 | 0.002 |
| LV dilatation | 2 (8%) | 9 (50%) | 0.002 |
| LV end-diastolic diameter (mm) | 50.83±4.34 | 54.18±3.82 | 0.016 |
| LVEF (%) | 59.80±5.69 | 37.12±7.34 | <0.001 |
| Pulmonary systolic pressure (mmHg) | 32.53±8.44 | 38.67±13.75 | 0.134 |
| E/E' | 8.15±1.86 | 10.80±5.1 | 0.094 |
| Any degree of mitral regurgitation | 8 (32%) | 12 (67%) | 0.028 |
| RV dysfunction | 3 (12%) | 4 (22%) | 0.407 |
| DC=Dilated cardiomyopathy; LA=left atrium; LV=left ventricle; LVEF=left ventricular ejection fraction; RV=right ventricle; TIC=tachycardia induced cardiomyopathy | | | |

Association of LVEF recovery with HF admission in the follow up.

DC patients were admitted more frequently for HF during follow up (median clinical follow-up 60 months) (44% vs 0% p<0.001) (Figure 3)

Discussion

To the best of our knowledge, this is the first study that includes CMR and ECG in a comprehensive model to predict LVEF recovery in patients admitted for heart failure and SVT. This study demonstrates that a QRS duration ≥100 ms, LVEF < 40% and presence of LGE predict independently lack of LVEF recovery. These novel findings may be of major clinical value to readily differentiate TIC from DC. Furthermore, our findings have biological plausibility orienting to an established myocardial damage (9, 10).

In a previous study, comparing 25 patients with TIC and other 25 with DC, QRS was wider in the DC group (11). However, only 6 patients with DC had SVT unlike our DC group where all patients had SVT. Furthermore, in that study no CMR data were provided.

LGE on CMR has proved to be useful to assess myocardial fibrosis in several cardiomyopathies (10). However, data about LGE prevalence and characteristics in patients with TIC are lacking. Some CMR studies focused in arrhythmia-induced cardiomyopathy caused by premature ventricular contractions and/or ventricular tachycardia (12). Hasdemir et al studied 27 patients with LVEF <50% caused by premature ventricular contractions or ventricular tachycardia. Improvement in LVEF after ventricular arrhythmia treatment was observed in 22 patients. After LVEF recovery CMR was performed in those patients and only 1 patient had LGE. The remaining 5 patients who did not recovered LVEF also underwent CMR and 4 had LGE (11). In that study, however, there was no information about the localization of LGE. In our study the prevalence of LGE in the TIC group is 16% and, interestingly enough, in all cases LGE is localized in the mid-myocardium.

In the CAMERA-MRI study (13), that randomized 68 patients with persistent AF and LVEF \leq 45% either to catheter ablation (CA) or medical rate control (MRC), all patients underwent to CMR. Although both CA and MRC groups showed the same prevalence of LGE in those undergoing CA, the presence of LGE was associated with lack of LVEF normalization at 6 months (12). Moreover, in a retrospective analysis of patients undergoing CA for AF, the presence of ventricular LGE was associated with a lack of recovery of LVEF (14).

Previous studies also suggested that TIC patients do not show LV remodeling once the tachycardia is under control (15). Interestingly, in our study, TIC patients presented lower LV end-diastolic dimensions and volumes than DC patients supporting the theory of a “reversible” myocardial damage related to the heart rate. Notably, DC patients present also mitral regurgitation more frequently at follow up.

The long-term prognosis of TIC remains controversial. A study by Fujino et al. demonstrated that cardiac death and recurrent hospitalizations were lower in the TIC group than the DC group (4). However, a small study reported a relevant number of sudden deaths during follow up in TIC patients (16). Furthermore animal studies provide evidence for a worse outcome in TIC setting (17). In our study TIC was associated with better outcomes than DC since none of 25 patients in the TIC group had an admission for HF during follow up.

Our study has several limitations. First of all, it is an observational study, therefore, we cannot rule out the potential risk of selection bias. Secondly, due to the small sample size, the study could be unpowered and, therefore, our results should be only considered as hypothesis generating. However, most previous studies addressing the differences between TIC and DC had a similar sample size (9, 11, 12). In addition, some patients in the TIC group might have had an early-phase DC that responded quickly to medical treatment. However, at present, no other definite criteria are available for TIC. Furthermore, our study has included patients during a decade. Although therapies for HF (18) and SVT (19) have been improving since 2008, ACEIs, BB and mineralocorticoids receptor antagonists have been the cornerstone of HF in the

last two decades and our patients were on optimal medical therapy at discharge. Remarkably, in both groups no differences were detected in the treatment at discharge. In spite of these limitations we think that this study provides new information about the usefulness of ECG and CMR to differentiate TIC from DC in real life clinical practice.

Conclusion

This study demonstrates for the first time that QRS duration ≥ 100 ms, LVEF < 40% on CMR and presence of LGE independently predict lack of LVEF recovery in patients with systolic dysfunction admitted for HF and SVT. Furthermore, our findings suggest that DC is associated with a worse clinical outcome during follow up.

Abbreviations

ACEI= angiotensin-converting-enzyme inhibitors

AF=atrial fibrillation

AFT=Atrial flutter

BBB= Bundle branch block

CKD=chronic kidney disease

CMR=cardiac magnetic resonance

COPD= chronic obstructive pulmonary

DC=Dilated cardiomyopathy

ECG=Electrocardiogram

LA=left atrium

LBBB= Left bundle branch block

LGE=late gadolinium enhancement

LV=left ventricle

LVEF=left ventricular ejection fraction

MR=mitral regurgitation

MRA= Mineralcorticoid receptor antagonist

RBBB= Right bundle branch block

RV=right ventricle

RVEF=right ventricular ejection fraction

SVT= supraventricular tachyarrhythmia

TIC=tachycardia induced cardiomyopathy

Declarations

Ethics approval and consent to participate. Not applicable

Consent for publication. Not applicable

Availability of data and materials. The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request

Competing interests. The authors declare that they have no competing interests

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Authors' contributions.

AV has made substantial contributions to conception, design and acquisition, analysis and interpretation of data. He has been involved in drafting the manuscript and revising it critically for important intellectual content. He has given final approval of the version to be published

AC has made substantial contributions to conception, design and acquisition, analysis and interpretation of data. He has been involved in drafting the manuscript and revising it critically for important intellectual content. He has given final approval of the version to be published

PMV has made substantial contributions to conception, design and acquisition, analysis and interpretation of data. He has been involved in drafting the manuscript and revising it critically for important intellectual content. He has given final approval of the version to be published

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FA has made substantial contributions to conception, design and acquisition, analysis and interpretation of data. He has been involved in drafting the manuscript and revising it critically for important intellectual content. He has given final approval of the version to be published

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Figures

FIGURE 1

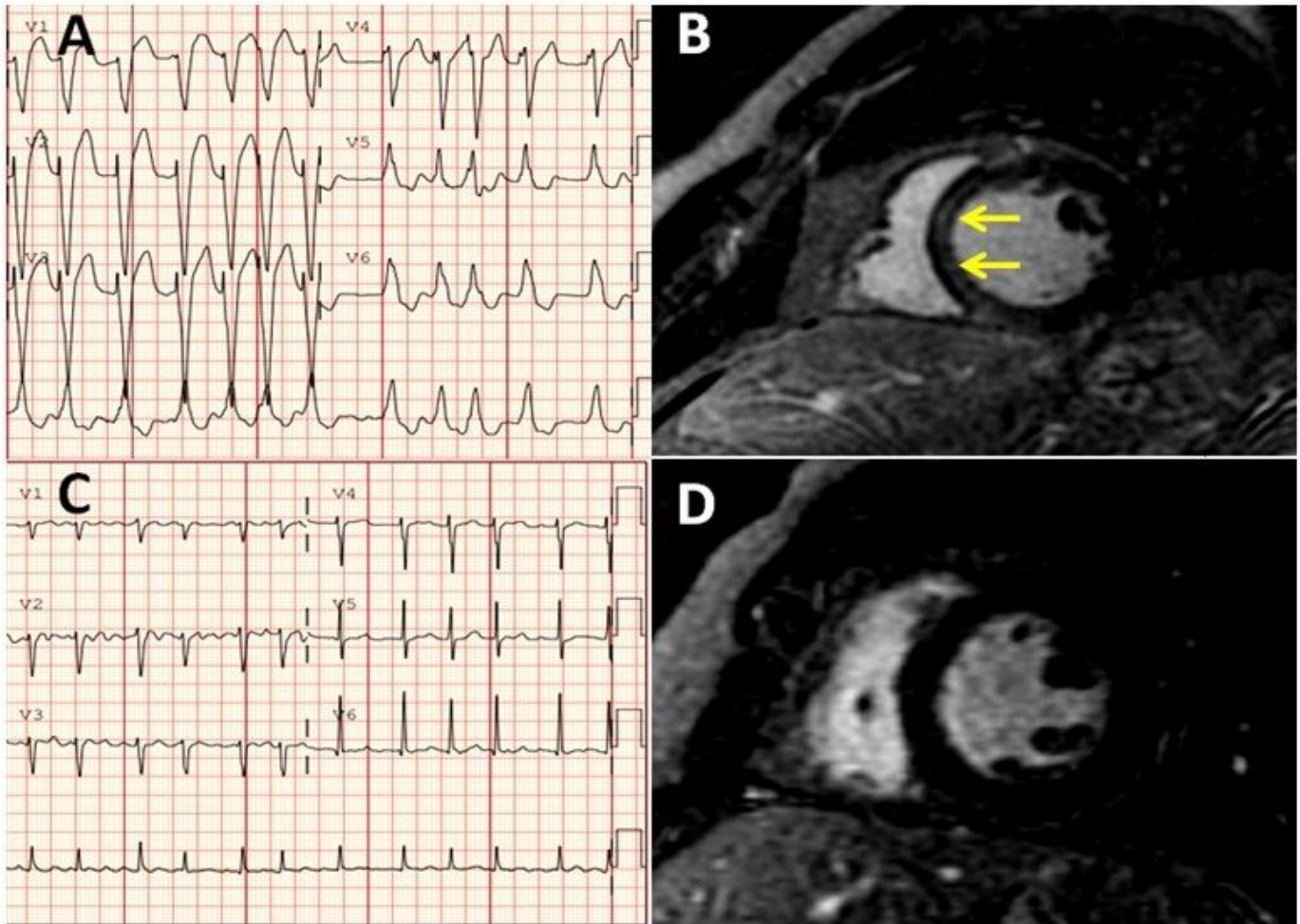


Figure 1

Examples of ECG and LGE findings. A and B, ECG and LGE short axis view belonging to a patient with lack of LVEF recovery in the follow-up. QRS duration of 150ms and a mid myocardial LGE (yellow arrows) are shown. C and D, ECG and LGE short axis view belonging to a patient with LVEF recovery in the follow-up. ECG showed QRS duration of 83 ms and LGE was absent.

FIGURE 2

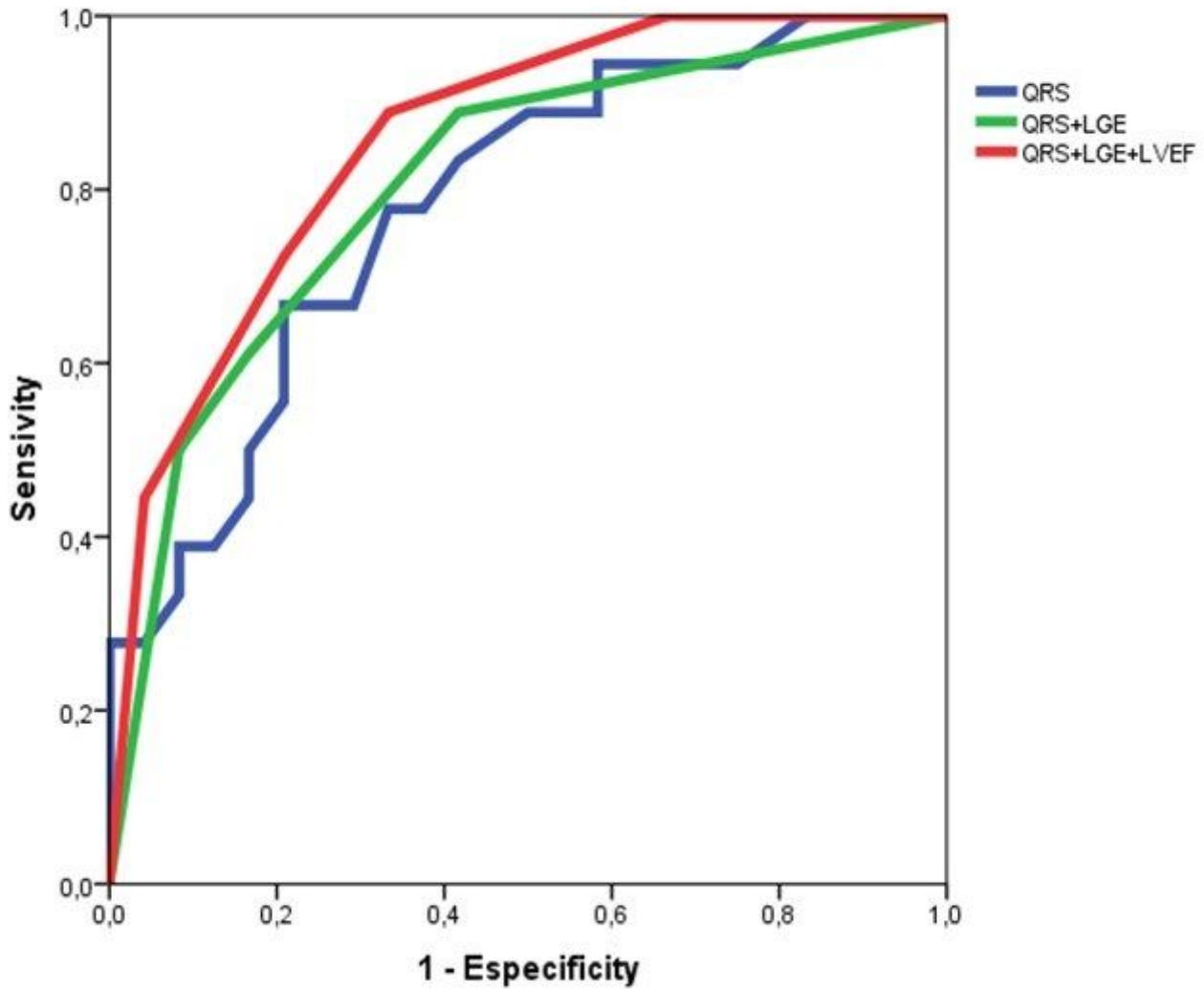


Figure 2

ROC curves for additive models associated with lack of EF recovery. Blue, QRS (AUC= 0.78 p=0.002. Green, QRS+LGE (AUC 0.81 p=0,001). Red, QRS+LGE+LVEF (AUC 0,86 p<0,001). AUC, area under curve; LGE, late gadolinium enhancement; LVEF, left ventricular ejection fraction.

FIGURE 3

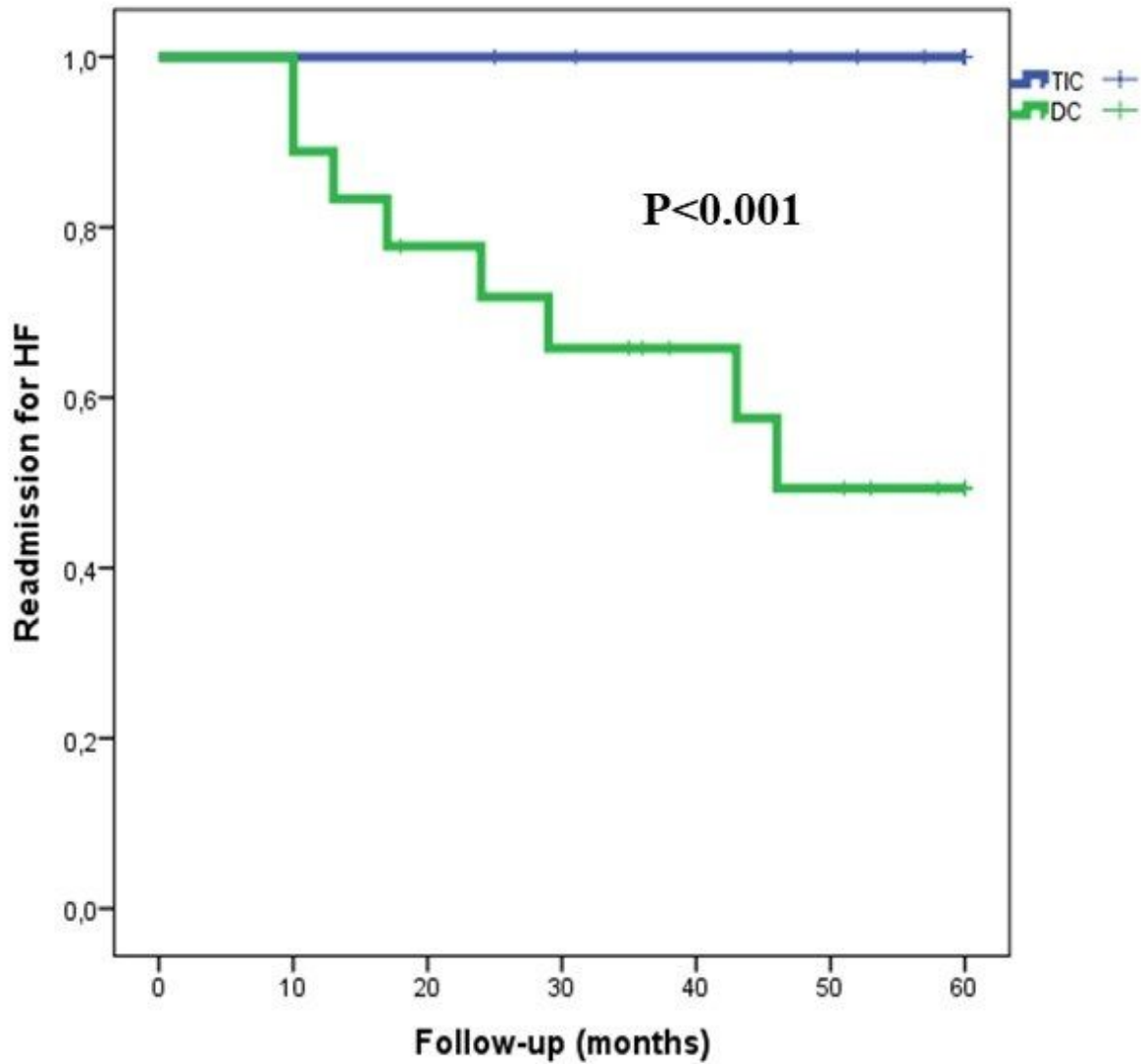


Figure 3

Readmission for heart failure ($p < 0.001$) DC=Dilated Cardiomyopathy. TIC=Tachycardia-induced cardiomyopathy

Supplementary Files

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- [Table1Supplementalmaterial.docx](#)