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Dissociation between structural reverse remodeling and clinical outcomes in ischemic cardiomyopathy undergoing cardiac resynchronization therapy: a real-world analysis

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Abstract

Patients with non-ischemic cardiomyopathy (NICM) generally exhibit better responses to cardiac resynchronization therapy (CRT) due to more pronounced reverse remodeling compared to ischemic cardiomyopathy (ICM) patients. However, the prognostic impact of CRT in ICM patients remains uncertain. This study aimed to evaluate the impact of heart failure (HF) etiology on reverse remodeling, clinical outcomes, and survival following CRT in a real-world population.

We conducted a retrospective observational study including patients who underwent CRT-P (pacing)/CRT-D (defibrillator) implantation. Patients were stratified according to HF etiology (ICM vs. NICM). The primary composite endpoint was the occurrence of all-cause mortality and HF hospitalization. Secondary outcomes focused on echocardiographic measures of left ventricular reverse remodeling. Survival analysis was performed using Kaplan-Meier curves and Cox regression models.

Of the 95 patients initially screened, 83 were included in the final analysis, of whom 38 (45.8%) had ICM. The mean age was 69.5 ± 10.4 years, and 75.8% were male. Both groups received similar medical therapy. NICM patients demonstrated a significant improvement in reverse remodeling after CRT [left ventricular ejection fraction (LVEF): $32.3 \pm 11.5\%$ to $44.0 \pm 13.2\%$, $p=0.001$; end-diastolic volume (LVEDV): 189.6 ± 74.3 mL to 143.9 ± 48.9 mL, $p=0.010$] with post-CRT LVEF being higher than in ICM patients ($44.0 \pm 13.2\%$ vs. $35.5 \pm 13.5\%$, $p=0.003$). NICM patients were more likely to be super-responders (58.1% vs. 25.0%, $p=0.014$). No significant differences were observed between groups in the primary composite endpoint (27.3% vs. 42.1%, $p=0.171$) or overall survival over a mean follow-up of 35 months (log-rank $p=0.733$).

Although ICM patients showed less pronounced reverse remodeling following CRT, long-term clinical outcomes and survival were comparable to those observed in NICM. These findings reinforce the role of CRT as a disease-modifying therapy that prevents disease progression irrespective of HF etiology and support its systematic use in all eligible patients.

Key words: cardiac resynchronization therapy, ventricular remodeling, ischemic heart disease, cardiomyopathy.

Introduction

Cardiac resynchronization therapy (CRT) is a well-established non-pharmacological treatment strategy for patients with heart failure (HF) and reduced left ventricular ejection fraction (LVEF), who are in sinus rhythm or atrial fibrillation and remain symptomatic despite optimized medical therapy. It is particularly indicated in patients exhibiting electromechanical desynchrony, most commonly associated with intraventricular conduction delays, typically presenting as complete left bundle branch block (LBBB) [1-5].

Initial clinical trials, such as MIRACLE, demonstrated significant symptomatic improvement with CRT. Subsequent landmark studies—COMPANION and CARE-HF—confirmed its effectiveness in improving major clinical outcomes, including reductions in HF hospitalizations and all-cause mortality. These findings firmly established CRT as a cornerstone in international HF management guidelines [1,6-8].

However, as CRT use expanded, it became increasingly evident that the therapeutic response is heterogeneous. Beyond clinical outcomes, the degree of reverse ventricular remodeling—measured by improvements in LVEF and reductions in left ventricular volumes—has emerged as a growing focus of scientific interest. This variability in response has led to the categorization of patients as responders, super-responders, or non-responders, primarily based on echocardiographic parameters. It is estimated that up to one-third of patients do not exhibit significant reverse remodeling despite fulfilling current implantation criteria [3,6-9].

The lack of standardized definitions for these response categories across studies raises important concerns about their clinical utility and the risk of misinterpretation. Such ambiguity may lead to the under-referral of patients who could benefit from CRT, based on characteristics perceived to predict a poor response—potentially compromising patient survival [9].

One key factor influencing CRT response appears to be the etiology of HF. Patients with ischemic cardiomyopathy (ICM) often exhibit a diminished remodeling response and poorer clinical outcomes compared to those with non-ischemic cardiomyopathy (NICM), likely due to the presence of extensive myocardial fibrosis, which limits the potential for mechanical recovery [4,5,9,10]. Nonetheless, major CRT trials have not demonstrated a significant interaction between HF etiology and CRT benefit with respect to composite outcomes such as mortality and HF hospitalizations [6,8,11,12]. This inconsistency suggests a potentially divergent relationship between reverse remodeling and long-term clinical outcomes in ischemic versus non-ischemic HF.

This study aims to investigate the role of HF etiology in influencing reverse remodeling, outcomes, and survival among patients undergoing CRT in a real-world clinical setting.

Objectives

The primary objective of this study was to evaluate whether long-term clinical outcomes and survival after CRT differ according to HF etiology. Secondary objectives included the assessment of left ventricular reverse remodeling and characterization of CRT response patterns in patients with ICM versus NICM in a real-world clinical setting.

Materials and Methods

Study design

This was a retrospective, observational, single-center study with both descriptive and analytical components that included consecutive 95 patients who underwent CRT-P (pacing) or CRT-D (defibrillator) implantation between January 1, 2020, and December 31, 2024, at a district general hospital cardiology department.

Study population and sample

Eligible patients were adults (≥ 18 years) who met European Society of Cardiology guideline-based indications for CRT implantation, regardless of HF etiology. Exclusion criteria comprised: acute coronary syndrome (ACS) within the preceding three months, incomplete electronic medical records, follow-up performed at another institution after device implantation, loss to follow-up, or failure to meet standard CRT eligibility criteria.

After application of inclusion and exclusion criteria, patients were stratified into two groups based on HF etiology: ICM and NICM.

Data collection

Collected data included demographic information, cardiovascular (CV) risk factors, HF etiology, pharmacologic treatment, and echocardiographic measurements.

Echocardiographic evaluation included LVEF, left ventricular end-diastolic volume (LVEDV), and left ventricular end-systolic volume (LVESV), assessed both prior to CRT implantation and at the most recent follow-up examination. LVEF was calculated using the modified Simpson biplane method.

In this study, patients were classified as "responders" or "super-responders" according to echocardiographic criteria. "Responders" were defined by the presence of at least one of the following: increase in LVEF $\geq 5\%$, reduction in LVEDV $\geq 10\%$, or reduction in LVESV $\geq 15\%$. "Super-responders" were defined by at least one of the following: increase in LVEF $\geq 10\%$, reduction in LVEDV $\geq 20\%$, or reduction in LVESV $\geq 30\%$.

Electrocardiography (ECG) data and arrhythmias documented during follow-up were also recorded. Outcomes such as HF hospitalization and all-cause mortality were analyzed.

To obtain this information, the patient's clinical records were accessed using two informatic platforms. No valid questionnaires for assessment of quality of life were used.

Outcomes

The primary outcome was a composite endpoint of all-cause mortality and hospitalization for HF, assessed over a mean follow-up period of 35 months. The secondary outcome was a focus on reverse left ventricular remodeling, considering the etiology of HF.

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics (version 29.0.2.0, IBM Corp, Armonk, NY, USA). A 95% confidence interval (CI) was considered, and a p-value < 0.05 indicates statistical significance.

The sample under study was characterized through descriptive statistics. Categorical variables were presented as frequencies and percentages, and continuous variables as means and standard deviations, or medians and 25-75 percentile for variables with skewed distribution or a significant Shapiro-Wilk test. Comparison between groups was performed using the Chi-squared test or Fisher's exact test for categorical variables and a t-test (paired t-test or two-sample t-test), if the variable had a normal distribution, or the Mann-Whitney U test and Wilcoxon Signed-Rank test, if the variable had a non-normal distribution, as appropriate.

For survival analysis, the Kaplan-Meier technique was used in association with the Log-rank test, with the creation of survival curves considering the etiology of HF.

Ethical considerations and conflicts of interest

The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013) and approved by institutional ethics board of Unidade Local de Saúde do Algarve (NO.: UAI

128/2025; date: 12/2025). Due to the retrospective nature of the study, informed consent was waived by institutional ethics board of Unidade Local de Saúde do Algarve.

Results

Sample characterization

Of the 95 patients who underwent CRT-P or CRT-D during the study period, 83 met the inclusion criteria and were included in the final analysis. Among these, 38 (45.8%) had ICM and 45 (54.2%) had NICM. The mean age was 69.5 ± 10.4 years, and most patients were male (75.8%). All available characteristics are summarized on Table 1.

The most prevalent risk factors were dyslipidemia (69, 74.2%), hypertension (64, 68.8%), and diabetes mellitus (52, 55.9%). Atrial fibrillation was present in 27 (29%) of patients, and 31 (34.1%) had chronic kidney disease. Baseline LVEF was $33.3 \pm 11.1\%$, improving significantly to $40.7 \pm 14.1\%$ during follow-up ($p = 0.009$).

Overall, 34 (55.7%) patients met criteria for CRT “response”, while 24 (39.3%) were classified as “super-responders”.

Comparative analysis by etiology

Baseline clinical characteristics were largely similar between groups, except for a higher prevalence of diabetes mellitus (76.3% vs. 40.9%, $p = 0.001$) and dyslipidemia (89.5% vs. 65.9%, $p = 0.012$) among patients with ICM. The proportion of patients receiving guideline-directed medical therapy was comparable between groups (Table 1).

Baseline QRS duration was similar between groups (166 ± 24.4 ms in NICM vs. 169 ± 23.9 ms in ICM, $p=0.631$), with all patients presenting with LBBB. The distribution of true complete LBBB and LBBB-like patterns was also comparable between groups. Following CRT, QRS duration was numerically shorter in both groups, with no significant difference in the magnitude of QRS shortening between groups (19.8 ± 20.2 ms vs. 23.3 ± 23.0 ms, $p = 0.473$).

Regarding CRT response, NICM patients had a significantly higher proportion of “super-responders” (58.1% vs. 25.0%, $p = 0.014$). LVEF and LVEDV in the NICM group significantly improved post-CRT (from $32.3 \pm 11.5\%$ to $44.0 \pm 13.2\%$, $p=0.001$ and 189.6 ± 74.3 ml to 143.9 ± 48.9 ml, $p=0.010$, respectively), with post-CRT LVEF significantly higher than in the ICM group ($44.0 \pm 13.2\%$ vs. $35.5 \pm 13.5\%$, $p = 0.003$). Nevertheless, biventricular pacing was significantly higher in the ICM group ($97.8 \pm 2.82\%$ vs $95.1 \pm 6.73\%$, $p=0.022$). Among patients with atrial fibrillation, mean biventricular pacing was $93.7 \pm 11.4\%$, with no

significant difference between NICM and ICM groups ($91.8 \pm 15.3\%$ vs. $98.9 \pm 0.9\%$, $p=0.141$).

Outcome analysis

Table 2 shows the incidence of outcomes according to HF etiology over a mean follow-up of 35 months, revealing no statistically significant differences between groups. The primary composite outcome (all-cause mortality and HF hospitalizations) occurred in 12 (27.3%) NICM and in 16 (42.1%) ICM patients ($p = 0.171$). Individual components of the primary outcome also showed no differences between groups.

Survival analysis

Figure 1 shows the Kaplan–Meier curves for all-cause mortality according to heart failure etiology. During the follow-up period, there were no significant differences in time to the studied endpoint between patients with NICM and ICM undergoing CRT (log-rank $p = 0.733$).

Discussion

The present study provides real-world evidence that the clinical outcomes following CRT were comparable irrespective of HF etiology. Although patients with ICM exhibited significantly attenuated left ventricular reverse remodeling compared with those with NICM, this did not translate into worse long-term clinical outcomes or survival. These findings support the concept that the therapeutic effects of CRT extend beyond echocardiographic markers of remodeling, reinforcing its role as a disease-modifying therapy across the spectrum of HF etiologies.

In this cohort, patients with ICM undergoing CRT showed less pronounced left ventricular reverse remodeling—indicated by smaller improvements in LVEF and ventricular volumes during follow-up—compared to those with NICM. This aligns with findings from existing literature, including sub-analyses of major trials like MIRACLE, MADIT-CRT, and CARE-HF [13-15]. The attenuated reverse remodeling in ICM patients is often attributed to non-viable myocardial scar tissue, which hinders mechanical recovery and limits the potential for improvement. However, due to the absence of systematic assessment of scar burden by cardiac magnetic resonance or nuclear imaging, our study cannot establish a causal relationship between myocardial fibrosis and attenuated remodeling.

Despite this attenuated remodeling, our study showed that clinical outcomes and survival rates did not significantly differ between the two etiological groups. This supports previous observations from large randomized clinical trials—such as CARE-HF, COMPANION, MADIT-CRT, and RAFT—which found no significant interaction between HF etiology and CRT effectiveness with respect to all-cause mortality and HF hospitalization [6,8,11,12]. Interestingly, patients with ICM exhibited significantly higher percentages of biventricular pacing, which may have contributed to their preserved clinical benefit despite reduced structural improvement. In our cohort, mean biventricular pacing among patients with AF was relatively high in both groups, with no significant difference between NICM and ICM patients. Thus, inadequate biventricular pacing in patients with AF was unlikely to be a major determinant of the differences in reverse remodeling observed between the two groups, although the small number of patients with AF limits definitive conclusions. Also, prior studies have suggested that in ischemic HF, even a modest degree of reverse remodeling may confer a meaningful reduction in adverse events, whereas a similar degree of improvement in NICM may not correspond to the same prognostic benefit. This raises the possibility that reverse remodeling carries different clinical weight depending on underlying etiology [16].

Furthermore, the therapeutic effects of CRT may extend beyond what is captured by traditional echocardiographic measures. For instance, positron emission tomography studies have demonstrated metabolic improvements in myocardial efficiency and energy utilization following CRT in both ICM and NICM, with no clear distinction between the two groups. This suggests the presence of "molecular reverse remodeling" that may occur independently of visible changes in chamber size or LVEF—highlighting the limitations of relying solely on echocardiographic parameters to assess CRT response [17].

It is also essential to recognize that patients with ICM typically have a higher baseline risk for adverse CV events. Thus, the relatively higher event rates seen in this group likely reflect their underlying risk profile rather than reduced therapeutic efficacy of CRT. In fact, the absolute benefits of CRT may be more pronounced in this population [16].

These findings contribute to the ongoing debate surrounding the use of the term “non-responder”, which is frequently based on the absence of significant reverse remodeling. This binary classification may be overly simplistic and potentially harmful, leading to passive management without specialty care [18]. As demonstrated in the present analysis, lack of substantial remodeling effect and LVEF improvement does not necessarily equate to poor outcomes. Therefore, CRT should be considered a disease-modifying therapy—capable of

stabilizing or partially reversing the course of HF (Partial Remission) or accomplish full structural recovery (Full Remission). Given the chronic and progressive nature of HF, the goal of CRT should not be complete remission but rather sustained clinical improvement and risk reduction [3]. Despite the progressive remodeling in some patients, chronic CRT continues to provide hemodynamic augmentation in the failing heart in most patients [19].

So, patients who meet established guideline criteria for CRT implantation should be offered this therapy regardless of HF etiology. The clinical benefits of CRT, including reductions in hospitalizations and mortality, have been consistently demonstrated across diverse patient populations. Our real-world findings reaffirm the broad applicability and therapeutic value of CRT regardless of HF etiology.

Conclusions

Clinical outcomes following CRT were comparable regardless of HF etiology. Although ICM patients showed less pronounced reverse remodeling effect, with less LVEF and ventricular volumes improvements, and fewer super responders following CRT, their clinical outcomes were not significantly worse than those with NICM. CRT should therefore be considered a cornerstone therapy across etiologies, offering substantial benefits even in the absence of marked structural improvement.

Study limitations

This study has several limitations that should be acknowledged. First, its retrospective and single-center design may limit the generalizability of the findings and introduces the potential for selection bias and residual confounding, despite careful data collection and statistical adjustment. Second, the relatively small sample size reduces statistical power, particularly for subgroup analyses and survival comparisons, increasing the risk of type II error. Furthermore, although not statistically significant, women were numerically more represented in the non-ischemic cohort. Given that female sex is widely associated with an enhanced physiological response to cardiac resynchronization therapy, this demographic imbalance acts as a potential unmeasured confounder that could have influenced the degree of reverse remodeling, and our sample size precluded a robust multivariable adjustment for gender-specific interactions. Third, the lack of systematic assessment of myocardial scar burden using cardiac magnetic resonance imaging or nuclear techniques precludes direct evaluation of the relationship between myocardial fibrosis and the degree of reverse remodeling, particularly in patients with

ischemic cardiomyopathy. Additionally, echocardiographic follow-up was not performed at standardized time intervals, which may have introduced variability in the assessment of remodeling response. Finally, patient-reported outcomes and quality-of-life measures were not systematically collected, limiting the evaluation of functional and symptomatic improvement after CRT. These limitations should be considered when interpreting the results, and further prospective multicenter studies with larger populations and comprehensive imaging assessment are warranted.

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Table 1. Baseline characteristics and outcomes of patients undergoing cardiac resynchronization therapy, stratified by etiology.

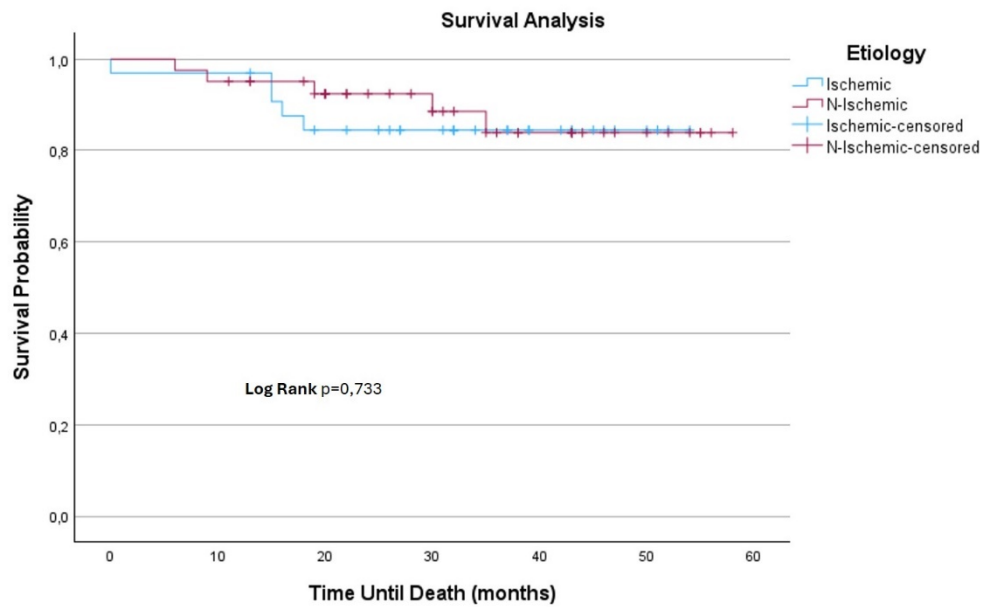
		N-Ischemic (n=45, 54.2%)	Ischemic (n=38, 45.8%)	Total (n=95)	p
Gender					
Male	n (%)	32.0 (71.1)	31.0 (81.6)	72.0 (75.8)	0.052
Female	n (%)	13.0 (28.9)	7.0 (18.4)	23.0 (24.2)	
Age	Mean±SD - years	68.8±10.5	69.9±10.1	69.5±10.4	0.618
Cardiovascular Risk Factors					
<i>Diabetes Mellitus</i> (Type 2)	n (%)	18.0 (40.9)	29.0 (76.3)	52.0 (55,9)	0.001
Dyslipidemia	n (%)	29.0 (65.9)	34.0 (89.5)	69.0 (74,2)	0.012
Arterial Hypertension	n (%)	26.0 (59.1)	29.0 (76.3)	64.0 (68,8)	0.098
Smoking <i>Status</i>	n (%)	17.0 (38.6)	17.0 (44.7)	37.0 (39,8)	0.576
Obesity	n (%)	9.00 (20.5)	12.0 (31.6)	22.0 (23,7)	0.250
Comorbidities					
Alcohol	n (%)	12.0 (27.3)	11.0 (28.9)	23.0 (24.7)	0.866
Chronic kidney disease	n (%)	15.0 (34.9)	15.0 (40.5)	31.0 (34.1)	0.602
Atrial fibrillation (AF)	n (%)	14.0 (31.8)	7.00 (18.4)	27.0 (29.0)	0.166
AF ablation	n (%)	2.00 (4.5)	0.00 (0.0)	2.00 (2.20)	0.297
Ventricular tachycardia	n (%)	2.00 (4.50)	6.00 (15.8)	10.0 (10.8)	0.136
Optimized medical therapy					
ARNI	n (%)	23.0 (52.1)	22.0 (59.5)	51.0 (55.4)	0.517
ACEi/ARB	n (%)	16.0 (36.4)	14.0 (37.8)	32.0 (34.8)	0.891
MRA	n (%)	29.0 (67.4)	26.0 (70.3)	62.0 (68.1)	0.786
SGLT2i	n (%)	29.0 (67.4)	23.0 (62.2)	60.0 (65.9)	0.622
Beta-blocker	n (%)	39.0 (90.7)	33.0 (89.2)	79.0 (86.8)	0.556
AAR	n (%)	10.0 (23.3)	7.00 (18.9)	21.0 (23.1)	0.636
Specificities of QRS and CRT					
QRS duration baseline	Mean±SD - ms	166±24.4	169±23.9	166±23.5	0.631
LBBB morphology	n (%)	45.0 (100.0)	38.0 (100,0)	95.0 (100.0)	--
tcLBBB	n (%)	20.0 (47.6%)	13.0 (35.1%)	37.0 (42.0)	0.361
lpLBBB	n (%)	22.0 (52.4%)	24.0 (64.9%)	51.0 (58.0)	
LBBB ≥ 150ms	n (%)	33.0 (78.6)	24.0 (75.0)	63.0 (77.8)	0.717
QRS duration after CRT	Mean±SD - ms	152±25.7	148±19.5	150±22.5	0.540
QRS variation	Mean±SD - ms	19.8±20.2	23.3±23.0	20.2±20.9	0.473
BiV	Mean±SD - %	95.1±6.73	97.8±2.82	95.7±7.47	0.022
BiV in AF patients	Mean±SD - %	91.8±15.3	98.9±0.90	93.7±11.4	0.141
Upgrade	n (%)	2.00 (16.7)	5.00 (38.5)	7.00 (25.9)	0.378
Need ventricular pacing	n (%)	15.0 (33.3)	4.00 (15.4)	19.0 (26.8)	0.100
Ventricular remodeling					
LVEF – Before CRT	Mean±SD - %	32.3±11.5	32.4±10.3	33.3±11.1	0.872
LVEF – After CRT	Mean±SD - %	44.0±13.2	35.5±13.5	40.7±14.1	0.003
		p=0.001	p=0.280	p=0.009	
LVEDV – Before CRT	Mean±SD - ml	189.6±74.3	203.3±74.1	196.9±77.5	0.567
LVEDV – After CRT	Mean±SD - ml	143.9±48.9	175.4±81.7	168.7±92.9	0.472
		p=0.010	p=0.059	p=0.010	

Data are expressed as n (%) or mean±SD as appropriate. n (%), total percentages; SD, standard deviation; AAR, antiarrhythmic drugs; ACEi/ARB, angiotensin converting enzyme inhibitors/angiotensin receptor blockers; ARNI, angiotensin receptor neprilysin inhibitor; CRT, cardiac resynchronization therapy; ICM, ischemic cardiomyopathy; LBBB, left bundle branch block; lpLBBB, left bundle branch block-like pattern; tcLBBB, true complete left bundle branch block; LVEDV, left ventricular end diastolic volume; LVEF, left ventricular ejection fraction; MRA, mineralocorticoid receptor antagonists; SGLT2i, sodium-glucose transport protein 2 inhibitors.

Table 2. Outcome occurrence compared based on IC etiology.

Mean follow-up 35months		N-Ischemic n=44, 53.7%	Ischemic n=38, 46.3%	Total n=95 (M 13.7)	p
Primary outcome	n (%)	12.0 (27.3)	16.0 (42.1)	28.0 (34.1)	0.171
All cause mortality	n (%)	7.0 (15.9)	10.0 (26.3)	17.0 (20.7)	0.284
HF hospitalization	n (%)	8.0 (17.8)	12.0 (31.6)	20.0 (24.1)	0.198

Data are expressed as n (%). n (%): total percentages; M(%): missing values percentages; CRT: cardiac resynchronization therapy; ICM: ischemic cardiomyopathy; NICM: non-ischemic cardiomyopathy.



Number at Risk:

Ischemic:	32	23	13	8	3	1	0
N-Ischemic:	40	31	22	15	8	6	0
Time (months)	0	10	20	30	40	50	60

Figure 1. Kaplan-Meier survival analysis of all-cause mortality by heart failure etiology, including the number of patients followed up (at risk) at each time interval.