Outcomes comparison between catheter ablation of ventricular tachycardia in Chagas disease versus ischemic and dilated cardiomyopathy — a retrospective cohort study

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Summary

Background Chagas cardiomyopathy (ChC) is associated with a high burden of ventricular arrhythmias (VA), but long-term outcomes of catheter ablation (CA) in this population remain poorly characterized, especially when compared to other cardiomyopathies.

Methods We performed a single-center retrospective cohort of consecutive patients with structural heart disease undergoing catheter ablation for sustained monomorphic VT (2011–2020) at a tertiary hospital in Brazil, grouped as ChC (n = 164), ischemic cardiomyopathy (ICM; n = 76) or idiopathic dilated cardiomyopathy (DCM; n = 48). The primary endpoint was a composite of all-cause death, heart transplantation, or VT recurrence; time-to-event outcomes were assessed with Kaplan–Meier and multivariable Cox models, and VT recurrence was additionally evaluated using Fine–Gray competing-risk analyses.

Findings We analysed 378 VT ablation procedures in 288 patients (mean age 61 ± 10 years; 208 [72%] male, 80 [28%] female; mean LVEF 35 ± 11%). Compared with ICM and idiopathic DCM, ChC more often required epicardial access (78% versus 15% in ICM and 31% in DCM; p < 0.001) and had lower acute non-inducibility (46% versus 62% in ICM; p < 0.001). Over a median follow-up of 29.0 months (IQR 3.3–69.1), for the last procedure the composite endpoint (death, heart transplant, or VT recurrence) occurred in 71.9% of ChC, 48.6% of ICM, and 58.3% of DCM (overall p = 0.068; pairwise ChC versus ICM p = 0.028). In adjusted Cox models, ChC was associated with higher risk of the composite endpoint (HR 1.73, 95% CI 1.16–2.59; p = 0.008) and higher all-cause mortality (HR 2.41, 1.00–5.78; p = 0.049), but not for VT recurrence, which did not differ by etiology in Kaplan–Meier or competing-risk analyses (Fine–Gray: last procedure p = 0.824; first procedure p = 0.305). Overall mortality was higher in ChC than non-ChC (36.0% versus 21.7%; p = 0.034), driven largely by non-cardiovascular death (p = 0.047) rather than cardiovascular death (p = 0.134). For the composite endpoint, higher LVEF was protective (per 1% increase: HR 0.97, 0.95–0.99; p = 0.017), while major intraprocedural complications conferred the greatest risk (HR 13.70, 3.27–57.39; p < 0.001).

Interpretation Chagas cardiomyopathy was associated with worse adjusted long-term outcomes after VT ablation—driven primarily by higher mortality. Across models, higher LVEF was protective, while markers of clinical instability and major intraprocedural complications identified patients at highest risk. These findings underscore the need for meticulous procedural strategy—particularly when epicardial access is anticipated—and comprehensive post-procedural heart failure and comorbidity management in ChC.

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Keywords: Catheter ablation; Ventricular tachycardia; Chagas disease

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Research in context

Evidence before this study

Chagas cardiomyopathy is associated with a high burden of ventricular arrhythmias, often of epicardial origin. Although catheter ablation is an established therapy for scar-related ventricular tachycardia, evidence in Chagas disease is limited to one randomized trial and small observational series, with limited follow-up and no direct comparison with other structural heart diseases. We searched PubMed in August 2025 using the terms "Chagas disease," "ventricular tachycardia," and "catheter ablation," without date restrictions; studies were excluded only if the full text was unavailable.

Added value of this study

By directly comparing Chagas cardiomyopathy with ischemic and idiopathic dilated cardiomyopathy in a large, well-characterized cohort, this study clarifies that the excess adverse outcomes observed after VT ablation in Chagas disease are driven primarily by mortality rather than arrhythmia recurrence. It provides novel insights into the substantial contribution of non-cardiac death to long-term prognosis, a dimension that has been poorly characterized in prior studies. In addition, our analysis identifies procedural complexity—particularly the need for epicardial access and lower acute success–as key contributors to risk, and defines clinical and procedural predictors of adverse outcomes, enabling more refined risk stratification and patient selection.

Implications of all the available evidence

Taken together, the evidence positions Chagas cardiomyopathy as a distinct, high-risk phenotype among candidates for VT ablation, in whom excess adverse outcomes are driven primarily by mortality. This underscores two priorities: first, rigorous procedural risk mitigation, particularly when anticipating epicardial access, minimizing intraprocedural complications, careful patient selection and pre-procedural stabilization. Second, a comprehensive disease management beyond the electrophysiology laboratory, including heart-failure optimization and comorbidity care. Prospective studies should evaluate tailored mapping/ablation approaches, adjunctive therapies, and alternative modalities to improve safety and survival. Finally, because Chagas disease imposes a disproportionate mortality burden and significant non-cardiovascular hazards, health-system responses in endemic and non-endemic regions should include targeted resource allocation, specialized referral pathways, and longitudinal care models that extend well beyond arrhythmia control.

Introduction

Chagas Disease (ChD) is an endemic protozoa-driven infectious disease in Brazil and in Latin America that is mainly transmitted by a bug-like insect bite. Besides being a regional disease, due to increasing international migration, it has become an emerging problem worldwide. In its chronic form, ChD can cause Chagas cardiomyopathy (ChC), a form of Dilated cardiomyopathy (DCM) that is associated with a progressive Heart Failure with reduced Ejection Fraction (HFrEF) and ventricular arrhythmias (VA), including scar-related monomorphic ventricular tachycardia (VT).[1,2] Patients with ChC have a worse prognosis compared to those with other etiologies of HFrEF.[3,4] Furthermore, these patients experience a higher burden of VT, with frequent recurrences despite antiarrhythmic therapy, which is primarily based on amiodarone.[5–10]

Catheter ablation (CA) is considered an effective treatment for recurrent symptomatic sustained monomorphic VT (SMVT) or ICD shocks for SMVT in non-ischemic cardiomyopathies and receives a class IIa-C indication for such patients in whom AADs are ineffective, contraindicated, or not tolerated, according to recent guidelines.[11–13] Regarding ChC, however, the evidence specifically guiding pharmacological and interventional management of arrhythmias remains limited, and general practice is largely extrapolated from ischemic and non-Chagas DCM studies.

In ChC patients, the myocardial scar is predominantly located on the meso-epicardial surface of the left ventricle (LV), particularly in the basal inferolateral wall and in the apex.[14,15] Consequently, the resulting VT are typically of epicardial origin.[16] Therefore, CA in this population yields better results when a combined endo-epicardial approach is utilized. However, while CA is a valuable option for managing VA, long-term outcome data in this population remains limited, and direct comparison with other etiologies are lacking.[17–19]

This study aimed to compare the outcomes of CA for VT in ChC versus those with Ischemic cardiomyopathy (ICM) and Idiopathic dilated cardiomyopathy. The analysis was conducted at a high-volume tertiary center in Brazil and assessed VT recurrence, mortality and heart transplantation over a long follow-up period. We hypothesized that ChC patients will have worse outcomes against other forms of cardiomyopathies.

Methods

This retrospective cohort study conforms with the STROBE[20] guidelines for reporting of Cohort studies.

Patient population

We analyzed 378 VT ablation procedures performed on 288 patients with Chagas cardiomyopathy, ischemic cardiomyopathy, or idiopathic dilated cardiomyopathy

from January 2011 to December 2020. Patients with other etiologies, such as Hypertrophic cardiomyopathy, Arrhythmogenic Right Ventricular cardiomyopathy, Cardiac Sarcoidosis, Congenital Heart Disease or Bundle Branch Reentry VT were excluded from the analysis (Fig. 1 – Flow Diagram). All patients gave written informed consent before undergoing the procedure and the study was approved by the institutional review board (Comissão de Ética para Análise de Projetos de Pesquisa do HCFMUSP–CAPPesq). Demographic data were obtained from patients' medical records, as documented at the time of clinical evaluation and study enrollment.

Electrophysiology procedures

All procedures were performed at the electrophysiology laboratory of the Heart Institute (InCor) of the University of Sao Paulo Medical School. Across the study period, five primary operators with comparable training performed the ablations. Due to incomplete operator identifiers in the registry, per-operator outcomes could not be reliably ascertained, but case assignment followed routine scheduling rather than patient risk. General anesthesia was combined with inhalational anesthetic agents (sevoflurane), propofol and/or intravenous fentanyl. Per our institutional protocol, a central venous catheter, an arterial line, and a urinary catheter for urinary output monitoring was used in all cases unless contraindicated (suspected/confirmed local infection or vascular thrombosis/occlusion at the intended access site, or urethral stenosis).

A protocol of programmed ventricular stimulation (PVS) with driving cycle lengths of 600 and 430 ms with up to 3 extrastimuli at a minimum of 200 ms, in two different locations in the right ventricle (apex and outflow tract), was performed (EP-TRACER software version 2.2, Cardiotek B.V., Netherlands), to induce VT and also for final testing after ablation.

Electroanatomical mapping (EAM) was performed mainly with the CARTO system (Biosense Webster, Diamond Bar, CA), and rarely with Ensite system (Abbott, Chicago, IL), to identify ablation targets. Scar tissue was defined by low-voltage areas (bipolar <1.5 mV and unipolar <8.3 mV for left ventricle or <5.5 mV for the right ventricle) while on RV apical pacing. Mapping was initiated in the ventricular chamber most likely to harbor the VT substrate, guided by PVS-induced and clinical VT morphologies and by imaging evidence of scar localization. If VT remained inducible or the target was not adequately identified, mapping proceeded to the contralateral ventricle and/or epicardial access was pursued. The primary targets for ablation were late potentials (LP, defined as local ventricular potentials detected after the end of the surface QRS complex) located within or adjacent to these scar areas. Ablation was performed using a cooled-tip irrigated catheter.

When EAM was unavailable, ablation was performed using an 8 mm solid-tip catheter (St. Jude Medical, St. Paul, MN, USA) with power limited to 50 W and temperature to 65 °C. Activation and entrainment mapping guided ablation when VT was

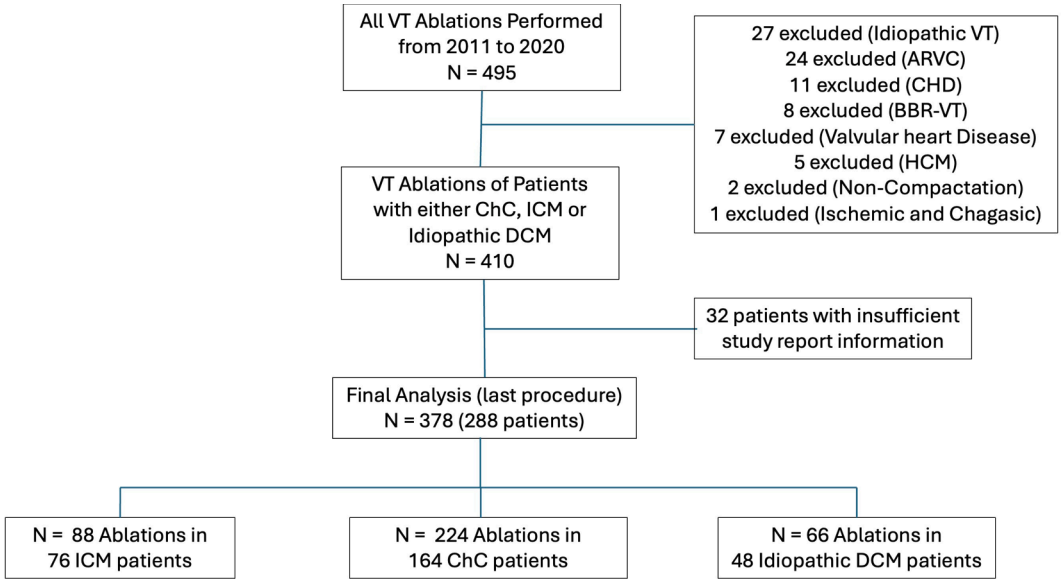

Fig. 1: Flow of patients and procedures. Flow of patients and procedures included in the analysis. Among 495 ventricular tachycardia (VT) ablations performed between 2011 and 2020, 410 met inclusion criteria (Chagas [ChC], ischemic cardiomyopathy [ICM], or idiopathic dilated cardiomyopathy [DCM]). After exclusions, 378 procedures in 288 patients were analyzed.

hemodynamically tolerated; otherwise, a substrate-based approach targeting late potentials and pace mapping (≥11/12 lead match) was used. Procedural endpoints included complete or ≥50% reduction of abnormal electrograms and non-inducibility of VT at final programmed stimulation. Ablation was typically endocardial, with epicardial access added when indicated—frequently at the outset in patients with Chagas cardiomyopathy. Although this study spans a 10-year period, there were no changes in the technique used. Only after 2020 did our center begin performing EAM with a functional substrate approach. During this period, there were improvements in the technologies employed and in the quality of three-dimensional imaging, however, the core mapping strategy has remained consistent.

Endocardial access was achieved via either a retroaortic (arterial) or transeptal (venous) approach. The retrograde route was generally preferred in younger patients, whereas a transseptal approach was favored in older individuals with atherosclerotic aortic disease; both routes could be used sequentially to optimize catheter manipulation. Epicardial ablation was performed using the traditional Tuohy needle technique described by Sosa et al.,[21] targeting a posterior pericardial entry. An anterior approach was reserved for cases with posterior adhesions or limited epicardial catheter maneuverability, in which a transseptal sheath was also employed to enhance catheter support and stability. Second-year electrophysiology fellows could perform posterior subxiphoid epicardial access under direct, scrubbed supervision of a senior attending, who verified needle trajectory and pericardial entry, confirmed wire position, and managed any complications.

Complications were analyzed based on their clinical relevance. For instance, pericardial bleeding was defined as clinically significant if more than 80 mL of blood was drained from the pericardium. Procedure success was assessed at the end by confirming LP abolition (complete scar homogenization was aimed) and using PVS protocol similar to the initial one, in at least one site in the RV, and defined as[1] complete success if no VT was inducible after ablation,[2] partial success if baseline clinical VT was not inducible but at least 1 nonclinical faster VT was induced, and[3] unsuccessful if clinical VT remained inducible. Clinical VT was defined as the morphology documented on surface ECG or digital telemetry, or, when unavailable, as reproducibly inducible VT with a cycle length consistent with that recorded by the ICD. Following the procedure, patients were routinely transferred to the intensive care unit (ICU) for post-procedural care.

Follow-up

Patients returned to the arrhythmia clinic within 30 days for a standardized visit that included ICD interrogation. An "adequate acute follow-up" was defined a priori as either a documented post-discharge outpatient visit for those discharged alive or in-hospital death during the index admission. Additional tests (echo, Holter, labs) were obtained at clinician discretion rather than by protocol, with subsequent follow-ups every 3–6 months and unscheduled visits as needed. Clinical characteristics and procedural data were obtained from a comprehensive review of electronic medical records. These records included implantable cardioverter-defibrillator (ICD) interrogation reports, laboratory and imaging results, record of emergency department visits and documentation of unexpected hospital admissions.

Implantable cardioverter-defibrillator (ICD) programming followed contemporary guideline recommendations and manufacturer-specific features, typically including three VT detection zones. For patients with previously recorded VT, the detection threshold was programmed approximately 10 bpm below the documented VT cycle length. ICD settings were not substantially modified after ablation compared with pre-procedure programming.

Endpoints

The primary endpoint was a composite of time to the first occurrence of all-cause mortality, need for heart transplantation, or ventricular tachycardia (VT) recurrence. The VT recurrence was defined as documentation of sustained VT by any of the following: (i) 12- lead ECG or rhythm strip; (ii) ICD interrogation, including device-treated episodes and device-detected slow VT not meeting therapy criteria; or (iii) contemporaneous clinician documentation of sustained VT during ICU or emergency department care. All-cause death was defined as death from any cause, irrespective of etiology. Cardiovascular death was defined as death due to cardiac or vascular causes, including sudden arrhythmic death, progressive heart failure, and others. Non-cardiovascular death was defined as death clearly attributable to non-cardiac causes, such as malignancy, infection/sepsis and others. Heart Transplant candidacy and listing were determined by the institutional Advanced HF team according to contemporary criteria, with primary indications including refractory/advanced HF despite optimal therapy and, in select cases, refractory ventricular arrhythmias.

Secondary endpoints consisted of each component of the primary endpoint analyzed individually. For most analyses, the follow-up period for patients with multiple procedures commenced after their final procedure (final clinical benefit), but for the primary combined endpoint, an analysis regarding first-ever VT ablation procedure was also done.

Statistical analysis

Continuous variables with normal distribution are presented as mean ± standard deviation (SD), whereas those with non-normal distribution are expressed as

median and interquartile range (IQR). Categorical variables are summarized as absolute frequencies and corresponding percentages. The assumption of normality was assessed using the Kolmogorov–Smirnov test. Group comparisons for categorical variables were performed using the chi-square test or Fisher's exact test, as appropriate. For continuous variables, comparisons between groups were conducted using the one-way ANOVA or the Kruskal–Wallis test with correction to ties, according to data distribution. A complete-case analysis was undertaken because the overall proportion of missing data was below commonly accepted methodological thresholds (<10%) and the missingness was judged to be completely at random. Only two variables, "Length of stay after ablation" (15%) and "VT cycle length" (25%) had more than 10% of missingness. The two variables with greater-than-usual missingness reflected data-entry errors rather than systematic factors, supporting the assumption of missing completely at random and the appropriateness of the complete-case approach.

Time-to-event endpoints (primary and secondary) were analyzed using Kaplan–Meier estimates and compared with log-rank tests. Because risk sets became sparse beyond 8 years, follow-up was administratively censored at that time point. For the primary composite endpoint, univariable Cox proportional hazards models were first performed to identify baseline clinical and procedural predictors (derived from the last procedure–final clinical effect). Sensitivity analyses were conducted using covariates for the first procedure, including all candidate variables. For VT recurrence, Fine–Gray competing-risk models were additionally used, considering death or heart transplantation as competing events. The multivariable model included covariates with univariable associations at $p < 0.10$, using last-procedure data for both clinical (but—based on a strong clinical rationale—focused on comparisons between Chagas versus non-Chagas cardiomyopathies) and procedural variables. For the overall mortality endpoint, we applied the same analytical framework. Given the low frequency of each individual intraprocedural complication, we combined all major adverse intraprocedural events—significant pericardial bleeding, need for surgery, complete atrioventricular block, coronary artery injury, and intraprocedural cardiac arrest—into a single variable termed "major intraprocedural complication." Other covariates were coded according to their clinical scale, but for better model fit and to facilitate clinical interpretability, we made some ordinal recodifications: procedural duration = 1 (<200 min), 2 (200–299), 3 (300–399), 4 (≥400); procedural success = 0 (failure), 1 (partial), 2 (complete); VT cycle length = 1 (<300 ms), 2 (300–399 ms), 3 (≥400 ms) and, for the competing risk regression for VT recurrence analysis, LVEF 0 (<25%) or 1 (>25%). Collinearity among covariates included in the multivariate models was assessed prior to model entry to ensure the stability and reliability of the estimates. Proportional hazards were assessed with global and covariate-specific tests based on scaled Schoenfeld residuals (*estat phtest*, Stata). When the proportional hazards assumption was flagged by the variable-specific Schoenfeld residuals test ($p < 0.05$), we modeled a time-varying effect for such variable. Hazard ratios and 95% confidence intervals were estimated across the range of procedure duration. Statistical significance was defined as a two-tailed p value < 0.05. All statistical analyses were performed using STATA version 18.0 (StataCorp, College Station, TX, USA).

Results

Patient characteristics

Patient inclusion process is outlined in Fig. 1.

The baseline characteristics of the study population are summarized in Table 1. The mean age was 61 ± 10 years, and patients were predominantly male (208 patients; 72%) over female (80 patients, 28%). The mean left ventricular ejection fraction (LVEF) was 35 ± 11%.

Patients with ICM were older, exhibited lower LVEF, were more frequently classified as New York Heart Association (NYHA) functional class III or IV, had more comorbidities such as hypertension, diabetes, chronic kidney disease, and a smoking history than patients with ChC and idiopathic DCM. Additionally, they were more likely to be on a lower pre-ablation dose of amiodarone.

Conversely, patients with ChC had a higher incidence of prior stroke or transient ischemic attack (TIA) and demonstrated a higher overall prevalence of amiodarone use. Furthermore, the proportion of female patients was significantly greater in the ChC group than in the idiopathic DCM (44% versus 13%; $p = 0.004$).

Procedural data

Procedural characteristics are summarized in Table 2.

Patients with ChC had a longer median procedure duration (270 [IQR: 210–360] min versus 240 [IQR: 180–315] min and 235 [IQR: 180–300] min for ICM and idiopathic DCM, respectively, $p = 0.004$) and required a significantly higher rate of epicardial access than the other groups (78% versus 15% in ICM and 31% for idiopathic DCM, $p < 0.001$). Additionally, patients with ChC and idiopathic DCM had a lower non-inducibility rate (46% and 50%, respectively, versus 62% in ICM, $p < 0.001$). The EAM was used in 39% of the cases, with no significant difference among the different underlying cardiomyopathies.

The predominant site of myocardial scarring also differed across cardiomyopathy types (Fig. 2A). Patients with ChC exhibited a marked predominance of inferolateral basal scarring (Fig. 2B and C), whereas those with ICM and Idiopathic DCM more commonly presented with septal substrate involvement.

	Total	Ischemic	Chagas	Idiopathic dilated	p value
	N = 288	N = 76 (26%)	N = 164 (57%)	N = 48 (17%)	
Age, years, mean (SD)	61 (10)	64 (9)	60 (10)	60 (13)	**p = 0.016**
Sex, n (%)					
Male	208 (72%)	58 (76%)	108 (66%)	42 (87%)	**p = 0.009**
Female	80 (28%)	18 (24%)	56 (34%)	6 (13%)	**p = 0.009**
NYHA functional class, median (IQR)	2 (1–3)	2 (2–3)	2 (1–3)	2 (2–3)	**p < 0.001**
NYHA class III–IV, n (%)	95 (34%)	35 (49%)	42 (26%)	18 (38%)	**p = 0.002**
Left ventricular ejection fraction, %, mean (SD)	35 (11)	33 (11)	35 (11)	38 (11)	**p = 0.033**
Atrial fibrillation, n (%)	70 (24%)	19 (25%)	39 (24%)	12 (25%)	p = 0.97
Hypertension, n (%)	158 (55%)	68 (89%)	71 (43%)	19 (40%)	**p < 0.001**
Diabetes mellitus, n (%)	49 (17%)	21 (28%)	22 (13%)	6 (12%)	**p = 0.016**
Dyslipidaemia, n (%)	129 (45%)	71 (93%)	44 (27%)	14 (29%)	**p < 0.001**
Stroke or TIA, n (%)	34 (12%)	6 (7.9%)	26 (16%)	2 (4.2%)	**p = 0.041**
Smoking history, n (%)	86 (30%)	44 (58%)	32 (20%)	10 (21%)	**p < 0.001**
Chronic kidney disease, n (%)	95 (33%)	47 (63%)	31 (19%)	17 (35%)	**p < 0.001**
Prior catheter ablation, n (%)	85 (30%)	21 (28%)	48 (29%)	16 (33%)	p = 0.81
Admission via emergency department, n (%)	206 (72%)	61 (80%)	115 (70%)	30 (64%)	p = 0.11
Electrical storm, n (%)	58 (21%)	18 (25%)	32 (20%)	8 (19%)	p = 0.65
Medications					
Amiodarone use, n (%)	225 (80%)	53 (72%)	138 (85%)	34 (77%)	**p = 0.045**
Amiodarone dose, mg, median (IQR)	400 (200–600)	300 (0–400)	400 (200–600)	400 (100–600)	**p = 0.025**
Beta-blocker use, n (%)	254 (91%)	68 (93%)	150 (92%)	36 (84%)	p = 0.18
Lidocaine use, n (%)	50 (18%)	18 (24%)	23 (14%)	9 (20%)	p = 0.16
ACE inhibitor use, n (%)	118 (42%)	30 (41%)	68 (41%)	20 (47%)	p = 0.82
ARB use, n (%)	95 (34%)	27 (37%)	57 (38%)	11 (26%)	p = 0.43
Spironolactone use, n (%)	148 (53%)	40 (55%)	85 (52%)	23 (54%)	p = 0.91
Aspirin use, n (%)	106 (38%)	56 (77%)	42 (25%)	8 (18%)	**p < 0.001**
Anticoagulant use, n (%)	86 (31%)	18 (25%)	51 (31%)	17 (40%)	p = 0.24
Furosemide use, n (%)	145 (52%)	36 (49%)	90 (55%)	19 (44%)	p = 0.41
Pre-procedure cardiac device, n (%)					
ICD	159 (55%)	36 (47%)	96 (59%)	27 (56%)	
CRT-D	17 (5.9%)	6 (7.9%)	8 (4.9%)	3 (6.2%)	
CRT-P	6 (2.1%)	1 (1.3%)	5 (3.0%)	0 (0%)	
Pacemaker	17 (5.9%)	2 (2.6%)	13 (7.9%)	2 (4.2%)	
None	89 (31%)	31 (41%)	42 (26%)	16 (33%)	
In-hospital length of stay, days, median (IQR)	7 (4–13)	7 (4–12)	6.5 (4–12)	6 (3–15.5)	p = 0.85

NYHA: New York Heart Association; LV: Left Ventricle; TIA: Transient Ischemic Attack; ICD: Implantable cardioverter-Defibrillator; CRT-D — cardiac resynchronization therapy defibrillator; CRT-P — cardiac resynchronization therapy pacemaker; IQR: Interquartile range; values in bold denote statistically significant differences (p < 0.05).

Table 1: **Baseline patients characteristics.**

The median post-procedural length of stay was 6.5 days (IQR 4–12) for patients with Chagas cardiomyopathy, 6 days (IQR 3–15.5) for those with idiopathic dilated cardiomyopathy, and 7 days (IQR 4–12) for those with ischemic cardiomyopathy (p = 0.85).

Complications

Procedural complications occurred in a notable portion of this cohort. Clinically relevant pericardial bleeding was the most frequent event, occurring in 37 of 378 procedures (9.8%). The incidence varied by etiology, occurring in 28/224 (12.5%) in ChC, 5/66 (7.5%) of idiopathic DCM and 4/88 (4.5%) in ICM cases (p = 0.088). Urgent cardiac surgery due to refractory bleeding was necessary in 13/378 (3.4%). Other complications included acute decompensated heart failure in 13/378 (3.4%), AV Block in 10/378 (2.6%), major vascular access complications 7/378 (1.8%), intraprocedural CPR in 5/378 (1.3%), stroke/TIA in 4/378 (1%), 3/378 (1%) phrenic nerve injury, 2/378 (0.5%) aortic dissection, 1/378 (0.2%) coronary artery injury.

Outcomes

Combined endpoint of death, heart transplant or VT recurrence

During a median follow-up of 29.0 months (IQR 3.3–69.1), 187 of 288 patients (65.0%) experienced the primary composite endpoint of death, heart transplantation, or VT recurrence. Adequate acute follow-up

	Total	Ischemic	Chagas	Idiopathic dilated	p value
	N = 378	N = 88 (23%)	N = 224 (59%)	N = 66 (18%)	
Number of procedures					p = 0.52
First	263 (69%)	64 (72%)	153 (68%)	46 (70%)	
Second	76 (20%)	18 (20%)	43 (19%)	15 (23%)	
Third	26 (6.9%)	4 (4.5%)	19 (8.5%)	3 (4.5%)	
Fourth	10 (2.6%)	1 (1.1%)	8 (3.6%)	1 (1.5%)	
Unknown	3 (0.8%)	1 (1.1%)	1 (0.4%)	1 (1.5%)	
Procedure duration, min, median (IQR)	240 (210–330)	240 (180–315)	270 (210–360)	235 (180–300)	**p = 0.004**
Number of induced VTs, median (IQR)	2 (1–3)	2 (1–3)	2 (1–3)	2 (1–3)	p = 0.71
VT cycle length, ms, median (IQR)	417 (345–472)	422 (356–500)	415 (342–451)	427 (354–480)	p = 0.17
Haemodynamically not tolerated VT, n (%)	431 (54%)	82 (44%)	270 (57%)	79 (54%)	**p = 0.006**
Electroanatomical mapping, n (%)	150 (39%)	27 (31%)	127 (56.7%)	27 (41%)	p = 0.15
Ablation strategy, n (%)					p = 0.41
Substrate mapping	150 (40%)	27 (31%)	95 (42%)	28 (42%)	
VT activation mapping	33 (8.7%)	12 (14%)	16 (7.1%)	5 (7.6%)	
Both	190 (50%)	47 (53%)	111 (50%)	32 (48%)	
Unclear	5 (1.3%)	2 (2.3%)	2 (0.9%)	1 (1.5%)	
Type of ablation, n (%)					**p < 0.001**
Endocardial only	168 (44%)	74 (85%)	49 (22%)	45 (68%)	
Epicardial only	56 (15%)	2 (2.3%)	48 (21%)	6 (9.1%)	
Endocardial + epicardial	152 (40%)	11 (13%)	126 (56%)	15 (23%)	
Procedural outcome (final inducibility), n (%)					**p < 0.001**
No VT inducible	191 (51%)	55 (62%)	103 (46%)	34 (52%)	
Only non-clinical VT	126 (33%)	21 (24%)	85 (38%)	20 (30%)	
Clinical VT inducible	50 (13%)	7 (8.0%)	35 (16%)	8 (12%)	
Not reported	10 (2.6%)	5 (5.7%)	1 (0.4%)	4 (6.1%)	

VT: Ventricular Tachycardia; IQR: Interquartile range; ES: Electrical Storm; VT: Ventricular Tachycardia; Cardiopulmonar Arrest (CPR); values in bold denote statistically significant differences (p < 0.05).

Table 2: **Procedure-level data; includes all induced VTs from all procedures (patients may contribute multiple observations).**

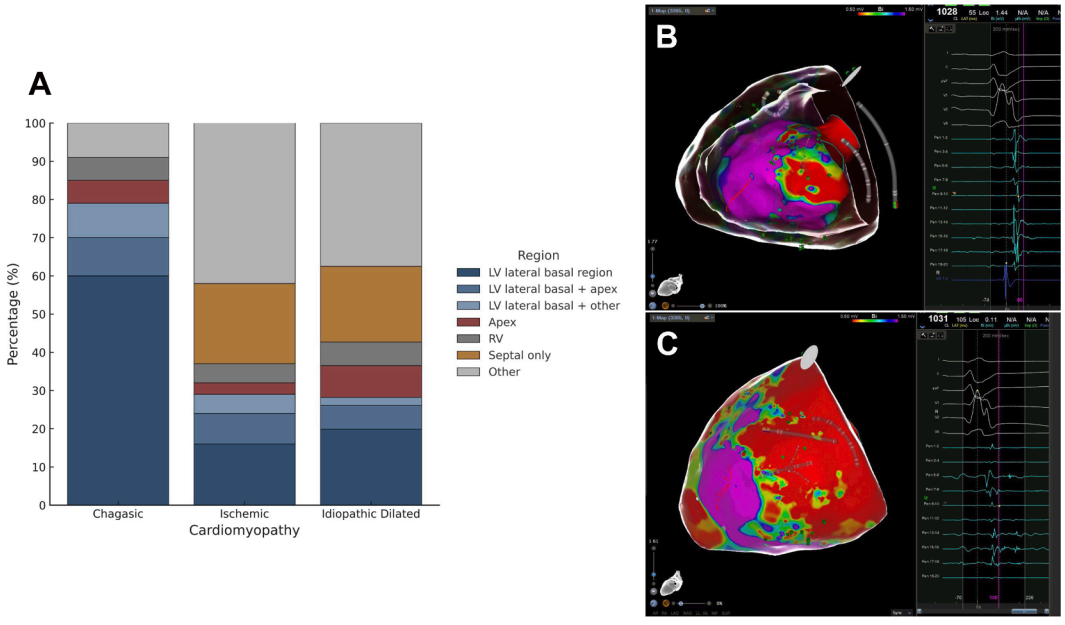

Fig. 2: Distribution of left ventricular scar location. (A) Representative distribution of left ventricular scar location in Chagas, ischemic, and idiopathic dilated cardiomyopathy. Chagas cardiomyopathy showed a predominance of inferolateral basal scars, whereas ischemic and idiopathic dilated cardiomyopathies demonstrated more frequent septal involvement. (B) Typical example of a Chagas scar in the endocardial basal lateral wall showing transmural involvement with predominance of epicardial (C) extension.

was achieved in 269 of 288 patients (93.4%) and did not differ by etiology (Chagas 95.73%, idiopathic dilated 89.58%, ischemic 90.79%; p = 0.181).

At eight years, when stratified by cardiomyopathy type, the primary endpoint was numerically inferior in patients with ICM (48.6% [37/76]) versus ChC (71.9% [118/164]), and idiopathic DCM (58.3% [28/48], p = 0.068). The overall difference was driven primarily by the comparison between ChC and ICM (p = 0.028 for pairwise log-rank test; Fig. 3A).

An analysis regarding the first-ever VT ablation procedure was also done (Fig. 3B). In this analysis, patients with ICM demonstrated a significantly higher event-free survival compared with both Chagas (pairwise p = 0.010) and idiopathic DCM (p = 0.040), with an overall log-rank p = 0.029. No significant difference was observed between Chagas and idiopathic dilated groups (p = 0.846).

VT recurrence

VT recurrence did not differ by aetiology over long-term follow-up. Kaplan–Meier estimates suggested a nonsignificant trend toward higher recurrence in ChC versus ICM (56.1% versus 43.4%; p = 0.093), with no differences between ChC and idiopathic DCM (56.1% versus 52.1%; p = 0.434) or between ICM and idiopathic DCM (43.4% versus 52.1%; p = 0.535; Fig. 4A). Results were concordant in competing-risk analyses: Fine–Gray models showed no between-group differences for the last procedure analysed (p = 0.824; Fig. 4B) or for first procedures only (p = 0.305; Fig. 4C).

All-cause death

All-cause death occurred in 87 of the 288 patients (30.2%) over the follow-up period. The median time from procedure to death was 134 days (IQR 23–804). There was no significant difference in the median time to death among the three etiologies (p = 0.876): ICM 170 days (IQR 30–682), ChC 115 days (IQR: 22–845), and idiopathic DCM 130 days (IQR 27–1083).

Kaplan–Meier analysis revealed that patients with ChC showed a higher overall long-term mortality compared to non-ChC disease (ChC: 36% versus non-ChC 21.7%; p = 0.034; Fig. 4A). This difference was driven primarily by the pairwise comparison between ChC and Idiopathic DCM (Chagas versus Idiopathic DCM p = 0.036; Fig. 5A).

An analysis by cause of death indicated that this difference was attributable mainly to a higher rate of non-cardiovascular death in the ChC group (Fig. 5B and C).

Predictors of death, heart transplant or VT recurrence

Due to its observational study design, Kaplan–Meier curves and log-rank comparisons are susceptible to confounding. Many factors were associated with the primary endpoint on univariate analysis (Table 3). In the multivariable Cox regression model, a higher risk of the composite endpoint was independently associated with cardiomyopathy type (HR 1.73, 95% CI 1.16–2.59; p = 0.008), electrical storm at/within 24 h (HR 1.94, 1.17–3.22; p = 0.011), in-hospital lidocaine use (HR 2.24, 1.24–4.03; p = 0.007), and major intraoperative complications (HR 13.70, 3.27–57.39; p < 0.001). Protective factors included higher left ventricular ejection fraction (HR 0.97, 0.95–0.99; p = 0.017) and aspirin use (HR 0.53, 0.31–0.90; p = 0.018).

A combined clinical and procedural variable multivariate cox regression was also done for first procedure, bringing somewhat similar predictors, with higher LVEF being consistently protective, and major intraoperative complication being a strong adverse predictor in both analysis. Associations diverged for other covariates: in the first-procedure model, dyslipidemia remained independently protective and acute ablation success predicted lower risk, whereas cardiomyopathy

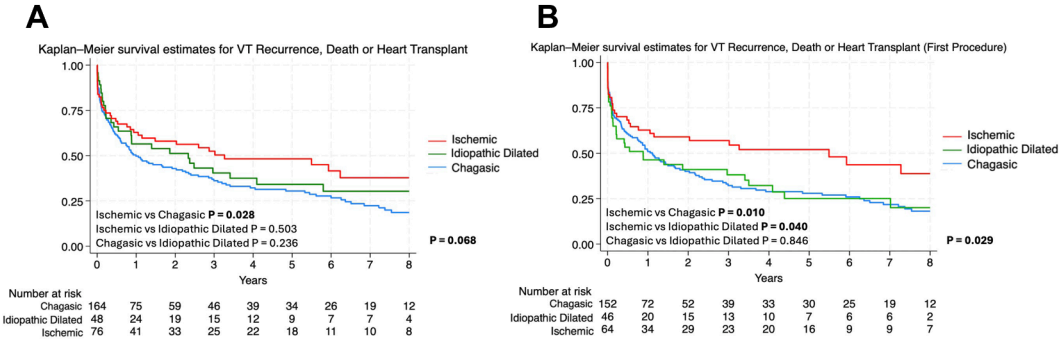

Fig. 3: Kaplan–Meier analysis of the primary composite endpoint. (A) Kaplan–Meier estimates of the composite endpoint of death, heart transplantation, or VT recurrence regarding last procedure. (B) Kaplan–Meier analysis restricted to the first procedure per patient. Outcomes were more favorable in ischemic cardiomyopathy, with more pronounced differences when only the first procedure was considered.

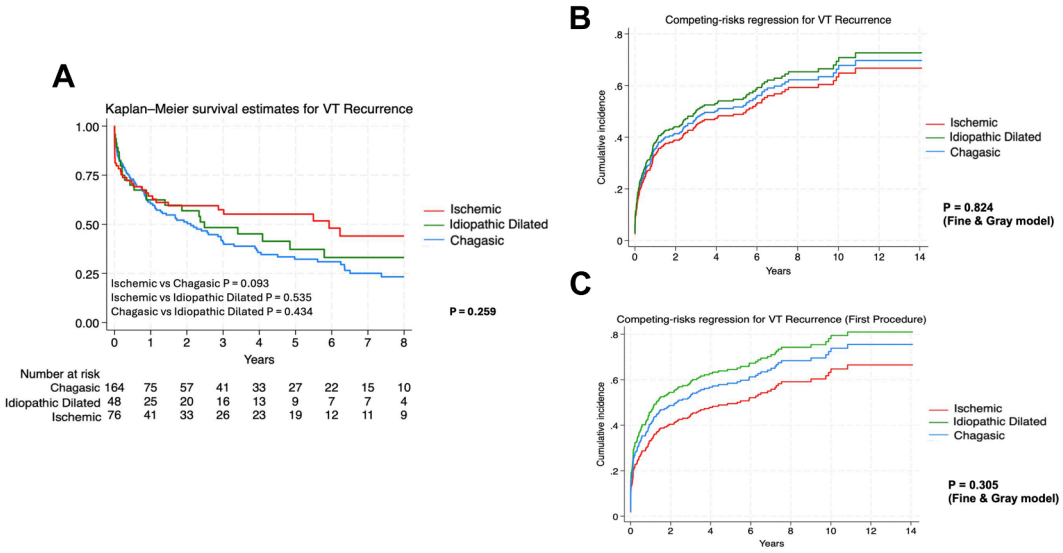

Fig. 4: VT Recurrence and competing-risk analyses. (A) Kaplan–Meier curves for VT recurrence after catheter ablation. (B) Competing-risk analysis for VT recurrence regarding the last procedure. (C) Competing-risk analysis for the first procedure. No statistically significant differences were observed among cardiomyopathy types in either analysis.

type, electrical storm and lidocaine use did not retain significance (Supplementary Table S1).

Predictors of VT recurrence

In Fine–Gray competing-risk models with death or heart transplant as competing events, a higher left ventricular ejection fraction (LVEF ≥25%) independently predicted a lower cumulative incidence of recurrence (adjusted SHR 0.44; 95% CI 0.24–0.81; p = 0.008), whereas induction of ≥2 VTs during programmed stimulation (adjusted SHR 1.63; 95% CI 1.02–2.59; p = 0.040) and having >1 ablation session (adjusted SHR 1.55; 95% CI 1.00–2.40; p = 0.049) were associated with higher risk. There were suggestive

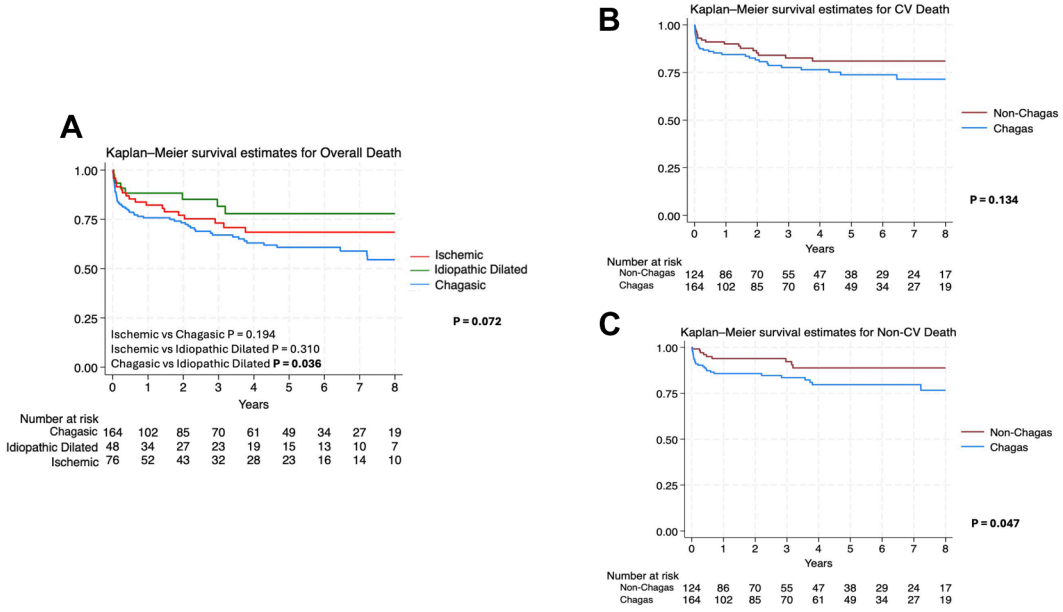

Fig. 5: Kaplan–Meier analysis of mortality. (A) Stratified analysis demonstrating higher mortality in Chagas compared with idiopathic DCM (p = 0.036), with no significant difference between Chagas and ischemic cardiomyopathy. (B) Cardiovascular mortality did not differ significantly among groups (p = 0.134). (C) Non-cardiovascular mortality was higher in Chagas compared with non-Chagas patients (p = 0.047).

Variable	Univariate HR (95% CI)	p value	Multivariable HR (95% CI)	p value
Cardiomyopathy type	1.25 (1.05–1.50)	p = 0.014	1.73 (1.16–2.59)	p = 0.008
Age (per year)	1.01 (0.99–1.02)	p = 0.46	–	–
Sex (male)	1.15 (0.83–1.59)	p = 0.42	–	–
NYHA class at admission (per class)	1.25 (1.07–1.46)	p = 0.004	1.02 (0.78–1.32)	p = 0.91
LVEF (per 1% increase)	**0.95 (0.93–0.96)**	**p < 0.001**	**0.97 (0.95–0.99)**	**p = 0.017**
Electrical storm ≤24 h	**2.24 (1.60–3.14)**	**p < 0.001**	**1.94 (1.17–3.22)**	**p = 0.011**
Dyslipidaemia	0.61 (0.45–0.82)	p = 0.001	0.87 (0.51–1.50)	p = 0.62
Chronic kidney disease	1.31 (0.97–1.78)	p = 0.078	1.24 (0.74–2.08)	p = 0.42
Active smoking	0.50 (0.18–1.34)	p = 0.17	–	–
Prior smoking	0.83 (0.60–1.14)	p = 0.24	–	–
COPD	0.71 (0.38–1.35)	p = 0.30	–	–
Hypertension	0.80 (0.60–1.07)	p = 0.13	–	–
Diabetes mellitus	0.75 (0.50–1.13)	p = 0.16	–	–
Prior myocardial infarction	0.78 (0.57–1.08)	p = 0.14	–	–
Surgical or stent revascularisation	0.99 (0.66–1.49)	p = 0.97	–	–
Atrial fibrillation	1.56 (1.12–2.16)	p = 0.009	1.05 (0.59–1.87)	p = 0.88
Stroke or TIA	1.17 (0.75–1.81)	p = 0.49	–	–
Prior amiodarone use	1.80 (1.20–2.71)	p = 0.005	0.73 (0.39–1.37)	p = 0.33
Beta-blocker use	2.18 (1.18–4.02)	p = 0.012	2.06 (0.81–5.26)	p = 0.13
In-hospital lidocaine use	**2.50 (1.77–3.52)**	**p < 0.001**	**2.24 (1.24–4.03)**	**p = 0.007**
ARB use	1.00 (0.74–1.35)	p = 0.98	–	–
ACE inhibitor use	0.91 (0.68–1.22)	p = 0.52	–	–
Spironolactone	1.50 (1.12–2.00)	p = 0.006	1.15 (0.74–1.79)	p = 0.54
Anticoagulant	1.90 (1.40–2.57)	p < 0.001	1.25 (0.72–2.17)	p = 0.43
Aspirin	**0.66 (0.48–0.90)**	**p = 0.009**	**0.53 (0.31–0.90)**	**p = 0.018**
Statin	1.01 (0.92–1.12)	p = 0.82	–	–
Furosemide	1.86 (1.38–2.49)	p < 0.001	1.50 (0.94–2.38)	p = 0.088
Number of sessions	1.57 (1.31–1.88)	p < 0.001	1.27 (0.99–1.64)	p = 0.063
Procedure time (min)	1.00 (1.00–1.00)	p = 0.054	1.00 (1.00–1.00)	p = 0.59
Acute procedural success	0.72 (0.59–0.88)	p = 0.001	0.88 (0.66–1.18)	p = 0.40
Number of induced VTs	1.16 (1.06–1.27)	p = 0.002	1.08 (0.93–1.27)	p = 0.31
VT cycle length	1.44 (1.08–1.90)	p = 0.012	1.38 (0.94–2.03)	p = 0.10
Endocardial ablation	1.31 (0.88–1.95)	p = 0.18	–	–
Epicardial access	1.23 (0.92–1.65)	p = 0.16	–	–
Retroaortic access	0.95 (0.70–1.29)	p = 0.74	–	–
Transseptal access	1.36 (0.86–2.17)	p = 0.19	–	–
Major intraprocedural complication	**14.75 (4.47–48.65)**	**p < 0.001**	**13.70 (3.27–57.39)**	**p < 0.001**
Largest scar location	1.00 (1.00–1.00)	p = 0.42	–	–
EAM use	1.20 (0.89–1.62)	p = 0.22	–	–
Technique used	1.06 (0.92–1.24)	p = 0.42	–	–

Hazard ratios (HR) are presented with corresponding 95% confidence intervals (CI). ChC: Chagas cardiomyopathy; NYHA: New York Heart Association functional class; LVEF: left ventricular ejection fraction; ED: emergency department; ICD: implantable cardioverter-defibrillator; ES: electrical storm; COPD: chronic obstructive pulmonary disease; CKD: chronic kidney disease; MI: myocardial infarction; TIA: transient ischemic attack; ACE inhibitor: angiotensin-converting enzyme inhibitor; ARB: angiotensin II receptor blocker; AF: atrial fibrillation; HR: hazard ratio; CI: confidence interval; values in bold denote statistically significant differences (p < 0.05).

Table 3: **Cox proportional hazards regression to evaluate the association between clinical and procedural variables and the composite outcome of VT recurrence, overall death or heart transplant.**

associations for amiodarone use prior to admission and in-hospital lidocaine use (adjusted SHRs 1.77 [0.91–3.43], p = 0.092; and 1.55 [0.95–2.54], p = 0.080, respectively) (Supplementary Table S2).

Predictors of all-cause death

For the outcome of all-cause death, in the multivariable Cox model, protective factors included higher left ventricular ejection fraction (adjusted HR per 1% increase 0.94, 95% CI 0.90–0.98, p = 0.003) and aspirin use (0.39, 0.16–0.94, p = 0.035). In contrast, risk factors were ChC compared with non-ChC etiologies (2.41, 1.00–5.78, p = 0.049), furosemide use (4.33, 1.75–10.75, p = 0.002), and major intraprocedural complications (84.47, 8.79–811.92, p < 0.001), the latter conferring the greatest increase in risk (Supplementary Table S3).

Discussion

This unicentric retrospective cohort with long follow-up represents, to our knowledge, the largest series of VT

ablation in ChC with direct comparison to ICM and idiopathic DCM. All procedures were performed in a high-volume center with established expertise in ventricular arrhythmia ablation.

In adjusted analyses, ChC was independently associated with a higher risk of the composite endpoint of death, transplant, or VT recurrence (HR 1.73, 95% CI 1.16–2.59) and with all-cause mortality (HR 2.41, 95% CI 1.00–5.78). In contrast, the VT recurrence rate did not significantly differ by etiology. Taken together, these findings indicate that the excess adverse outcomes observed in ChC are driven primarily by mortality rather than by arrhythmic recurrence per se.

This higher mortality rate among patients with ChC may be explained by several factors. First, these patients already have a high baseline mortality due to the severity of the underlying cardiomyopathy, regardless of whether ablation is performed. In a systematic review including 1041 patients with ChC and ICDs, an annual mortality of 9% was reported.[22] In addition, heart failure–related mortality has been shown to be higher in ChC compared with other etiologies, a pattern likely to persist in the post-procedural period despite arrhythmia control.[3,4]

Second, the strongest adjusted risk factor for overall mortality was the occurrence of intraprocedural complications. Although the confidence interval was quite wide—likely due to the small number of complications among non-ChC patients—the associated risk of death was extremely high in these cases. The occurrence of any major complication was associated with increased risk, with particular relevance to epicardial access, which was more frequent in Chagas disease. Although a direct causal link was not statistically demonstrable in our cohort, the greater need for epicardial puncture among Chagas patients may partly contribute to the higher mortality observed in this group.

Third, the higher rate of non-cardiovascular mortality in ChC suggests the contribution of comorbidity burden and social determinants of health that extend beyond arrhythmia control.[22,23]

Although the only cohort previously published comparing ChC and ICM included a much smaller number of patients, it reported findings similar to ours regarding the absence of a difference in VT recurrence rates between these two groups. Hadid et al.[24] reported the follow-up results of 38 patients with various cardiomyopathies undergoing ablation in the setting of electrical storm. Although the 16 included patients with ChC were younger and had a higher LVEF (0.40 versus 0.28; $p < 0.001$), the recurrence rate over 35 months of follow-up was similar to that of the other patients, who were predominantly of ischemic etiology (68%). Compared to Hadid et al.'s study, which reported a recurrence rate of 40%, the higher recurrence in our ChC cohort (56.7%) may be explained by the lower average LVEF and the longer follow-up period.

Across models, higher left ventricular ejection fraction was consistently protective, in line with prior reports.[25] Aspirin use was also associated with lower adjusted mortality, possibly reflecting confounding by indication given its higher prevalence among patients with ICM. In contrast, furosemide and in-hospital lidocaine use were associated with increased risk, likely serving as markers of greater disease severity and hemodynamic instability rather than causal effects. Major intraprocedural complications conferred the greatest increase in risk, underscoring the importance of meticulous pericardial management and careful vascular access planning, particularly when epicardial mapping or ablation is anticipated.

Persistent inducibility of any VT, also seen in our cohort, is a consistent marker of recurrence. Reduced LVEF likewise predicts adverse outcomes, Dyslipidemia appeared protective, but its predominance in ICM patients suggests it served as a surrogate marker not fully adjusted for multivariate analysis.

A longer VT cycle length was also linked to higher event rates. Tokuda et al.[26] showed that slower VTs are more common in repeat ablations, supporting the view that such arrhythmias reflect more advanced substrates and worse prognosis.

Non-inducibility was also lower in ChC (46% versus 62%), consistent with the more complex arrhythmogenic substrate involving epicardial and intramural circuits. Prior studies[27–29] have shown variable recurrence rates, influenced by follow-up duration, EAM use, baseline LVEF and pre-ablation amiodarone use.[30] As expected, fewer ChC patients underwent endocardial-only ablation (22% versus 85% ICM, 68% DCM). The importance of epicardial access was confirmed by Pisani et al.[16] who showed higher non-inducibility with combined endo/epicardial ablation (86% versus 40%).

The VT recurrence rates among non-ChC patients in our cohort were consistent with prior reports. In a multicenter cohort of 780 patients with NICM from 12 international centers, patients with DCM had a 1-year VT recurrence rate of 32% which is comparable to the 33% rate observed in our patients with the same cardiomyopathy over the same follow-up period.[31] Similarly, our 1-year VT recurrence rate of 34% in ICM patients is comparable to the 33% recurrence rate reported in the landmark VANISH trial.[32]

Although the overall incidence of the composite endpoint—VT recurrence, death, or heart transplantation—was high in our entire cohort, this likely reflects the combination of advanced disease severity and extended long-term follow-up. The consistency of these results with prior studies reinforces the external validity and reliability of our findings.

Complications—particularly clinically significant pericardial bleeding—were frequent and prognostically

important. Pericardial bleeding occurred in 9.8% of procedures, with 3.4% requiring surgical repair, rates comparable to prior epicardial VT ablation series (3–13%).[33–36] Major intraprocedural complications were independently associated with substantially higher risk of both the composite endpoint (adjusted HR 13.70, 95% CI 3.27–57.39; p < 0.001) and overall mortality (adjusted HR 84.47, 95% CI 8.79–811.92; p < 0.001). Two factors may plausibly contribute to this burden: first, the epicardial predominance and frequent biventricular involvement in Chagas cardiomyopathy, in which a thinner right-ventricular free wall could predispose to perforation and large-volume hemopericardium after inadvertent puncture (hypothesis-generating); and second, the operational realities of a teaching environment, where supervised trainee participation may increase procedural complexity.

Developing strategies to improve VT ablation outcomes remains a major challenge in Chagas cardiomyopathy (ChC). Functional mapping has shown promise; in a historical cohort, Wilnes et al.[18] reported a hazard ratio of ~0.10 for recurrence when decremental potentials were targeted, though prospective validation is needed. Stereotactic arrhythmia radioablation (STAR) is another innovative therapy, consistently reducing arrhythmic burden; a meta-analysis found ≥61% reduction in 95% of patients,[37] and the first Latin American case in ChC confirmed feasibility.[38] Given the high bleeding risk of epicardial access in ChC, refinements of the original Sosa technique[21]—such as the SAFER approach,[39] needle-in-needle,[40] and carbon dioxide insufflation[41]—deserve further evaluation to improve safety.

Limitations

This study has several limitations inherent to its retrospective design and reliance on medical records, with potential variability and incomplete data. Because the sample size was determined by case availability rather than a prospectively calculated target, the study may be underpowered to detect modest associations, which could be the case for the VT recurrence rate between groups. Although performed at a high-volume academic center, a proportion of procedures lacked electroanatomical mapping due to public healthcare resource constraints, which may have affected strategies and outcomes. Medication regimens and other management aspects may have changed during the up to 8-year follow-up; because time-updated data were unavailable, exposures were analyzed at baseline only. This may result in exposure misclassification over time and attenuate true associations. Potential misclassification of substrate location may have occurred in procedures performed without electroanatomic mapping; however, with fluoroscopic guidance and intracardiac electrogram criteria, gross localization is generally accurate. Any bias is more likely an underrepresentation of scar extent than incorrect assignment of the predominant scar region. We lacked consistent operator identifiers and thus could not adjust for operator-specific effects; residual confounding by operator cannot be excluded. Post-procedure testing (imaging, biomarkers, ambulatory monitoring) was not protocolized and was obtained at clinician discretion, which may introduce variability in the intensity of surveillance and event detection. Nevertheless, all patients underwent ICD interrogation at the 30-day visit. Early post-discharge retention was high across groups; however, follow-up duration varied, which reduces precision at later time points and may underdetect late events, particularly among patients from remote regions facing socioeconomic and logistical barriers to long-term care.

Conclusion

In this large single-center cohort, Chagas cardiomyopathy was independently associated with a higher adjusted risk of the composite endpoint of VT recurrence, death, or heart transplant; this excess risk was driven primarily by overall mortality, whereas VT recurrence did not differ statistically by etiology. Across models, higher LVEF was consistently protective, while markers of clinical instability (e.g., diuretic use, electrical storm at presentation) were associated with higher risk. Major intraprocedural complications conferred the greatest risk increase, underscoring the need for meticulous procedural planning and execution—particularly when epicardial access is anticipated. Collectively, these findings identify mortality as the principal challenge in Chagas cardiomyopathy after VT ablation and emphasise the importance of procedural risk mitigation and comprehensive heart-failure and comorbidity management alongside efforts to reduce arrhythmic recurrence.

Contributors

RMK: Conception and design of the study, Data collection, Data analysis and interpretation, Drafting or revising the manuscript, Access to data and responsibility for submission.

LVR: Data collection, Data analysis and interpretation, Drafting or revising the manuscript.

CFP: Conception and design of the study, Data analysis and interpretation, Drafting or revising the manuscript, Approval of the final version.

MOC: Drafting or revising the manuscript.

CAH: Drafting or revising the manuscript.

SLM: Drafting or revising the manuscript.

MIS: Conception and design of the study, Drafting or revising the manuscript, Approval of the final version.

Data sharing statement

Data available on reasonable request from the corresponding author.

Declaration of interests

RMK: Received honoraria for speaking at events (Abbott and Johnson & Johnson) and research grant support from Johnson & Johnson.

LVR: Nothing to disclose for this manuscript.

CFP: Received honoraria for speaking at events (Abbott and Johnson & Johnson) and research grants from both companies.

MOC: Received honoraria for speaking at events (Abbott and Johnson & Johnson).

CAH: Received honoraria for speaking at events (Abbott and Johnson & Johnson).

SLM: Nothing to disclose for this manuscript.

MIS: Received honoraria for speaking at events (Abbott and Johnson & Johnson) and research grants from both companies.

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Appendix A. Supplementary data

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