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Renal Dysfunction Predicts Adverse Clinical Outcomes Independent of Arrhythmic Control After VT Ablation in Structural Heart Disease

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Abstract

Background

The prognostic significance of renal dysfunction (RD) in patients undergoing catheter ablation for ventricular tachycardia (VT) in structural heart disease (SHD) remains incompletely defined. Whether RD reflects a marker of arrhythmia-independent disease severity or also compromises ablation efficacy is unknown.

Objective

To evaluate the association between RD and outcomes after VT ablation, focusing on whether RD affects arrhythmic efficacy (VT recurrence) independently of the composite endpoint of left ventricular assist device (LVAD) implantation, heart transplantation (HTx), or death.

Methods

We retrospectively analyzed 499 consecutive patients with SHD undergoing index VT ablation (2019–2025) at a tertiary center, stratified by RD status. The primary composite endpoint comprised LVAD, HTx, or death; VT recurrence was analyzed separately. Multivariable Cox regression assessed the independent association between RD and outcomes, adjusting for age, left ventricular ejection fraction (LVEF), sex, and cardiomyopathy etiology.

Results

Of 499 patients, 109 (21.8%) had RD. Patients with RD were older, had lower LVEF, and greater comorbidity burden (all $p < 0.005$). Procedural characteristics and VT recurrence were comparable between groups (47.7% vs. 47.9%, $p = 1.00$). The composite endpoint occurred more often with RD (48.6% vs. 21.3%, $p < 0.001$), with diverging event-free survival (log-rank $p < 0.001$). RD remained an independent predictor of the composite endpoint (HR 1.77, 95% CI 1.22–2.58, $p = 0.003$), consistent across LVAD (HR 1.79, $p = 0.036$), HTx (HR 2.22, $p = 0.046$), and death (HR 1.76, $p = 0.022$).

Conclusion

RD is associated with worse clinical outcomes after VT ablation despite preserved arrhythmic efficacy, and should be integrated into post-ablation risk stratification.

Introduction

Ventricular tachycardia (VT) in patients with structural heart disease (SHD) carries substantial morbidity and mortality, and catheter ablation is now an established strategy for reducing arrhythmic burden [1]. Yet despite advances in electroanatomical mapping and ablation technique, a considerable proportion of patients continue to experience adverse clinical outcomes during follow-up — including progressive heart failure, need for advanced heart failure therapies, and death [2–4]. Recent work has shown that arrhythmic efficacy and heart failure–related clinical outcomes after VT ablation do not necessarily track together: patients may achieve comparable freedom from VT recurrence yet experience markedly divergent long-term prognoses depending on the severity of underlying structural and hemodynamic disease [5, 6]. This dissociation suggests that risk stratification after VT ablation should not rely on arrhythmic endpoints alone, and that non-arrhythmic modifiers of prognosis warrant systematic investigation.

Renal dysfunction (RD) is a well-established determinant of adverse outcomes in heart failure via the cardiorenal syndrome [7, 8], but its role after VT ablation has received little attention. Whether it primarily marks arrhythmia-independent disease severity, or also compromises procedural/arrhythmic efficacy, remains unclear.

We therefore evaluated the association between RD and clinical outcomes after VT ablation in SHD, focusing on whether it affects arrhythmic efficacy (VT recurrence) independently of the composite endpoint of left ventricular assist device (LVAD) implantation, heart transplantation (HTx), or death — i.e., whether procedural success can be equated with patient clinical success.

Methods

Study Design and Population

This retrospective cohort study included 499 consecutive patients with structural heart disease (SHD) undergoing catheter ablation for sustained ventricular tachycardia (VT) between 2019 and 2025 at a tertiary electrophysiology center. Included etiologies comprised ischemic cardiomyopathy (ICM), non-ischemic cardiomyopathy (NICM), hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, and familial/genetic cardiomyopathies. Patients with congenital heart disease, channelopathies, or primary electrical disorders were excluded. In patients undergoing multiple VT ablation procedures during the study period, only the first procedure was considered the index ablation for analysis. Baseline demographic, clinical, imaging, and procedural data were obtained from institutional electronic health records at the time of the index ablation. Renal dysfunction was defined as an estimated glomerular filtration rate (eGFR) < 50 mL/min/1.73m². This threshold was chosen a priori to identify patients with clinically relevant, moderate-to-severe renal impairment (rather than the broader CKD threshold of eGFR < 60 mL/min/1.73m² used to define any chronic kidney disease), while preserving adequate group size for multivariable analysis. Sustained VT was defined according to the 2022 ESC Guidelines for the management of ventricular arrhythmias and the prevention of sudden cardiac death

[1]. The study was conducted in accordance with the Declaration of Helsinki as revised in 2013 and was approved by the local ethics committee.

Electrophysiological study and ablation strategy

All patients provided written informed consent prior to the procedure. Ablations were performed under general anesthesia via standard femoral venous access, with intravenous heparin administered during left ventricular mapping to maintain an activated clotting time ≥ 300 seconds, or prophylactic anticoagulation otherwise. A decapolar diagnostic catheter (Inquiry™, Abbott) was positioned in the coronary sinus and a quadripolar catheter (Supreme™, Abbott) at the right ventricular apex. Electroanatomical mapping (CARTO 3, Johnson & Johnson MedTech; or EnSite Precision/X, Abbott) was performed using dedicated high-density mapping catheters (OPTRELL™/OCTARAY™, J&J MedTech; Advisor HD Grid™, Abbott) via transseptal and/or retrograde aortic access as appropriate to assess chamber anatomy and substrate characteristics.

Right and left ventricular mapping was performed using transseptal and/or retrograde aortic approaches as appropriate. Programmed ventricular stimulation was used for VT induction, followed by activation mapping of hemodynamically tolerated VT whenever feasible, with pace mapping reserved for unstable arrhythmias. Epicardial access was obtained via a subxiphoid approach according to the technique described by Sosa et al. [9] when clinically indicated. Radiofrequency ablation used open-irrigated catheters (ThermoCool SMARTTOUCH SF/QDOT™, J&J MedTech; FlexAbility™/TactiCath™/TactiFlex™, Abbott) with energy delivery up to 50 W; low-voltage areas were defined as bipolar voltages of 0.1–1.5 mV. Programmed stimulation followed a standardized stepwise protocol using up to three extrastimuli and progressively shorter drive-train cycle lengths when VT remained non-inducible; the procedural endpoint was elimination of clinical VT and non-inducibility of any VT at the end of the procedure, confirmed by repeat stimulation.

Follow-up

Follow-up was defined as the period from the index ablation procedure to the most recent documented clinical evaluation and included review of inpatient and outpatient medical records as well as communication with referring physicians. Routine ICD interrogations were performed at scheduled intervals every 3–6 months and additionally through remote monitoring. The primary composite clinical endpoint comprised LVAD implantation, HTx, or all-cause death. VT recurrence, analyzed separately as a measure of arrhythmic efficacy, was defined as any documented sustained ventricular arrhythmia, including sustained VT or ventricular fibrillation, irrespective of termination by antitachycardia pacing or ICD therapy.

Statistical analysis

Continuous variables are presented as mean ± SD and compared using Student's t-test; categorical variables as frequencies/percentages, compared using chi-square. Time-to-event analysis used the Kaplan–Meier method with log-rank comparison. Multivariable Cox regression assessed the independent association between RD and the composite endpoint, adjusting for age, LVEF (per 5% increment), sex, and etiology (ischemic vs. non-ischemic; VIF < 1.2 for all covariates), and was applied separately to each individual component (LVAD, HTx, death). As a sensitivity analysis, the primary model for the composite endpoint was additionally adjusted for NYHA class (III–IV vs. I–II), diabetes mellitus, and atrial fibrillation (Supplementary Table 3). Model discrimination used Harrell's C-statistic; two-sided $p < 0.05$ was considered significant. As an exploratory, descriptive analysis susceptible to immortal time bias, patients were additionally stratified jointly by RD and VT recurrence status ascertained during follow-up. A complete-case approach was used; all analyses were performed in SPSS version 24.0 (IBM Corp., Chicago, IL, USA).

Results

Baseline Characteristics

Of 499 patients undergoing catheter ablation for VT, 109 (21.8%) had RD at baseline (Table 1). Patients with RD were older (66.2 vs. 62.3 years, $p = 0.001$), had lower LVEF (28.0% vs. 36.1%, $p < 0.001$) and larger LVEDD, and carried a higher burden of comorbidity, including NYHA III–IV symptoms, atrial fibrillation, hypertension, diabetes, and COPD, and more frequent CRT-D use, consistent with more advanced heart failure (Table 1).

Table 1

Baseline, procedural, and outcome characteristics according to renal function status *Continuous variables presented as mean ± SD unless otherwise noted; categorical variables as percentages. RD = renal dysfunction.*

Baseline Characteristics			
Variable	**No RD (n = 390)**	**RD (n = 109)**	**p-value**
Age, years	62.3 ± 12.9	66.2 ± 10.0	**0.001**
Male sex	89.2%	88.1%	0.87
BMI, kg/m²	28.0 ± 6.6	28.6 ± 5.1	0.30
LVEF, %	36.1 ± 13.7	28.0 ± 11.1	**< 0.001**
LVEDD, mm*	60.6 ± 11.4	64.2 ± 11.0	**0.009**
ICM	55.0%	67.9%	**0.022**
NICM	45.0%	32.1%	**0.022**
NYHA III–IV	33.1%	57.8%	**< 0.001**
Atrial fibrillation	35.6%	56.0%	**< 0.001**
Hypertension	53.8%	70.6%	**0.003**
Diabetes mellitus	17.4%	29.4%	**0.009**
Prior stroke/TIA	11.0%	12.8%	0.72
COPD	8.5%	15.6%	**0.044**
ICD (excl. CRT-D)	62.3%	46.8%	**0.005**
CRT-D	28.2%	48.6%	**< 0.001**
ICD shocks pre-ablation	65.4%	73.4%	0.14
Procedural Characteristics			
Variable	**No RD (n = 390)**	**RD (n = 109)**	**p-value**
Ablation duration, min†	134 [109–165]	134 [110–168]	0.56
Mapping time, min†	24 [14–37]	24 [15–32]	0.33
No. of induced VTs	1.6 ± 1.1	1.9 ± 1.0	**0.003**
Procedural complications	1.5%	2.8%	0.66
Clinical Outcomes			
Variable	**No RD (n = 390)**	**RD (n = 109)**	**p-value**
VT recurrence	47.9%	47.7%	1.00

Baseline Characteristics			
Variable	**No RD (n = 390)**	**RD (n = 109)**	**p-value**
Redo ablation	17.5%	8.3%	**0.030**
LVAD implantation	7.9%	22.9%	**< 0.001**
Heart transplantation	6.4%	11.9%	0.086
Death	11.5%	29.4%	**< 0.001**
Composite endpoint‡	21.3%	48.6%	**< 0.001**
** LVEDD available in n = 392/499 (78.6%; 107 patients with missing baseline echocardiographic assessment excluded).*			
† Presented as median [interquartile range]; compared using the Mann-Whitney U test.			
‡ Composite endpoint: left ventricular assist device (LVAD) implantation, heart transplantation, or death, whichever occurred first (see Methods, Follow-up and outcome definitions). Presented alongside VT recurrence (arrhythmic outcome) to distinguish arrhythmic efficacy from non-arrhythmic clinical outcome.			
BMI = body mass index; COPD = chronic obstructive pulmonary disease; ICD = implantable cardioverter-defibrillator; CRT-D = cardiac resynchronization therapy with defibrillator; ICM = ischemic cardiomyopathy; LVAD = left ventricular assist device; LVEDD = left ventricular end-diastolic diameter; LVEF = left ventricular ejection fraction; NICM = non-ischemic cardiomyopathy; NYHA = New York Heart Association; RD = renal dysfunction; TIA = transient ischemic attack; VT = ventricular tachycardia.			

Procedural Characteristics and Arrhythmic Efficacy

Despite this more advanced baseline risk profile, acute procedural characteristics did not differ meaningfully between groups: ablation duration, mapping time, and complication rates were comparable (all $p > 0.3$; Table 1). Mean follow-up was 15.6 ± 20.0 months, similar between groups ($p = 0.24$). Critically, VT recurrence was virtually identical between groups (47.7% vs. 47.9%, $p = 1.00$), indicating that RD did not compromise arrhythmic efficacy. Patients with RD underwent redo ablation less frequently (8.3% vs. 17.5%, $p = 0.030$), consistent with competing clinical events (LVAD/HTx/death) occurring before a redo could be considered.

Clinical Outcomes

In contrast to the comparable arrhythmic outcomes, patients with RD experienced substantially worse clinical outcomes during follow-up. The composite endpoint — LVAD implantation, HTx, or all-cause death, whichever occurred first — occurred in 48.6% of patients with RD compared with 21.3% of patients without ($p < 0.001$). Kaplan–Meier analysis demonstrated significantly lower event-free survival in

patients with RD (log-rank $p < 0.001$), with event-free rates of 71% vs. 89% at 12 months, 51% vs. 80% at 24 months, and 31% vs. 69% at 36 months (Fig. 1).

Multivariable Predictors of the Composite Endpoint

On multivariable Cox regression adjusting for age, LVEF, sex, and etiology, RD remained an independent predictor of the composite endpoint (HR 1.77, 95% CI 1.22–2.58, $p = 0.003$; Supplementary Table 2), as were lower LVEF (HR 0.77 per 5% increase, $p < 0.001$) and female sex (HR 1.99, $p = 0.005$); age and ICM were not independently associated. Model discrimination was acceptable (Harrell's C = 0.722, indicating correct risk-ordering in approximately 72% of comparable patient pairs). A landmark analysis confirmed RD remained predictive at 1 and 3 months (HR 1.67 and 1.85, respectively; both $p \leq 0.012$), indicating the association was not confined to the early post-procedural period. In a sensitivity analysis additionally adjusting for NYHA class, diabetes, and atrial fibrillation, RD remained an independent predictor of the composite endpoint (HR 1.54, 95% CI 1.05–2.27, $p = 0.028$; Harrell's C = 0.730; Supplementary Table 3).

Predictors of Individual Endpoint Components

RD was independently associated with each individual component when modeled separately: LVAD implantation (HR 1.79, $p = 0.036$), HTx (HR 2.22, $p = 0.046$), and all-cause death (HR 1.76, $p = 0.022$), arguing against a signal driven by a single outcome category (full model in Supplementary Table 2).

To assess whether the excess risk was mediated by arrhythmic control, patients were stratified jointly by RD and VT recurrence status (Fig. 2). The composite endpoint occurred in 12.3% of patients without RD/VT recurrence, 31.4% without RD but with VT recurrence, 35.7% with RD but without VT recurrence, and 61.5% with both (overall log-rank $p < 0.001$). RD without recurrence still predicted worse outcome than no RD/no recurrence (35.7% vs. 12.3%; $p = 0.005$), indicating that RD predicts adverse outcome even without arrhythmic failure.

Cause of Death

To explore the mechanism underlying excess mortality in RD, documented causes of death were reviewed (Supplementary Table 1; documented in 46/77 deaths, 60%). Most were attributable to terminal heart failure (39%) or infectious/device-related complications (35%), while only 17% were arrhythmia-related — supporting that RD predicts non-arrhythmic disease progression rather than arrhythmic treatment failure.

Discussion

In this cohort of 499 patients with SHD undergoing VT ablation, RD was associated with substantially worse long-term clinical outcomes despite preserved arrhythmia-related efficacy. Four principal findings

emerged. First, VT recurrence was virtually identical between patients with and without RD, indicating that renal impairment did not compromise the procedural or arrhythmic efficacy of ablation. Second, despite this comparable arrhythmic outcome, RD independently predicted a nearly 2-fold higher risk of the composite endpoint of LVAD implantation, HTx, or death, after adjustment for age, LVEF, sex, and cardiomyopathy etiology. Third, this association held consistently across all three individual components, arguing against a signal driven by a single outcome category. Fourth, only a minority of documented deaths were attributable to refractory arrhythmia, further supporting that RD primarily reflects progression of advanced heart failure rather than failure of the ablation procedure itself.

Renal dysfunction as a non-arrhythmic determinant of outcome

VT recurrence is the efficacy metric most commonly reported in ablation studies, implicitly assuming that arrhythmia control is the principal determinant of long-term prognosis. Our findings challenge this assumption in patients with RD: despite closely comparable rates of VT recurrence and procedural complications, patients with RD experienced markedly divergent clinical trajectories, with event-free survival diverging early and progressively over follow-up (Fig. 1). The lower redo-ablation rate observed in patients with RD most plausibly reflects competing clinical deterioration — via LVAD implantation, HTx, or death — rather than a genuinely lower arrhythmia burden. These findings suggest that non-arrhythmic disease burden, whether reflected by ventricular or renal dysfunction, constitutes an independent axis of risk that is not adequately captured by arrhythmia-focused endpoints alone. Consistent with this interpretation, RD independently predicted all three individual components of the composite endpoint rather than a single outcome category, arguing against a single unifying mechanism and instead supporting RD as a broad marker of vulnerability to multiple modes of adverse outcome. This is corroborated by the cause-of-death analysis, in which most fatal events reflected progressive heart failure or infectious/device-related complications rather than refractory arrhythmia: taken together, these findings suggest that renal dysfunction identifies a phenotype in whom ablation successfully suppresses ventricular arrhythmias but does not alter the underlying trajectory of heart failure.

Cardiorenal interaction and comparison with prior literature

The bidirectional relationship between cardiac and renal function is well established in the heart failure literature, where impaired renal function is consistently associated with increased mortality and cardiovascular events, reflecting hemodynamic, neurohormonal, and inflammatory mechanisms that mutually accelerate disease progression in both organs [7, 8]. Our findings extend this cardiorenal paradigm to the VT ablation population: RD tracked with a distinctly more advanced baseline risk profile — including lower LVEF, larger LVEDD, more frequent NYHA III–IV symptoms, atrial fibrillation, and CRT-D use — consistent with RD functioning as a marker of advanced structural and hemodynamic disease rather than a driver of arrhythmia recurrence itself. Notably, the prognostic impact of RD was consistent across the spectrum of left ventricular function, with no significant interaction between RD and LVEF (p

= 0.59), indicating that RD identifies high-risk patients independent of the severity of underlying ventricular dysfunction. RD also remained an independent predictor after additional adjustment for NYHA class, diabetes, and atrial fibrillation (Supplementary Table 3), arguing against confounding by these established markers of overall disease burden. This is further supported by contrast with reversible arrhythmia-induced cardiomyopathy, in which renal function has been shown to improve following successful arrhythmia suppression [10] — a mechanism unlikely to apply here, since scar-related VT in structural heart disease is a consequence rather than a cause of the underlying myocardial substrate. Our findings are consistent with prior work identifying RD as an independent predictor of adverse outcome after VT ablation: in a cohort of 328 patients with SHD, Aldhoon et al. incorporated impaired renal function, alongside LV dysfunction, older age, and higher NYHA class, into a simple bedside mortality risk score [11]. Notably, established periprocedural tools such as the PAINESD score, developed to predict acute hemodynamic decompensation, do not include a renal function component [12]; our data suggest that renal function may capture longer-term prognostic information not reflected in current periprocedural risk scores and could represent a candidate variable for future risk-model refinement.

Age, sex, and other predictors of outcome

Beyond RD, our expanded multivariable analysis (Supplementary Table 2) identified two additional predictors that merit brief comment. Younger age was independently associated with HTx (HR 0.96 per year, p = 0.001), consistent with established transplant-eligibility practice favoring younger candidates. Female sex was independently associated with all-cause mortality (HR 2.62, p < 0.001) but not with higher rates of LVAD implantation (HR 1.83, p = 0.14) or HTx (HR 1.35, p = 0.63). Although this asymmetry may reflect sex-related differences in referral for advanced heart failure therapies, our study was not designed to address this question, and the finding should be considered hypothesis-generating.

Clinical implications

These findings suggest that renal function should inform patient counseling and post-ablation management planning, independent of anticipated arrhythmic outcome. Management should therefore extend beyond arrhythmia control: patients with RD undergoing VT ablation may benefit from a structured, multidisciplinary pathway instituted around the time of ablation — including earlier heart failure referral and follow-up, earlier evaluation for LVAD or transplant candidacy, systematic nephrology involvement to guide volume management, and proactive optimization of guideline-directed medical therapy where tolerated. Renal function may also warrant consideration as a candidate variable in future post-ablation risk-stratification tools, complementing existing scores that focus primarily on periprocedural hemodynamic risk.

Limitations

This study has several limitations: its retrospective, single-center design limits generalizability; RD was assessed as a binary baseline variable, precluding dose-response or trajectory analysis; and the observational design precludes causal inference, as RD may mark overall disease severity rather than an

independent causal contributor. A specific cause of death was documented for only 60% of deaths, limiting the cause-of-death analysis. Finally, interim renal function changes, dialysis initiation, and nephrology co-management during follow-up were not captured.

Conclusions

Among patients undergoing VT ablation for SHD, RD independently predicted substantially worse clinical outcomes despite preserved arrhythmic efficacy, consistently across LVAD implantation, HTx, and death. Renal function provides prognostic information beyond arrhythmia control and should be integrated into post-ablation risk stratification.

Abbreviations

COPD: chronic obstructive pulmonary disease; CRT-D: cardiac resynchronization therapy with defibrillator; HTx: heart transplantation; ICD: implantable cardioverter-defibrillator; ICM: ischemic cardiomyopathy; LVAD: left ventricular assist device; LVEDD: left ventricular end-diastolic diameter; LVEF: left ventricular ejection fraction; NICM: non-ischemic cardiomyopathy; NYHA: New York Heart Association; RD: renal dysfunction; SHD: structural heart disease; VT: ventricular tachycardia.

Declarations

Disclosures:

- C.S. received research support and lecture fees from Medtronic, Abbott, Boston Scientific, and J&J MedTec; is a consultant for Medtronic, Boston Scientific, and J&J MedTec.

- P.S. is Member of Advisory Board for Abbott, Boston Scientific, J&J MedTech and Medtronic.

All other authors state: None

Competing Interests

C.S. received research support and lecture fees from Medtronic, Abbott, Boston Scientific, and J&J MedTec; is a consultant for Medtronic, Boston Scientific, and J&J MedTec.P.S. is Member of Advisory Board for Abbott, Boston Scientific, J&J MedTech and Medtronic.

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Author Contribution

A.D. conceptualized and designed the study. V.N. collected and curated the data. A.D. performed the statistical analyses. A.D. and V.N. interpreted the results. A.D. drafted the manuscript. All authors critically revised the manuscript for important intellectual content and approved the final version of the work.

Data Availability

Data will be made available from the authors on reasonable request.

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Figures

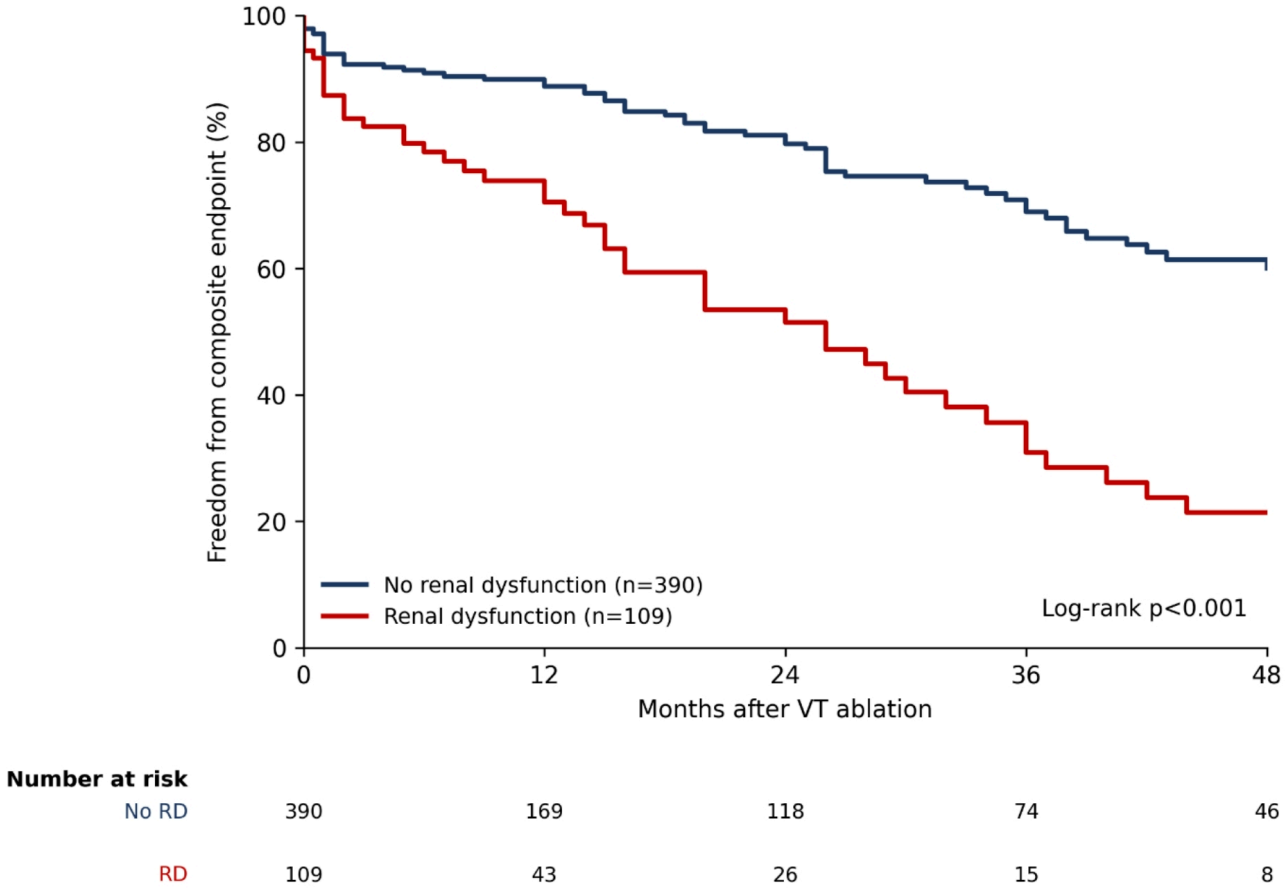
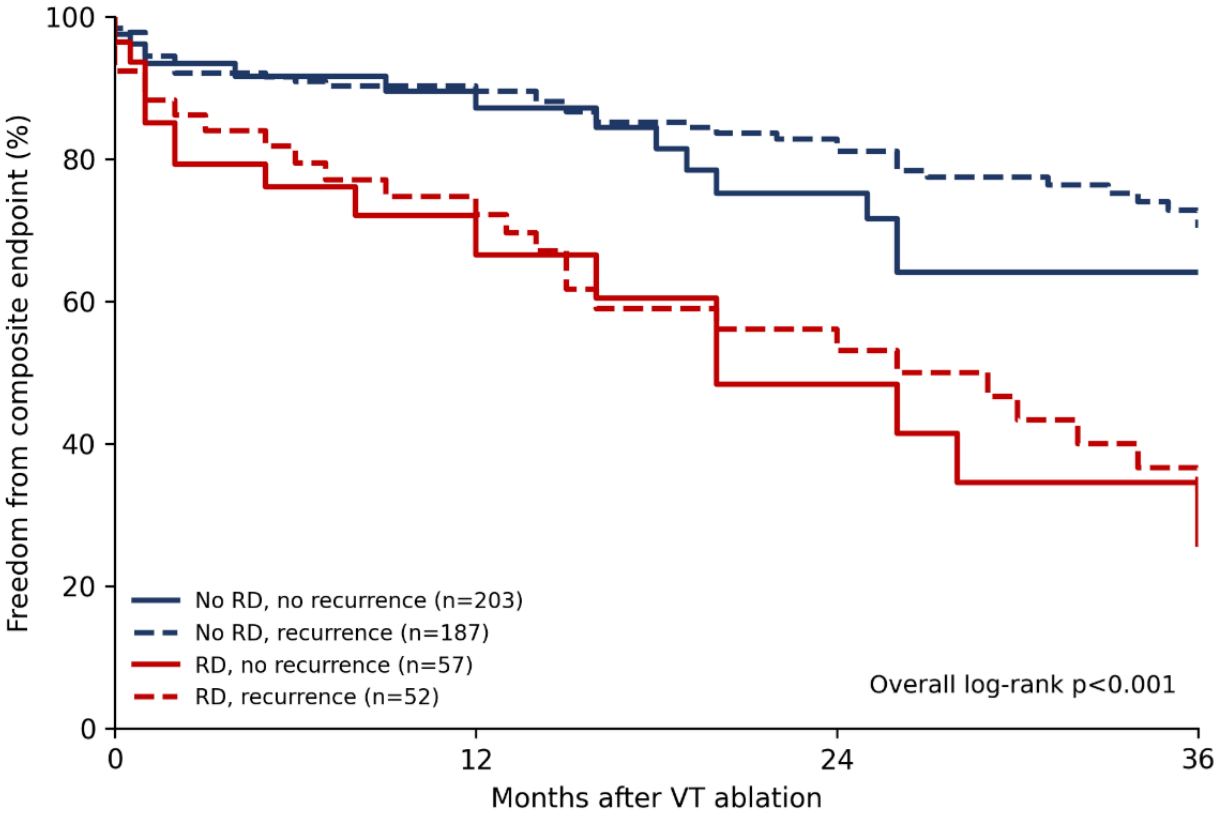


Figure 1

Kaplan–Meier estimates of freedom from the composite endpoint (LVAD implantation, HTx, or death) by renal function status (no RD, n=390; RD, n=109; log-rank p<0.001).



Number at risk				
No RD/no recur	203	38	21	14
No RD/recur	187	131	97	60
RD/no recur	57	13	7	4
RD/recur	52	30	19	11

Figure 2

Kaplan–Meier estimates of the composite endpoint (LVAD, HTx, or death) by RD and VT recurrence status (n=203/187/57/52; overall log-rank p<0.001). RD without recurrence still predicted worse outcome than no RD/no recurrence (p=0.005), indicating an arrhythmia-independent effect.

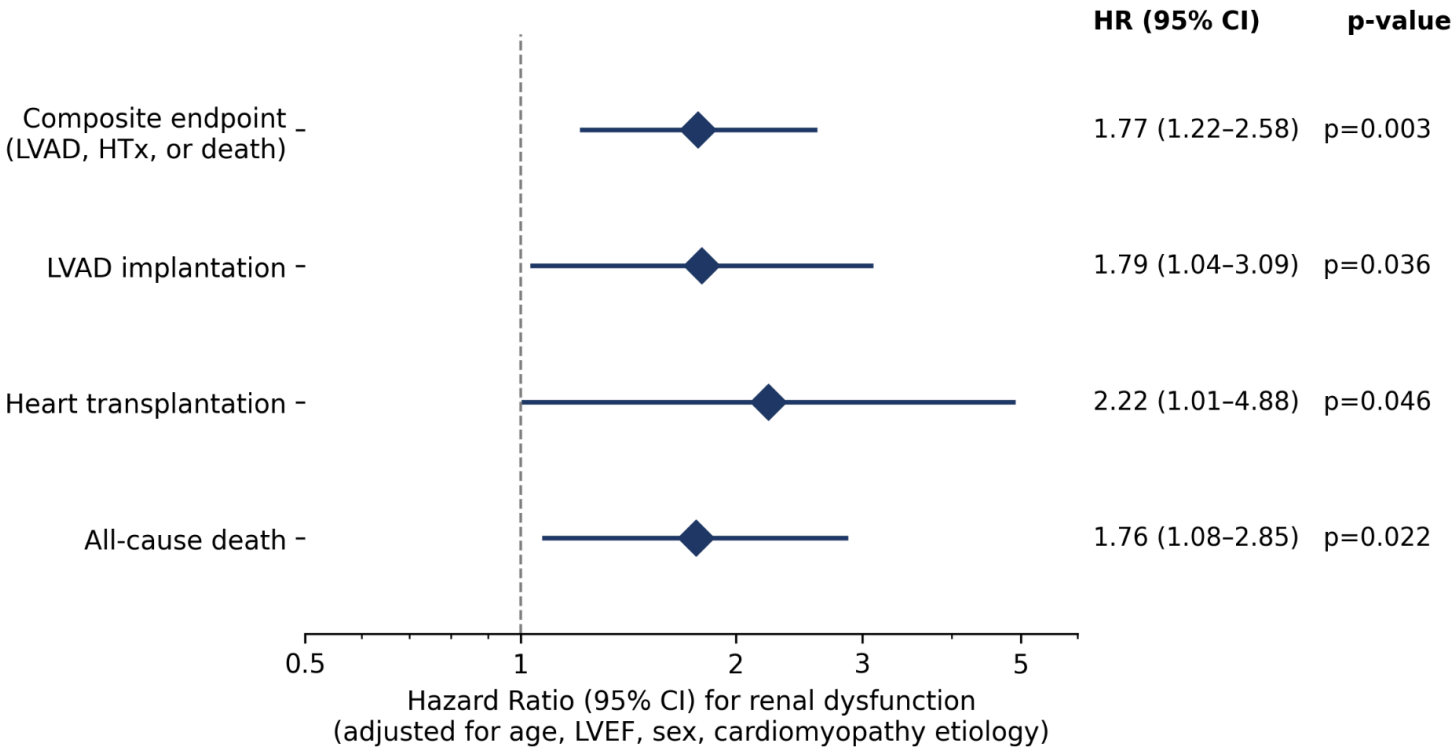


Figure 3

Forest plot of adjusted hazard ratios for renal dysfunction predicting the composite endpoint and its components (adjusted for age, LVEF, sex, etiology; see Supplementary Table 2). Diamonds = point estimates; lines = 95% CI; dashed line = HR 1.

Supplementary Files

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- Supplementarymaterial.docx