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Original Article

Identification of Long-Term Mortality Predictors and Risk Score Development in Left Ventricular Assist Device Recipients

Berhan Keskin,[1] Aykun Hakgör,[1] İbrahim Demir,[2] Korhan Erkanlı,[2] Beytullah Çakal,[1] Yahya Yıldız,[3] Bilal Boztosun,[1] İbrahim Oğuz Karaca[1]

[1]Department of Cardiology, İstanbul Medipol University, Medipol Mega University Hospital, İstanbul, Türkiye
[2]Department of Cardiovascular Surgery, İstanbul Medipol University, Medipol Mega University Hospital, İstanbul, Türkiye
[3]Department of Anesthesiology, İstanbul Medipol University, Medipol Mega University Hospital, İstanbul, Türkiye

Abstract

Objective: Long-term mortality remains a significant concern in patients receiving left ventricular assist devices (LVADs). Identifying reliable prognostic factors and developing a validated risk score could improve patient selection and long-term management.

Methods: In this retrospective single-center study, 35 patients who underwent LVAD implantation between August 2019 and May 2025 were evaluated. Patients who died during the index hospitalization were excluded. Baseline clinical, laboratory, echocardiographic, and hemodynamic parameters were collected. Long-term mortality predictors were identified using Cox regression analyses. A risk score (CACA score) was developed based on significant predictors.

Results: Four variables – age, prior coronary artery bypass grafting (CABG), creatinine, and albumin levels – were associated with long-term mortality in univariate analysis. Among these, only age remained an independent predictor in multivariate analysis (hazard ratio [HR]: 1.48; 95% CI: 1.03–2.12; p=0.032). The CACA risk score was derived using the β-coefficients from multivariate analysis. Patients were stratified into low, intermediate, and high-risk groups with corresponding mortality rates of 0%, 45.4%, and 75%, respectively. The CACA score demonstrated superior discriminative ability compared to individual variables (area under the curve: 0.88).

Conclusion: Age, prior CABG, renal function, and albumin levels are key predictors of long-term mortality in LVAD recipients. The proposed CACA score effectively stratifies mortality risk and may serve as a practical tool for clinical decision-making.

Keywords: Advanced heart failure; left ventricular assist device; mortality, risk score.

Sol Ventrikül Destek Cihazı Bulunan Hastalarda Uzun Dönem Mortalite Belirleyicilerinin Tanımlanması ve Risk Skoru Geliştirilmesi

Özet

Amaç: Uzun dönem mortalite, sol ventrikül destek cihazı (LVAD) takılan hastalarda önemli bir problem olmaya devam etmektedir. Güvenilir prognostik faktörlerin belirlenmesi ve geçerli bir risk skorunun geliştirilmesi, hasta seçimini ve uzun dönem yönetimi iyileştirebilir.

Yöntem: Bu retrospektif, tek merkezli çalışmaya, Ağustos 2019 ile Mayıs 2025 tarihleri arasında LVAD implantasyonu yapılan 35 hasta dahil edilmiştir. İndeks hastane yatışı sırasında hayatını kaybeden hastalar çalışma dışında bırakılmıştır. Hastaların klinik, laboratuvar, ekokardiyografik ve hemodinamik verileri retrospektif olarak analiz edilmiş, uzun dönem mortalite ile ilişkili faktörler Cox regresyon analizi ile belirlenmiştir. İstatistiksel anlamlı prediktörler kullanılarak CACA isimli bir risk skoru oluşturulmuştur.

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Address for Correspondence:
Berhan Keskin

Department of Cardiology, İstanbul Medipol University, Medipol Mega University Hospital, İstanbul, Türkiye

E-mail: berhankeskin92@gmail.com

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Bulgular: Yaş, geçirilmiş koroner arter bypass cerrahisi (CABG), kreatinin ve albümin düzeyleri tek-değişkenli Cox regresyon analizinde uzun dönem mortalite ile ilişkili bulunmuştur. Çok değişkenli analizde ise sadece yaş bağımsız bir prediktör olarak kalmıştır (HR: 1.48; 95% GA: 1.03–2.12; p = 0.032). CACA skoru, çok değişkenli analize alınan değişkenlerin β-katsayılarına göre oluşturulmuştur. Skor ile hastalar düşük, orta ve yüksek riskli gruplara ayrılmış ve bu gruplarda sırasıyla %0, %45.4 ve %75 mortalite oranları saptanmıştır. CACA skoru, ROC analizinde bireysel değişkenlere kıyasla daha yüksek ayırt edici güç göstermiştir (AUC: 0.88).

Sonuç: Yaş, geçirilmiş CABG, renal fonksiyon ve albümin düzeyleri, LVAD takılan hastalarda uzun dönem mortaliteyi öngören başlıca faktörlerdir. Geliştirilen CACA skoru, mortalite riskini etkili bir şekilde sınıflandırmakta olup klinik karar süreçlerinde pratik bir araç olarak kullanılabilir.

Anahtar sözcükler: İleri evre kalp yetersizliği; sol ventrikül destek cihazı; mortalite; risk skoru.

Introduction

Left ventricular assist devices (LVADs) have become an established therapy for patients with advanced heart failure who are not eligible for transplantation, offering improved survival and quality of life compared with optimal medical treatment.[1] Despite this knowledge and advancements in device technology, mortality rates remain still high in this population. Historical data, such as the REMATCH trial, reported 1- and 2-year survival rates of only 52% and 23%, respectively.[1] These patients often face complications, such as right ventricular failure, bleeding, stroke, arrhythmic events, and device-related issues, including infection, pump thrombosis, and suction events, all of which contribute to the multifactorial causes of death.

The presence of multifactorial causes of death makes it challenging to identify objective risk factors for mortality in LVAD recipients. The etiologies of short-term and long-term mortality differ among these patients. For instance, INTERMACS status has been shown to strongly predict early mortality after LVAD implantation;[2,3] however, its predictive value for long-term mortality diminishes in previous studies.[4–10] Similarly, early post-operative sepsis accounts for substantial early mortality, while later deaths are more often related to chronic comorbidities, bleeding, or device-related issues. Predictors of long-term mortality remain controversial and vary across studies.[4–11] Several risk scores incorporating laboratory, clinical, and hemodynamic parameters have been proposed,[4–11] but their discriminatory and predictive power for long-term mortality in LVAD recipients remains uncertain. The present study, therefore, sought to evaluate potential determinants of long-term mortality among LVAD patients and to develop a new risk score to support individualized risk stratification.

Materials and Methods

This retrospective, single-center study included patients who underwent implantation of a LVAD as destination therapy for advanced heart failure with reduced ejection fraction between August 1, 2019, and May 30, 2025. Patients who died during the early post-operative hospitalization period were excluded (n=9), resulting in a final cohort of 35 adult patients. All data were retrospectively retrieved from the hospital electronic medical records and individual patient charts, recorded at the time of admission.

Clinical information included demographic characteristics, existing comorbidities, ongoing medication regimens, and baseline laboratory results. In addition, comprehensive echocardiographic assessments and right and left heart catheterization measurements were obtained.

Transthoracic echocardiography was performed with a Vivid E95 ultrasound platform (General Electric Vingmed Ultrasound, Milwaukee, WI) using 3.5-MHz or M5S transducers. Parameters evaluated included left ventricular ejection fraction (LVEF) via the biplane Simpson method, left-sided chamber dimensions, and valvular pathologies. Pulmonary artery systolic pressure (PASP) was estimated from tricuspid regurgitant jet velocity using the Bernoulli equation with right atrial pressure estimation. Right ventricular function was assessed by tricuspid annular plane systolic excursion (TAPSE) and tissue Doppler-derived systolic velocity of the tricuspid annulus (RV-St). Measurements followed the standards of the American Society of Echocardiography and the European Association of Cardiovascular Imaging.[12]

All patients underwent right and left heart catheterization the day before LVAD implantation. Vascular access was obtained from the femoral vessels under ultrasound guidance. Pressure measurements were standardized with the mid-thoracic line as the zero reference. Aortic and intracardiac pressures were obtained using 6F pigtail catheters, while pulmonary capillary wedge pressure (PCWP) was recorded with a Swan-Ganz catheter. Cardiac output was determined by the Fick principle, and stroke volume, stroke volume index, and cardiac index were calculated according to body surface area and heart rate.

Derived indices of right ventricular–pulmonary arterial coupling were computed as follows:

- Right ventricular stroke work index (RVSWI): (mean pulmonary artery pressure – mean right atrial pressure) × stroke volume index,
- Pulmonary artery pulsatility index (PAPi): (systolic pulmonary artery pressure – diastolic pulmonary artery pressure) / mean right atrial pressure,
- RAP/PCWP ratio: Mean right atrial pressure/PCWP.

This study was approved by the İstanbul Medipol University (Date: 11.07.2025, Decision no: 810). This study was conducted in accordance with the Declaration of Helsinki. All procedures complied with the Declaration of Helsinki. Written informed consent for scientific use of clinical data was obtained from each participant at the time of hospital admission and before invasive procedures.

Statistical Analysis

Normality of variable distribution was evaluated using the Kolmogorov–Smirnov test and inspection of histograms. Continuous data were expressed as mean±standard deviation for normally distributed variables or as median with interquartile

Table 1. Univariate Cox regression analysis for the prediction of long-term mortality

Variables	HR	95% CI	p
Clinical characteristics			
INTERMACS class (1–3 vs. 4–7)	0.41	0.11–1.46	0.17
Age (per 1 year)	1.10	1.04–1.18	0.001
Sex (Male)	0.46	0.1–2.11	0.32
Body mass index (per 1 kg/m^2)	0.99	0.90-1.08	0.85
LVAD Type (HeartMate 2 vs. HeartMate 3)	0.69	0.23-2.07	0.51
VT/VF during follow-up	2.68	0.74–9.66	0.13
Pre-existing hypertension	0.90	0.25–3.26	0.88
Pre-existing diabetes mellitus	1.09	0.38–3.12	0.87
Pre-existing atrial fibrillation	1.20	0.40–3.62	0.73
Previous CAD	2.00	0.55–7.23	0.28
Previous PCI	0.60	0.21–1.74	0.35
Previous CABG	6.47	1.92–21.71	0.002
Bleeding events during follow-up	1.33	0.37–4.80	0.66
Regular medications			
ACEi/ARB usage	0.15	0.02-1.22	0.07
SGLT-2i usage	1.04	0.36–3.01	0.94
MRA usage	0.43	0.13–1.38	0.16
Diuretic usage	1.65	0.36-7.42	0.51
Laboratory parameters			
Hemoglobin (per 1 mg/dL)	0.91	0.69–1.18	0.47
CRP (per 1 mg/dL)	1.005	0.99–1.01	0.37
Creatinine (per 1 mg/dL)	1.73	1.005–2.98	0.047
AST (per 1 IU/L)	0.99	0.99–1.001	0.473
Albumin (per 1 g/dL)	0.32	0.14–0.73	0.006
Sodium (per 1 mEq/L)	0.91	0.79–1.05	0.23
Laboratory parameters			
Total bilirubin (per 1 mg/dL)	1.31	0.62–2.79	0.47
TSH (per 1 mIU/L)	0.62	0.37–1.01	0.057
Neutrophil-to-lymphocyte ratio	1.02	0.97-1.06	0.40
Lymphocyte-to-monocyte ratio	0.57	0.30-1.07	0.08
Echocardiographic parameters			
LVEF (per 1%)	1.14	0.96–1.36	0.13
LVEDD (per 1 cm)	0.80	0.36–1.75	0.58
TAPSE/PASP ratio (per 0.1 mm/mmHg)	0.21	0.001–43.08	0.571
RV-St (per 1 cm/sn)	1.07	0.66–1.75	0.77
RV basal diameter (per 1 cm)	1.39	0.38–5.05	0.61
Cardiac catheterization measurements			
Cardiac output (per 1 L/min)	1.25	0.76–2.06	0.37
Cardiac index (per 1 L/min/m^2)	2.29	0.74–7.07	0.14
Stroke volume (per 1 mL/beat)	1.02	0.98–1.06	0.244
Stroke volume index (per 1 mL/beat/m^2)	1.07	0.98–1.18	0.112
PCWP (per 1 mmHg)	0.96	0.89–1.04	0.332
PASP (per 1 mmHg)	0.98	0.95–1.02	0.52
PAPM (per 1 mmHg)	0.97	0.92–1.02	0.33
PAPD (per 1 mmHg)	0.96	0.89–1.03	0.31
PVR (per 1 Woods unit)	1.05	0.79–1.39	0.73
RAP	0.92	0.79-1.07	0.28
RAP/PCWP (per 0.1 increase)	0.46	0.01-25.85	0.70
RVSWI (per 100 mmHg·mL/m^2)	1.00	0.99-1.002	0.73
PAPi (per 1 increase)	1.15	0.81–1.62	0.42

HR: Hazard ratio; CI: Confidence interval; RAP: Right atrial mean pressure; PCWP: Pulmonary capillary wedge pressure, INTERMACS: Interagency registry for mechanically assisted circulatory support; ICD: Implantable cardioverter defibrillator; PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass graft surgery; CAD: Coronary artery disease; LVAD: Left ventricular assist device; ACEi: Angiotensin converting enzyme inhibitors; ARB: Angiotensin II receptor blocker; SGLT-2i: Sodium-glucose cotransporter-2 inhibitors; MRA:Mineralocorticoid receptor antagonist; AST: Aspartate aminotransferase; CRP: C-reactive protein; LVEF: Left ventricular ejection fraction; LVEDD: Left ventricular end-diastolic diameter; TAPSE: Tricuspid annular plane systolic excursion; PASP: Pulmonary artery systolic pressure; RV-St: Peak systolic velocity of tricuspid annulus by Tissue Doppler Imaging; PAPM: Pulmonary artery mean pressure; PAPD: Pulmonary artery diastolic pressure; PVR: Pulmonary vascular resistance; RVSWI: Right ventricular stroke work index; PAPi: Pulmonary artery pulsatility index; VT: Ventricular tachycardia; VF: Ventricular fibrillation.

ranges for skewed variables. Categorical data were presented as counts and percentages.

Patients were grouped according to survival status at long-term follow-up. Comparisons between groups were performed using the Chi-square test for categorical variables and the Mann–Whitney U test for continuous variables. The primary outcome was all-cause long-term mortality.

Univariate Cox proportional hazards regression was applied to identify potential predictors. Variables reaching significance were subsequently included in a multivariate Cox regression model. Forest plots were used to visualize the results of multivariate analysis.

A composite risk score was created from the β-coefficients of the multivariate model. Patients were divided into tertiles of risk, and mortality rates were compared among these categories using bar charts and Kaplan–Meier survival curves. Discriminative performance of the risk score was assessed with receiver operating characteristic (ROC) analysis and comparison against individual predictors.

Analyses were conducted using Python version 3.11 (Python Software Foundation, Wilmington, DE, USA). Statistical significance was defined as a two-tailed $p<0.05$.

Results

A total of 35 patients were analyzed, with 21 alive and 14 deceased at the end of follow-up. Median follow-up time for the entire cohort was 550 days (IQR: 169.5–1469.5).

Patients who died during follow-up were significantly older than survivors (68.2±8.2 vs. 55.9±9.4 years, $p<0.001$). Sex distribution was similar between groups (male: 85.7% vs. 76.2%, p=0.796). A greater proportion of deceased patients had a history of coronary artery bypass grafting (CABG) (35.7% vs. 4.7%,

Table 2. Multivariate Cox regression analysis for the prediction of long-term mortalitys

Variable	ß-coefficient	Hazards ratio	95% Confidence interval	p
Previous CABG	1.087	2.96	0.71–12.29	0.133
Age (per 5 years)	0.392	1.48	1.03–2.12	**0.032**
Albumin (per 1 g/dL)	-0.258	0.77	0.29–2.04	0.602
Creatinine (per 1 mg/dL)	0.474	1.60	0.85-3.02	0.141

CABG: Coronary artery bypass graft surgery

Table 3. Risk score values for risk groups and mortality rates

Group	Range (minimum-maximum)	Mean±Standard deviation	Mortality rate
Low-risk (n=12)	2.28–3.90	3.37±0.50	0
Intermediate-risk (n=11)	3.98–5.23	4.58±0.43	5 (45%)
High-risk (n=12)	5.29–7.69	6.23±0.73	9 (75%)

p=0.05). Other baseline clinical characteristics, comorbidities, medication use, and LVAD type were comparable (Appendix 1).

The deceased group showed worse baseline renal function, as reflected by higher creatinine values (1.88±0.93 vs. 1.40±0.79 mg/dL, p=0.030), along with lower serum albumin (3.43±0.62 vs. 4.04±0.50 g/dL, p=0.007), total cholesterol (126.0±40.2 vs. 169.3±39.9 mg/dL, p=0.005), and thyroid-stimulating hormone (TSH) levels (1.64±1.69 vs. 4.10±3.24 µIU/mL, p=0.007). Inflammatory ratios also differed, with a higher neutrophil-to-lymphocyte ratio (NLR) (11.00±7.43 vs. 7.47±9.48, p=0.021) and lower lymphocyte-to-monocyte ratio (LMR) (1.16±0.71 vs. 2.21±1.42, p=0.018) in the deceased group. Other laboratory markers were not significantly different between groups (Appendix 1).

Interestingly, patients who died exhibited higher baseline LVEF values compared with survivors (20.4±4.1% vs. 18.3±2.5%, p=0.039). Other echocardiographic indices (LV size, TAPSE/PASP, RV dimensions, RV-St) and invasive hemodynamic measurements (including PCWP, PAPi, RAP/PCWP, cardiac output and index, PVR, and RVSWI) were similar across groups (Appendix 2).

In univariate Cox regression analysis, advanced age, prior CABG, elevated creatinine, and reduced albumin were significantly associated with long-term mortality (Table 1). These variables were entered into the multivariate model. Of these, only age remained an independent predictor of long-term mortality (HR: 1.48 per 5 years; 95% CI: 1.03–2.12; p=0.032) (Table 2 and Fig. 1).

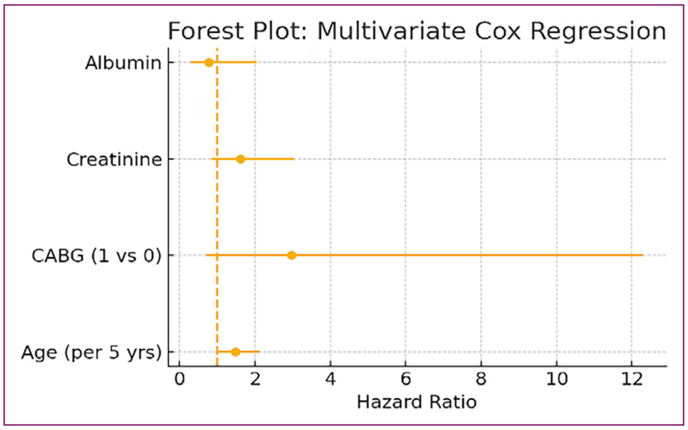

Figure 1. Forest plot of multivariate Cox regression analysis.

CABG: Coronary artery bypass grafting.

The CACA score was constructed from β-coefficients of the multivariate regression:

CACA = (0.392×Age/5) + (1.087×CABG [yes=1]) + (0.474×Creatinine) – (0.258×Albumin). Patients were classified into three categories based on score percentiles: low-risk (0–33rd), intermediate-risk (34–66th), and high-risk (67–100th). Mean scores were 3.37±0.50, 4.58±0.43, and 6.23±0.73, respectively (Table 3). Corresponding mortality rates were 0% in the low-risk, 45.4% in the intermediate-risk, and 75% in the high-risk group (Fig. 2).

In ROC analysis, the CACA score achieved the best discriminatory capacity for mortality (AUC: 0.88), outperforming each individual predictor included in the model (Fig. 3). Kaplan–Meier analysis demonstrated poorer survival in the high-risk group compared with the intermediate group, with borderline significance (log-rank p=0.055; Fig. 4).

Discussion

Different etiologies appear to contribute to mortality at different time points in LVAD recipients. Factors, such as INTERMACS status, multiple organ failure, sepsis, and right heart

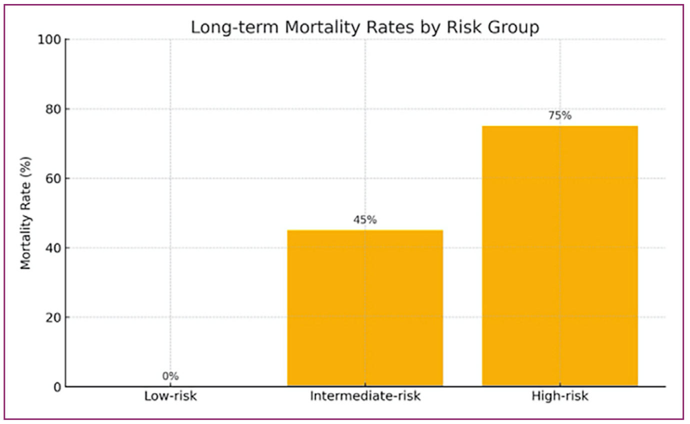

Figure 2. Long-term mortality rates according to risk groups.

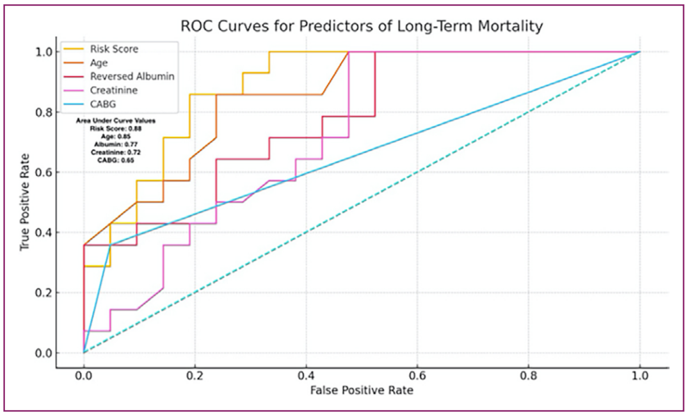

Figure 3. Receiver operating characteristic curves of risk score and its individual components for the prediction of long-term mortality.

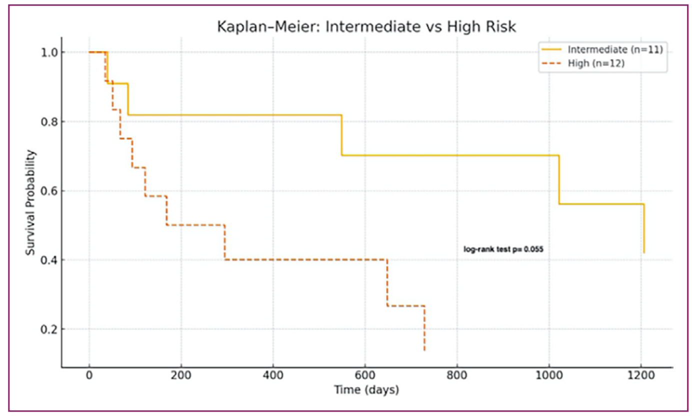

Figure 4. Kaplan–Meier survival curves of high- and intermediate risk groups.

failure remain among the most significant predictors of early mortality following LVAD implantation.[2,3] However, in the long term, the causes of mortality tend to shift, and factors, such as age, prior surgeries, comorbidities, such as renal dysfunction, laboratory markers, and right ventricular function parameters gain greater prognostic importance.[4–11]

In the HeartMate 3 risk score, predictors of long-term mortality included age, prior CABG surgery, elevated blood urea nitrogen, low serum sodium, small left ventricular size, and the RAP/PCWP ratio.[4] The J-MACS risk score identified age, history of cardiac surgery, serum creatinine, and the CVP/PCWP ratio as key components.[6] In the Penn-Columbia study, age, creatinine, total bilirubin, body mass index, right ventricular dysfunction, and aortic insufficiency were incorporated into the risk model.[7] Nayak et al.[11] reported that pre-implant blood urea nitrogen, prior CABG or valve surgery, post-implant ventricular arrhythmias, renal impairment, and hemocompatibility-related adverse events were key predictors for 5-year mortality. Inflammation surrogates, such as NLR and CRP have also been associated with long-term mortality.[9,10] In summary, although there are similarities among the components of these risk scores, no clear consensus exists. Furthermore, these risk scores may not accurately predict long-term mortality in other patient populations. For example, in the study by Moeller et al.,[13] the HeartMate 3 risk score was associated with post-implant survival, but it was not an independent predictor of long-term mortality after adjusting for ischemic etiology and severe diabetes. These discrepancies underscore the need for new, validated approaches.

Age appears to be a consistent predictor of long-term mortality following LVAD implantation.[4,6,7,14] In the study of Radhoe et al.,[14] older age was associated with higher mortality and an increased incidence of bleeding events, but not with heart failure hospitalization, right ventricular failure, ventricular arrhythmia, or device-related complications, such as pump thrombosis or device infection. In line with these findings, our cohort demonstrated that age was the only independent predictor of long-term mortality in the multivariate model.

Albumin is a marker of nutritional status, inflammation, hepatic function, and catabolism, making it a valuable global indicator of physiologic reserve. Previous reports linked low baseline albumin to worse long-term outcomes after LVAD implantation, although it has not been consistently incorporated into risk models.[15–17] Thus, the prognostic significance of pre-implantation albumin levels remains unclear.

A history of prior CABG surgery increases the risk of a complicated intraoperative and post-operative course due to prolonged cardiopulmonary bypass and cross-clamping times, increased intra-operative bleeding, and a higher likelihood of post-operative right heart failure.[18] Consequently, previous CABG surgery has been included as a risk factor in several established risk scores.[4,6,11] Consistent with these studies, a history of CABG was associated with long-term mortality in our study based on univariate Cox regression analysis.

Baseline creatinine level is also a well-established predictor of long-term mortality, and renal function markers – such as creatinine, blood urea nitrogen, and glomerular filtration rate – have been incorporated into various risk prediction models.[4,6,7,11]

Right ventriculo-arterial coupling parameters, such as the RAP/PCWP ratio and PAPi, have been shown to be associated with long-term survival after LVAD implantation in several studies.[3,4,19–22] An inadequate right ventricular response to elevated PCWP may indicate coexisting right ventricular failure in addition to left ventricular failure in patients undergoing LVAD implantation. After LVAD implantation, right ventricular failure may become more pronounced due to factors, such as increased RV preload, leftward septal shift, and loss of pericardial constraint.[23] Surrogates of right ventriculo-arterial coupling – particularly the RAP/PCWP ratio – have been included in some risk prediction models,[4,6] though not all studies have incorporated them.[7,11]

In our multivariable analysis, age emerged as the only independent predictor of long-term mortality. This finding is likely influenced by the limited sample size and event count, which may have reduced the statistical power needed to detect independent associations for other clinically relevant variables. Although prior CABG, creatinine, and serum albumin levels were all significant in univariate analysis – and have been consistently reported in the literature as prognostically important – they did not retain significance in the multivariable model, possibly due to the relatively small cohort and overlapping confidence intervals. Similarly, right ventricular–pulmonary arterial coupling parameters, such as RAP/PCWP and PAPi – known from prior studies to be strong predictors of right heart failure and adverse outcomes after LVAD implantation – did not reach statistical significance in our population, likely reflecting both cohort homogeneity and sample size limitations. In our study, several right ventricular–pulmonary arterial coupling parameters, including RAP/PCWP and PAPi, did not reach statistical significance despite their well-established prognostic importance in the LVAD literature. This absence of significance may be explained by several methodological characteristics of our cohort. First, the relatively small sample size and limited number of events inevitably reduce statistical power. Second, the values of these hemodynamic parameters were remarkably similar between survivors and non-survivors, which further attenuate the ability to detect meaningful differences. Third, early post-operative mortality related to acute right ventricular failure was intentionally excluded from the study population. As a result, the cohort represents a more homogeneous long-term LVAD population in whom baseline RV dysfunction may exert less pronounced prognostic impact compared with early post-implant phases. These factors likely contributed to the lack of statistical significance for RV parameters despite their recognized clinical relevance.

To avoid overlooking clinically meaningful predictors, we incorporated all univariate predictors into the CACA score using their β-coefficients. This strategy allowed us to integrate multiple physiologically relevant factors into a combined tool with superior discriminatory performance, as demonstrated by the high AUC value (0.88). Although age remained the only independent factor in the regression model, the composite score derived from these variables provided more robust risk stratification than any single parameter. The limited sample size may have led to an underestimation of the predictive value of certain parameters. Nonetheless, identifying strong predictors that maintain statistical significance even in small cohorts may offer valuable insights. Validation of the risk score developed in our study in larger patient populations through future research may help better define its prognostic value for long-term survival.

Study Limitations

This study has several limitations that should be acknowledged. First, it was conducted in a single-center with a relatively small sample size, which may limit the generalizability of the findings to broader populations. The small number of events, particularly in the low-risk group, may reduce the statistical power to detect associations with less prevalent variables. In addition, while the CACA risk score showed promising discriminatory performance within this cohort, it has not yet been externally validated. Therefore, prospective studies with larger, multicenter populations are necessary to confirm the predictive accuracy and clinical applicability of this risk model.

Conclusion

In this single-center experience, age, pre-operative serum albumin, renal function, and a history of CABG were identified as significant predictors of long-term mortality after LVAD implantation. From these variables, we derived the CACA score, which effectively stratified patients into low-, intermediate-, and high-risk groups with distinctly different survival outcomes. Although only age maintained independent significance in multivariate analysis, the overall score demonstrated superior discriminatory capacity compared with individual predictors (AUC 0.88). These findings emphasize the value of integrating simple clinical and laboratory parameters into a practical risk tool for long-term prognostication. While our findings highlight key prognostic factors, validation in larger, multi-center cohorts is necessary to confirm the clinical utility and generalizability of this risk stratification model.

Disclosures

Ethics Committee Approval: The study was approved by the İstanbul Medipol University Ethics Committee (no: 810, date: 11/07/2025).

Informed Consent: Informed consent was obtained.

Conflict of Interest Statement: All authors declared no conflict of interest.

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Appendix 1. Comparison of alive and deceased patients in terms of baseline clinical characteristics, medications, and laboratory parameters

Variables	Alive (n=21)		Dead (n=14)		p
	n	%	n	%	
Baseline clinical characteristics					
Age (years)	55.86±9.38		68.21±8.16		<0.001
Male sex	16	76.2	12	85.7	0.796
Body mass index (kg/m^2)	29.05±5.24		29.51±6.79		0.987
INTERMACS class					0.064
INTERMACS 1	0	0	1	7.1	
INTERMACS 2	0	0	1	7.1	
INTERMACS 3	10	47.6	1	7.1	
INTERMACS 4	6	28.5	8	57.1	
INTERMACS 5	2	9.5	3	21.4	
INTERMACS 6	2	9.5	0	0	
INTERMACS 7	1	4.7	0	0	
Presence of ICD	21	100	14	100	1.00
Hypertension	17	80.9	11	78.5	1.00
Diabetes mellitus	11	52.4	7	50	1.00
Chronic kidney disease	10	47.6	8	57.1	0.836
Cerebrovascular accident	0	0	1	7.1	0.836
Atrial fibrillation	7	33.3	5	35.7	1.00
Coronary artery disease	14	66.6	11	78.5	0.703
Previous CABG	1	4.7	5	35.7	0.055
LVAD type					0.703
HeartMate 3	16	76.2	9	64.3	
HeartMate 2	5	23.8	5	35.7	
Arrhythmic event during follow-up (VT/VF)	0	0	3	21.4	0.109
Regular medications					
Beta blocker usage	21	100	14	100	1.00
ACEi/ARB usage	7	33.3	1	7.1	0.162
SGLT-2i usage	10	47.6	6	42.8	1.00
MRA usage	13	61.9	4	28.6	0.112
Loop diuretic usage	17	80.9	12	85.7	1.00
Laboratory parameters					
Hemoglobin (g/dL)	11.81±1.97		11.01±2.38		0.252
WBC (cell count/L)	(9.11±3.00)×10^3		(9.54±3.77)×10^3		0.827
Platelets (cell count/mcL)	(208.76±71.69)×10^3		(195.93±77.88)×10^3		0.736
CRP (mg/dL)	27.93±25.69		49.42±51.29		0.195
Creatinine (mg/dL)	1.40±0.79		1.88±0.93		0.030
Urea (mg/dL)	56.90±40.54		84.06±34.96		0.005
AST (IU/L)	167.03±415.29		71.08±92.91		0.529
ALT (IU/L)	87.21±237.78		72.44±111.31		0.452
Albumin (g/dL)	4.04±0.50		3.43±0.62		0.007
Sodium (mEq/L)	136.70±3.44		134.69±3.22		0.082
Potassium (mEq/L)	4.16±0.58		4.12±0.52		0.699
Total Bilirubin (mg/dL)	0.74±0.34		1.01±1.00		0.843
LDH (units/L)	237.78±89.35		584.83±800.34		0.272

Appendix 1. Cont.

Variables	Alive (n=21)	Dead (n=14)	p
Total cholesterol (mg/dL)	169.26±39.92	126.01±40.18	0.005
LDL cholesterol (mg/dL)	101.95±29.16	82.70±36.84	0.161
TSH (mIU/L)	4.10±3.24	1.64±1.69	0.007
NT-proBNP (pg/mL)	5107 (3824–7018)	8315 (3775–21150)	0.305
NLR	7.47±9.48	11.00±7.43	0.021
LMR	2.21±1.42	1.16±0.71	0.018

INTERMACS: Interagency registry for mechanically assisted circulatory support; ICD: Implantable cardioverter defibrillator; VT: Ventricular tachycardia; VF: Ventricular fibrillation; CABG: Coronary artery bypass graft surgery; LVAD: Left ventricular assist device; ACEi: Angiotensin converting enzyme inhibitors; ARB: Angiotensin II receptor blocker; SGLT-2i: Sodium-glucose cotransporter-2 inhibitors; MRA: Mineralocorticoid receptor antagonist; WBC: White blood cell; NT-proBNP: N-terminal prohormone of brain natriuretic peptide; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; CRP: C-reactive protein; LDL: Low-density lipoprotein; LDH: Lactate dehydrogenase; TSH: Thyroid-stimulating hormone; NLR: Neutrophil-to-lymphocyte ratio; LMR: Lymphocyte-to-monocyte ratio.

Appendix 2. Comparison of alive and deceased patients in terms of echocardiographic parameters and cardiac catheterization measurements

Variables	Alive (n=21)		Dead (n=14)		p
	n	%	n	%	
Echocardiographic parameters					
LVEF (%)	18.25±2.45		20.36±4.14		0.039
Left atrium AP diameter (cm)	5.02±0.35		5.12±0.35		0.769
LV end-diastolic diameter (cm)	7.19±0.94		7.12±0.33		0.726
LV end-systolic diameter (cm)	6.42±0.65		6.17±0.61		0.335
TAPSE/PASP (mm/mmHg)	0.35±0.11		0.31±0.11		0.296
RV-St (cm/sn)	9.55±1.63		9.57±0.79		0.567
RV basal diameter (cm)	3.88±0.57		4.12±0.50		0.284
MR					0.141
Grade 0	1	4.7	1	7.1	
Grade 1	1	4.7	2	14.3	
Grade 2	3	14.2	4	28.6	
Grade 3	8	38.2	4	28.6	
Grade 4	8	38.2	3	21.4	
AR					0.526
Grade 0	12	57.1	6	42.8	
Grade 1	8	38.2	8	57.2	
Grade 2	1	4.7	0	7.6	
Grade 3	0	0	0	0	
Grade 4	0	0	0	0	
TR					0.420
Grade 0	0	0	0	0	
Grade 1	9	42.8	3	21.4	
Grade 2	8	38.2	9	64.4	
Grade 3	3	14.3	1	7.1	
Grade 4	1	4.7	1	7.1	
Left and right heart catheterization measurements					
Cardiac output (L/min)	3.77±0.69		4.20±1.25		0.536
Cardiac index (L/min/m²)	1.87±0.30		2.09±0.62		0.433
Stroke volume (mL/beat)	49.55±13.57		54.72±15.30		0.518
Stroke volume index (mL/beat/m²)	24.56±6.36		27.03±7.72		0.447
PCWP (mmHg)	25.84±7.16		24.14±9.36		0.475
PASP (mmHg)	48.79±18.80		46.14±16.74		0.729
PAPM (mmHg)	31.63±10.94		29.43±11.19		0.635
PAPD (mmHg)	21.84±7.93		20.93±8.59		0.742
PVR (Wood units)	1.89±1.85		2.31±2.10		0.573
Aortic systolic pressure (mmHg)	97.78±15.07		100.50±11.99		0.432
Aortic mean pressure (mmHg)	74.39±10.02		74.54±5.65		0.718
Aortic diastolic pressure (mmHg)	57.89±16.82		57.77±14.14		1.00
RAP (mmHg)	9.74±4.76		7.93±3.65		0.305
RVSWI (mmHg·mL/m²)	536.83±301.90		586.68±342.67		0.761
PAPi	3.07±1.41		3.55±1.43		0.172
RAP/PCWP	0.38±0.16		0.34±0.12		0.662

RAP: Right atrial mean pressure; PCWP: Pulmonary capillary wedge pressure, LVEF: Left ventricular ejection fraction; AP: Anteroposterior; TAPSE: Tricuspid annular plane systolic excursion; PASP: Pulmonary artery systolic pressure; RV-St: Peak systolic velocity of tricuspid annulus by Tissue Doppler Imaging, MR: Mitral regurgitation; AR: Aortic regurgitation; TR: Tricuspid regurgitation; PAPM: Pulmonary artery mean pressure; PAPD: Pulmonary artery diastolic pressure; PVR: Pulmonary vascular resistance; RVSWI: Right ventricular stroke work index; PAPi: Pulmonary artery pulsatility index.