

Prognostic Value of Myocardial Contraction Fraction for One-Year Cardiac Death After Transcatheter Aortic Valve Implantation in Severe Aortic Stenosis: A Simple Echocardiographic Metric for High-Risk Stratification

Miyokard Kontraksiyon Fraksiyonunun Prognostik Değeri: Yüksek Riskli Hastaların Belirlenmesinde Basit Bir Ekokardiyografik Ölçüt

ABSTRACT

Objective: Myocardial contraction fraction (MCF) is a volumetric index of myocardial shortening that relates left ventricular stroke volume to myocardial volume. In patients with severe aortic stenosis (AS), left ventricular ejection fraction (LVEF) may remain preserved despite advanced myocardial remodeling. This study aimed to evaluate the prognostic value of MCF for predicting 1-year cardiac death in patients undergoing transcatheter aortic valve implantation (TAVI).

Method: This retrospective, single-center study included 419 consecutive patients with severe AS who underwent TAVI at a tertiary center. MCF was calculated by dividing stroke volume by left ventricular myocardial volume and multiplying the result by 100. Left ventricular myocardial volume was derived by dividing left ventricular myocardial mass by the myocardial density, which was assumed to be 1.05 g/mL. The primary endpoint was 1-year cardiac death. Receiver operating characteristic (ROC) curve analysis was used to identify the optimal MCF cutoff value, and the patients were divided into low- and high-MCF groups according to this threshold.

Results: The ROC-derived MCF cutoff value for predicting 1-year cardiac death was 24%, with an area under the curve of 0.783 (95% CI, 0.716–0.851), a sensitivity of 78.1%, and a specificity of 71.8%. Patients with $MCF \leq 24\%$ had lower LVEF and stroke volume index values, higher myocardial mass index and pulmonary artery pressure values, and larger left ventricular diameters than those with $MCF > 24\%$. One-year cardiac death occurred in 32 patients (7.6%) and was significantly more frequent in the $MCF \leq 24\%$ group than in the $MCF > 24\%$ group (18.7% vs. 2.5%; $P < 0.001$). In univariable Cox regression analysis, $MCF \leq 24\%$ was strongly associated with 1-year cardiac death. In multivariable analysis, higher MCF, evaluated as a continuous variable, remained independently associated with a lower risk of 1-year cardiac death.

Conclusion: Reduced MCF was associated with adverse left ventricular remodeling and increased 1-year cardiac death after TAVI. MCF may provide additional prognostic information beyond conventional echocardiographic parameters in patients with severe AS undergoing TAVI.

Keywords: Aortic valve stenosis, echocardiography, left ventricular function, mortality, transcatheter aortic valve replacement

ÖZET

Amaç: Miyokard kontraksiyon fraksiyonu (MKF), sol ventrikül atım hacmini miyokard hacmiyle ilişkilendiren volumetrik bir miyokardiyal kısalma indeksidir. Şiddetli aort darlığı olan hastalarda, ileri düzey miyokardiyal yeniden şekillenmeye rağmen sol ventrikül ejeksiyon fraksiyonu korunmuş olabilir. Bu çalışmanın amacı, transkateter aort kapak implantasyonu (TAVI) uygulanan hastalarda MKF'nin 1 yıllık kardiyak ölümü öngörmedeki prognostik değerini değerlendirmektir.

Yöntem: Bu retrospektif, tek merkezli çalışmaya, üçüncü basamak bir merkezde TAVI uygulanan şiddetli aort darlığı olan 419 ardışık hasta dahil edildi. MKF, atım hacminin sol ventrikül miyokard hacmine bölünmesi ve elde edilen değerin 100 ile çarpılmasıyla hesaplandı. Sol ventrikül miyokard hacmi, sol ventrikül miyokard kütlelerinin 1.05 g/mL olarak kabul edilen miyokard yoğunluğuna bölünmesiyle elde edildi. Birincil sonlanım noktası 1 yıllık kardiyak ölümdü. Optimal MKF kesim değerini belirlemek için alıcı işletim karakteristiği (ROC) eğrisi analizi kullanıldı ve hastalar bu eşik değere göre düşük ve yüksek MKF gruplarına ayrıldı.

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Bulgular: Bir yıllık kardiyak ölümü öngörmek için ROC analiziyle belirlenen MKF kesim değeri %24 olup eğri altında kalan alan 0.783 (%95 GA, 0.716–0.851), duyarlılık %78.1 ve özgüllük %71.8 idi. $MKF \leq \%24$ olan hastalarda, $MKF > \%24$ olanlara kıyasla sol ventrikül ejeksiyon fraksiyonu ve atım hacmi indeksi daha düşük, miyokard kütle indeksi ve pulmoner arter basıncı daha yüksek ve sol ventrikül çapları daha büyüktü. Bir yıllık kardiyak ölüm 32 hastada (%7.6) görüldü ve $MKF \leq \%24$ grubunda $MKF > \%24$ grubuna göre anlamlı olarak daha sıkı (%18.7'ye karşı %2.5; $P < 0.001$). Tek değişkenli Cox regresyon analizinde $MKF \leq \%24$, 1 yıllık kardiyak ölümle güçlü şekilde ilişkili bulundu. Çok değişkenli analizde, sürekli değişken olarak değerlendirilen daha yüksek MKF, daha düşük 1 yıllık kardiyak ölüm riskiyle bağımsız olarak ilişkili kaldı.

Sonuç: Azalmış MKF, TAVİ sonrası olumsuz sol ventrikül yeniden şekillenmesi ve artmış 1 yıllık kardiyak ölümle ilişkili bulundu. MKF, şiddetli aort darlığı nedeniyle TAVİ uygulanan hastalarda konvansiyonel ekokardiyografik parametrelerin ötesinde ek prognostik bilgi sağlayabilir.

Anahtar Kelimeler: Aort kapak darlığı, ekokardiyografi, sol ventrikül fonksiyonu, mortalite, transkateter aort kapak implantasyonu

Transcatheter aortic valve implantation (TAVI) has become an established treatment for symptomatic severe aortic stenosis (AS) across a wide range of surgical risk categories.^{1,2} Although procedural outcomes have improved considerably with greater operator experience, device refinement, and careful patient selection, mortality after TAVI remains clinically significant, particularly among patients with advanced myocardial remodeling, low-flow physiology, renal dysfunction, frailty, and a high comorbidity burden. Therefore, accurate preprocedural risk stratification remains an essential component of TAVI planning.^{3,4}

Left ventricular ejection fraction (LVEF) remains the most widely used measure of left ventricular systolic function. However, in severe AS, LVEF may not adequately reflect the true extent of myocardial dysfunction. Chronic pressure overload induces concentric hypertrophy, increased myocardial mass, diastolic dysfunction, myocardial fibrosis, impaired coronary microcirculation, and, eventually, low-flow physiology.^{5,6} Therefore, patients may have preserved or mildly reduced LVEF despite advanced myocardial remodeling and impaired forward flow. In this setting, parameters that integrate myocardial structure and pump performance may provide additional prognostic value.

Myocardial contraction fraction (MCF) is a volumetric index of myocardial shortening calculated as the ratio of stroke volume to left ventricular myocardial volume.^{7,8} Unlike LVEF, which expresses stroke volume relative to left ventricular cavity volume, MCF expresses stroke volume relative to the amount of myocardium generating it. Therefore, MCF may be particularly useful in diseases characterized by increased myocardial mass, including hypertensive heart disease, hypertrophic cardiomyopathy, cardiac amyloidosis, and severe AS.^{9,10}

Previous studies have shown that MCF is associated with cardiovascular outcomes and may identify myocardial dysfunction even when LVEF is preserved.^{11,12} In patients undergoing TAVI, reduced MCF has been associated with increased 1-year mortality, and in patients with low-gradient severe AS and preserved LVEF, MCF has been shown to improve risk stratification beyond conventional echocardiographic parameters.^{13,14} However, data on the prognostic value of MCF in real-world TAVI populations remain limited. Therefore, the present study aimed to evaluate

ABBREVIATIONS

AS	Aortic stenosis
EuroSCORE II	European System for Cardiac Operative Risk Evaluation II
LVEF	Left ventricular ejection fraction
MCF	Myocardial contraction fraction
STS	Society of Thoracic Surgeons
TAVI	Transcatheter aortic valve implantation

the role of MCF in predicting 1-year cardiac death in patients undergoing TAVI for severe AS.

Materials and Methods

Study Design and Population

This retrospective, observational, single-center study included consecutive patients with severe AS who underwent TAVI at a tertiary referral center between 2016 and 2025. During the study period, a total of 501 patients were screened. Patients were eligible if they had symptomatic severe AS, underwent TAVI, had preprocedural echocardiographic data available for MCF calculation, and had 1-year follow-up data available for cardiac mortality assessment. Patients with missing echocardiographic variables required for MCF calculation or unavailable follow-up data were excluded from the analysis. No additional exclusion criteria were applied. After excluding patients with incomplete preprocedural echocardiographic data that precluded MCF calculation and those with unavailable 1-year follow-up data, 419 patients constituted the final study population.

Severe AS was diagnosed according to contemporary guideline recommendations using standard echocardiographic criteria, including aortic valve area, indexed aortic valve area, transvalvular gradients, peak aortic jet velocity, and flow status, when appropriate.^{1,2} The decision to perform TAVI was made by the institutional heart team based on clinical, anatomical, and procedural considerations.

The study was conducted in accordance with the principles of the Declaration of Helsinki. The study protocol was approved by Kocaeli University Non-Interventional Clinical Research Ethics Committee (Approval Number: KÜ GOKAEK-2026/06/21; Project

No. 2026/170; Date: 12.03.2026). Because of the retrospective design, the requirement for written informed consent was waived.

Clinical Data Collection

Baseline demographic, clinical, laboratory, echocardiographic, and procedural data were obtained from hospital records and the institutional electronic database. Clinical variables included age, sex, New York Heart Association (NYHA) functional class, AS type, hypertension, diabetes mellitus, peripheral artery disease, chronic obstructive pulmonary disease, prior stroke, malignancy, coronary artery disease, prior myocardial infarction, history of percutaneous coronary intervention, prior coronary artery bypass grafting, previous device implantation, and previous valvular interventions.

Laboratory parameters included serum creatinine, estimated glomerular filtration rate, hemoglobin, albumin, uric acid, C-reactive protein, N-terminal pro-B-type natriuretic peptide, and total cholesterol. Risk scores, including the European System for Cardiac Operative Risk Evaluation II (EuroSCORE II) and Society of Thoracic Surgeons (STS) score, were recorded when available.

Echocardiographic Assessment and MCF Calculation

All patients underwent comprehensive transthoracic echocardiography before TAVI. Echocardiographic parameters included LVEF, indexed aortic valve area, mean transvalvular gradient, peak aortic jet velocity, stroke volume index, left atrial diameter, left ventricular myocardial mass index, systolic pulmonary artery pressure, left ventricular end-diastolic diameter, left ventricular end-systolic diameter, and interventricular septal thickness in diastole.

Stroke volume was calculated using standard Doppler echocardiography by multiplying the left ventricular outflow tract area by the left ventricular outflow tract velocity-time integral. The left ventricular outflow tract area was derived from the left ventricular outflow tract diameter using the circular area formula. Left ventricular mass was calculated using the guideline-recommended linear echocardiographic method (Devereux formula), based on interventricular septal thickness, posterior wall thickness, and left ventricular end-diastolic diameter. Left ventricular myocardial volume was obtained by dividing left ventricular myocardial mass by the accepted myocardial density of 1.05 g/mL. MCF was calculated by dividing stroke volume by left ventricular myocardial volume and multiplying the result by 100.

MCF was calculated using the following formula:

$$\text{MCF (\%)} = \text{Stroke volume} / \text{Left ventricular myocardial volume} \times 100$$

where left ventricular myocardial volume = left ventricular myocardial mass / 1.05.

Severe AS phenotypes were classified according to baseline echocardiographic findings. High-gradient severe AS was defined as severe AS with a mean transvalvular gradient ≥ 40 mmHg and/or a peak aortic jet velocity ≥ 4.0 m/s. Non-high-gradient severe AS was defined as severe AS with an aortic valve area ≤ 1.0 cm² or an indexed aortic valve area ≤ 0.6 cm²/m² in the presence of a mean gradient < 40 mmHg and a peak aortic jet velocity < 4.0 m/s. Low-flow status was defined as a stroke volume index ≤ 35 mL/m². Patients with non-high-gradient severe AS were further classified as having classical low-flow, low-gradient AS

(LVEF $< 50\%$ and stroke volume index ≤ 35 mL/m²), paradoxical low-flow, low-gradient AS (LVEF $\geq 50\%$ and stroke volume index ≤ 35 mL/m²), or normal-flow, low-gradient AS (LVEF $\geq 50\%$ and stroke volume index > 35 mL/m²).

Receiver operating characteristic (ROC) curve analysis identified 24% as the optimal MCF cutoff value for predicting 1-year cardiac death. Patients were therefore divided into two groups: MCF $\leq 24\%$ and MCF $> 24\%$.

Endpoint Definition

The primary endpoint was 1-year cardiac death. Cardiac death was defined as death attributable to heart failure, sudden cardiac death, fatal myocardial infarction, fatal arrhythmia, valve-related death, or another documented cardiovascular cause. Mortality data were obtained from hospital records, institutional follow-up documentation, national health records, death registry information when available, and telephone follow-up. Because of the retrospective study design, events were not adjudicated by an independent clinical events committee.

Statistical Analysis

Statistical analyses were performed using SPSS software (version 25.0; IBM Corp., Armonk, NY, USA). Continuous variables were tested for normality using the Kolmogorov-Smirnov test. Normally distributed continuous variables were expressed as mean $\pm$ standard deviation, whereas non-normally distributed variables were expressed as median and interquartile range. Categorical variables were expressed as numbers and percentages.

Comparisons between the MCF $\leq 24\%$ and MCF $> 24\%$ groups were performed using the Student's t-test or Mann-Whitney U test for continuous variables, as appropriate. Categorical variables were compared using the chi-square test or Fisher's exact test. ROC curve analysis was performed to evaluate the discriminatory performance of MCF for 1-year cardiac death. The optimal cutoff value was determined using the Youden index. The area under the curve, 95% CI, sensitivity, and specificity were reported.

Univariable Cox regression analysis was performed to identify predictors of 1-year cardiac death. Variables that were statistically significant in the univariable analysis (EuroSCORE II, LVEF, indexed aortic valve area, and MCF), together with clinically relevant variables, were entered as candidate covariates into the multivariable Cox regression model. Independent predictors were then identified using backward stepwise (Wald) elimination, and only variables retained in the final model are reported. In the multivariable model, MCF was entered as a continuous variable. Multicollinearity among the covariates was assessed using variance inflation factors and tolerance statistics. Kaplan-Meier survival analysis was performed to compare cumulative survival between the MCF groups, and differences were evaluated using the log-rank test. A two-sided p value < 0.05 was considered statistically significant.

Results

Baseline Clinical Characteristics

A total of 419 patients with severe AS who underwent TAVI were included in the study. The mean age of the study population was 76.3 ± 7.5 years, and 196 patients (46.8%) were male. According to the ROC-derived cutoff value, 134 patients (32.0%) had MCF $\leq 24\%$, whereas 285 patients (68.0%) had MCF $> 24\%$.

Baseline demographic and clinical characteristics according to MCF group are shown in Table 1. Age was similar between the MCF $\leq 24\%$ and MCF $>24\%$ groups (76.2 ± 8.1 vs. 76.3 ± 7.3 years; $P = 0.781$). Male sex was more frequent in the MCF $\leq 24\%$ group than in the MCF $>24\%$ group (56.0% vs. 42.5% ; $P = 0.010$). The distribution of NYHA functional classes did not differ significantly between the groups, although patients with low MCF had a numerically higher frequency of class IV symptoms.

Regarding AS phenotype, high-gradient severe AS was present in 374 patients (89.3%). The distribution of AS phenotypes differed significantly between the MCF groups ($P < 0.001$). High-gradient AS was less frequent in the MCF $\leq 24\%$ group than in the MCF $>24\%$ group (79.9% vs. 93.7%), whereas low-gradient AS phenotypes were more frequently observed among patients with reduced MCF. Classical low-flow, low-gradient AS was present in 35 patients (8.4%) and was more common in the MCF $\leq 24\%$ group than in the MCF $>24\%$ group (16.4% vs. 4.6%). Paradoxical low-flow, low-gradient AS was present in 10 patients (2.4%) and was also numerically more frequent in the MCF $\leq 24\%$ group (3.7% vs. 1.8%). No patient met the criteria for normal-flow, low-gradient AS. These findings are consistent with the lower stroke volume index and lower transvalvular gradients observed in patients with reduced MCF.

All TAVI procedures were performed using transfemoral vascular access. Valve type distribution was also evaluated. Self-expanding valves were used in 209 patients (49.9%), and balloon-expandable valves were used in 210 patients (50.1%). Self-expanding valves were numerically more frequent in the MCF $\leq 24\%$ group, whereas balloon-expandable valves were numerically more frequent in the MCF $>24\%$ group; however, this difference did not reach statistical significance ($P = 0.055$).

Hypertension was more common in the low-MCF group (95.5% vs. 88.8% ; $P = 0.025$). Diabetes mellitus, peripheral artery disease, malignancy, coronary artery disease, prior myocardial infarction, history of percutaneous coronary intervention, prior coronary artery bypass grafting, and previous valvular intervention were similar between the groups. Prior pacemaker or implantable cardioverter-defibrillator implantation was more frequent in the MCF $\leq 24\%$ group (14.2% vs. 7.0% ; $P = 0.019$).

Among the laboratory variables, serum creatinine was higher in patients with MCF $\leq 24\%$ than in those with MCF $>24\%$ (1.23 ± 0.70 vs. 1.13 ± 0.65 mg/dL; $P = 0.010$). Estimated glomerular filtration rate, hemoglobin, albumin, uric acid, C-reactive protein, N-terminal pro-B-type natriuretic peptide, and total cholesterol did not differ significantly between the groups. EuroSCORE II was higher in the low-MCF group ($P = 0.006$), whereas the STS score did not differ significantly between the groups.

Baseline Echocardiographic Findings

Baseline echocardiographic findings are summarized in Table 2. Patients with MCF $\leq 24\%$ had a less favorable echocardiographic profile than those with MCF $>24\%$. The indexed aortic valve area was lower in the MCF $\leq 24\%$ group (0.37 ± 0.10 vs. 0.40 ± 0.10 cm²/m²; $P < 0.001$). The mean transvalvular gradient was also lower in the MCF $\leq 24\%$ group (46.2 ± 15.8 vs. 51.2 ± 15.2 mm Hg; $P < 0.001$), consistent with the higher frequency of non-high-gradient AS phenotypes.

Left ventricular systolic function and forward flow were significantly worse in patients with low MCF. LVEF was lower in the MCF $\leq 24\%$ group than in the MCF $>24\%$ group ($48.9 \pm 18.3\%$ vs. $58.4 \pm 14.3\%$; $P < 0.001$). Stroke volume index was also markedly lower in the low-MCF group (31.7 ± 6.9 vs. 40.1 ± 7.4 mL/m²; $P < 0.001$).

Patients with MCF $\leq 24\%$ had significantly more advanced myocardial remodeling. Myocardial mass index was higher in the MCF $\leq 24\%$ group (172.8 ± 33.9 vs. 136.2 ± 29.2 g/m²; $P < 0.001$). Left atrial diameter was greater (48.8 ± 5.6 vs. 46.8 ± 6.2 mm; $P < 0.001$), systolic pulmonary artery pressure was higher (40.4 ± 15.7 vs. 36.9 ± 27.2 mm Hg; $P = 0.010$), left ventricular end-diastolic diameter was greater (53.9 ± 7.2 vs. 47.5 ± 6.2 mm; $P < 0.001$), and left ventricular end-systolic diameter was greater (36.6 ± 9.3 vs. 30.9 ± 7.5 mm; $P < 0.001$). Interventricular septal thickness in diastole was also greater in the low-MCF group (13.9 ± 2.1 vs. 13.1 ± 1.4 mm; $P < 0.001$).

Clinical Outcomes

Clinical outcomes according to MCF group are shown in Table 3. Length of hospital stay was similar between patients with MCF $\leq 24\%$ and those with MCF $>24\%$ (8.5 ± 5.5 vs. 8.6 ± 4.8 days; $P = 0.672$). The rates of renal failure and postprocedural pacemaker or implantable cardioverter-defibrillator implantation were also comparable between the groups.

During the 1-year follow-up, cardiac death occurred in 32 patients (7.6%). The rate of 1-year cardiac death was significantly higher in the MCF $\leq 24\%$ group than in the MCF $>24\%$ group (25 patients [18.7%] vs. 7 patients [2.5%]; $P < 0.001$).

ROC Analysis for MCF

ROC curve analysis was performed to determine the optimal MCF cutoff value for predicting 1-year cardiac death. The optimal ROC-derived threshold was 24% . An MCF $\leq 24\%$ predicted 1-year cardiac death with an area under the curve of 0.783 (95% CI, 0.716 – 0.851), a sensitivity of 78.1% , and a specificity of 71.8% .

Regression Analysis

Univariable and multivariable Cox regression analyses of predictors of 1-year cardiac death are presented in Table 4. In the univariable analysis, EuroSCORE II was significantly associated with 1-year cardiac death (hazard ratio [HR], 1.045 ; 95% CI, 1.018 – 1.073 ; $P < 0.001$). LVEF showed an inverse association with mortality risk (HR, 0.981 ; 95% CI, 0.963 – 1.000 ; $P = 0.044$), indicating a higher risk of cardiac death with decreasing LVEF. The indexed aortic valve area was also significantly associated with the endpoint. Among the variables evaluated, reduced MCF, defined by the ROC-derived cutoff value of $\leq 24\%$, showed the strongest association with 1-year cardiac death in the univariable analysis (HR, 8.155 ; 95% CI, 3.527 – 18.858 ; $P < 0.001$) (Figure 1).

Although patients with MCF $\leq 24\%$ had significantly lower LVEF than those with MCF $>24\%$ ($48.9 \pm 18.3\%$ vs. $58.4 \pm 14.3\%$; $P < 0.001$), the prognostic performance of MCF was not solely explained by reduced LVEF. After adjustment in the multivariable model, LVEF was no longer independently associated with 1-year cardiac death (HR, 1.006 ; 95% CI, 0.984 – 1.018 ; $P = 0.602$), whereas MCF remained independently associated with the endpoint.

Table 1. Baseline demographic and clinical characteristics of patients according to MCF group

Variables	All patients (n=419)	MCF ≤24% (n=134)	MCF >24% (n=285)	P
Age, years	76.3±7.5	76.2±8.1	76.3±7.3	0.781
Male sex, n (%)	196 (46.8)	75 (56.0)	121 (42.5)	0.010
NYHA functional class, n (%)				0.080
I	3 (0.7)	1 (0.7)	2 (0.7)	
II	30 (7.2)	7 (5.2)	23 (8.1)	
III	168 (40.1)	40 (29.9)	128 (44.9)	
IV	218 (52.0)	86 (64.2)	132 (46.3)	
AS type, n (%)				<0.001
High-gradient AS	374 (89.3)	107 (79.9)	267 (93.7)	
Classical low-flow, low-gradient AS	35 (8.4)	22 (16.4)	13 (4.6)	
Paradoxical low-flow, low-gradient AS	10 (2.4)	5 (3.7)	5 (1.8)	
Valve type, n (%)				0.055
Self-expanding	209 (49.9)	76 (56.7)	133 (46.7)	
Balloon-expandable	210 (50.1)	58 (43.3)	152 (53.3)	
Comorbidities				
Hypertension, n (%)	381 (90.9)	128 (95.5)	253 (88.8)	0.025
Diabetes mellitus, n (%)	195 (46.5)	69 (51.5)	126 (44.2)	0.163
Peripheral artery disease, n (%)	75 (17.9)	25 (18.7)	50 (17.5)	0.782
COPD, n (%)	145 (34.6)	55 (41.0)	90 (31.6)	0.057
Prior stroke, n (%)	34 (8.1)	14 (10.4)	20 (7.0)	0.230
Malignancy, n (%)	36 (8.6)	8 (6.0)	28 (9.8)	0.189
Coronary artery disease, n (%)	178 (42.5)	63 (47.0)	115 (40.4)	0.198
Prior myocardial infarction, n (%)	133 (31.7)	48 (35.8)	85 (29.8)	0.219
History of PCI, n (%)	133 (31.7)	50 (37.3)	83 (29.1)	0.093
Prior CABG, n (%)	76 (18.1)	20 (14.9)	56 (19.6)	0.242
Prior ICD or pacemaker implantation, n (%)	39 (9.3)	19 (14.2)	20 (7.0)	0.019
Prior MVR, n (%)	15 (3.6)	4 (3.0)	11 (3.9)	0.783
Prior AVR, n (%)	12 (2.9)	5 (3.7)	7 (2.5)	0.533
Prior TAVI, n (%)	7 (1.7)	2 (1.5)	5 (1.8)	1.000
Laboratory findings				
Creatinine, mg/dL	1.15±0.70	1.23±0.70	1.13±0.65	0.010
eGFR, mL/min/1.73 m ²	62.2±20.1	60.2±20.3	63.2±19.9	0.138
Hemoglobin, g/dL	11.8±1.8	11.7±1.8	11.9±1.8	0.296
Albumin, g/L	39.4±4.2	38.9±4.6	39.6±3.9	0.130
Uric acid, mg/dL	6.6±2.1	6.9±1.9	6.5±2.0	0.180
CRP, mg/L	3.6 (1.43–8.8)	4.4 (1.9–14.1)	3.4 (1.3–8.1)	0.096
NT-proBNP, ng/L	2210 (930–5455)	3300 (1480–7680)	1847 (848–4490)	0.119
Total cholesterol, mg/dL	173.5±44.8	168.9±43.3	175.5±45.4	0.160
Risk scores				
EuroSCORE II	9.5±8.2	9.9±6.7	9.2±8.7	0.006
STS score	12.9±3.8	13.2±4.1	12.8±3.6	0.149

Data are presented as mean±standard deviation, median (interquartile range), or n (%). AS, Aortic stenosis; AVR, Aortic valve replacement; CABG, Coronary artery bypass grafting; COPD, Chronic obstructive pulmonary disease; CRP, C-reactive protein; eGFR, Estimated glomerular filtration rate; EuroSCORE II, European System for Cardiac Operative Risk Evaluation II; ICD, Implantable cardioverter-defibrillator; MCF, Myocardial contraction fraction; MVR, Mitral valve replacement; NT-proBNP, N-terminal pro-B-type natriuretic peptide; NYHA, New York Heart Association; PCI, Percutaneous coronary intervention; STS, Society of Thoracic Surgeons; TAVI, Transcatheter aortic valve implantation.

Table 2. Baseline echocardiographic findings according to MCF group in patients with severe AS

Variables	All patients (n=419)	MCF ≤24% (n=134)	MCF >24% (n=285)	P
Indexed AVA, cm ² /m ²	0.39±0.09	0.37±0.10	0.40±0.10	<0.001
Mean transvalvular gradient, mm Hg	49.6±15.6	46.2±15.8	51.2±15.2	<0.001
Peak aortic jet velocity, m/s	4.50±0.69	4.30±0.74	4.53±0.64	<0.001
LVEF, %	55.3±16.3	48.9±18.3	58.4±14.3	<0.001
Stroke volume index, mL/m ²	37.5±8.3	31.7±6.9	40.1±7.4	<0.001
Left atrial diameter, mm	47.5±6.1	48.8±5.6	46.8±6.2	<0.001
Left ventricular myocardial mass index, g/m ²	147.6±35.7	172.8±33.9	136.2±29.2	<0.001
Systolic PAP, mm Hg	38.1±24.2	40.4±15.7	36.9±27.2	0.010
Left ventricular end-diastolic diameter, mm	49.4±7.1	53.9±7.2	47.5±6.2	<0.001
Left ventricular end-systolic diameter, mm	32.8±8.6	36.6±9.3	30.9±7.5	<0.001
Interventricular septal thickness in diastole, mm	13.3±1.6	13.9±2.1	13.1±1.4	<0.001

Data are presented as mean±standard deviation. AS, Aortic stenosis; AVA, Aortic valve area; LVEF, Left ventricular ejection fraction; MCF, Myocardial contraction fraction; PAP, Pulmonary artery pressure.

Table 3. Clinical outcomes according to the ROC-derived MCF cutoff value

Variables	All patients (n=419)	MCF ≤24% (n=134)	MCF >24% (n=285)	P
Length of hospital stay, days	8.6±4.9	8.5±5.5	8.6±4.8	0.672
Renal failure, n (%)	55 (13.1)	18 (13.4)	37 (13.0)	0.899
Pacemaker or ICD implantation, n (%)	42 (10.0)	12 (9.0)	30 (10.5)	0.617
1-year cardiac death, n (%)	32 (7.6)	25 (18.7)	7 (2.5)	<0.001

Data are presented as mean±standard deviation or n (%). ICD, Implantable cardioverter-defibrillator; MCF, Myocardial contraction fraction; ROC, Receiver operating characteristic. Renal failure was defined as postprocedural stage 2 or 3 acute kidney injury according to Valve Academic Research Consortium/ Kidney Disease: Improving Global Outcomes criteria after TAVI.

In the final multivariable model, EuroSCORE II remained an independent predictor of 1-year cardiac death (HR, 1.041; 95% CI, 1.009–1.073; P = 0.011). The indexed aortic valve area was also independently associated with the endpoint (HR, 0.002; 95% CI, <0.001–0.164; P = 0.006). In this model, MCF was evaluated as a continuous variable, and each 1% increase in MCF was independently associated with a lower risk of 1-year cardiac death (HR, 0.882; 95% CI, 0.823–0.946; P < 0.001). These findings suggest that MCF provides prognostic information beyond conventional LVEF assessment in patients undergoing TAVI.

Kaplan–Meier Survival Analysis

Kaplan–Meier survival analysis demonstrated significantly lower 1-year survival among patients with MCF ≤24% than among those with MCF >24% (log-rank P < 0.001). The mean survival time was 10.71 months in the MCF ≤24% group and 11.76 months in the MCF >24% group (Figure 2). The survival curves showed early and persistent separation during follow-up, indicating poorer survival among patients with reduced MCF.

Mathematical Relationship Between MCF Components

Because MCF is calculated from stroke volume and left ventricular myocardial volume, differences in stroke volume index and myocardial mass index between the MCF groups are partly expected by definition. Therefore, these findings should not be interpreted as entirely independent observations but rather as confirmation that reduced MCF identifies a phenotype characterized by reduced forward flow relative to increased myocardial mass. The prognostic relevance of MCF in the present

Univariable predictors of 1-year cardiac death

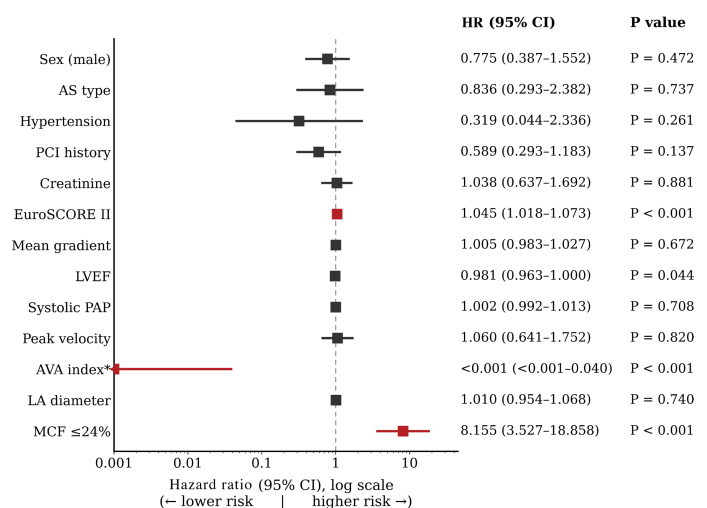


Figure 1. Forest plot of the univariable Cox regression analysis for predictors of 1-year cardiac death. Hazard ratios are plotted with 95% confidence intervals on a logarithmic scale; the dashed vertical line denotes a hazard ratio of 1. Statistically significant predictors are shown in red. Myocardial contraction fraction (MCF) ≤24% was the strongest predictor of 1-year cardiac death. *The indexed aortic valve area is expressed per 1 cm²/m²; its hazard ratio (<0.001) lies beyond the plotted range and is indicated by an arrow. AS, Aortic stenosis; AVA, Aortic valve area; LA, Left atrium; LVEF, Left ventricular ejection fraction; PAP, Pulmonary artery pressure; PCI, Percutaneous coronary intervention.

Table 4. Univariable and multivariable Cox regression analyses of predictors of 1-year cardiac death

Variables	Univariable HR (95% CI)	P	Multivariable HR (95% CI)	P
Male sex	0.775 (0.387–1.552)	0.472		
AS type	0.836 (0.293–2.382)	0.737		
Hypertension	0.319 (0.044–2.336)	0.261		
History of PCI	0.589 (0.293–1.183)	0.137		
Creatinine	1.038 (0.637–1.692)	0.881		
EuroSCORE II	1.045 (1.018–1.073)	<0.001	1.041 (1.009–1.073)	0.011
Mean transvalvular gradient	1.005 (0.983–1.027)	0.672		
LVEF	0.981 (0.963–1.000)	0.044	1.006 (0.984–1.018)	0.602
Systolic PAP	1.002 (0.992–1.013)	0.708		
Peak aortic jet velocity	1.060 (0.641–1.752)	0.820		
Indexed AVA	<0.001 (<0.001–0.040)	<0.001	0.002 (<0.001–0.164)	0.006
Left atrial diameter	1.010 (0.954–1.068)	0.740		
MCF	8.155 (3.527–18.858)	<0.001	0.882 (0.823–0.946)	<0.001

AS, Aortic stenosis; AVA, Aortic valve area; CI, Confidence interval; EuroSCORE II, European System for Cardiac Operative Risk Evaluation II; HR, Hazard ratio; LVEF, Left ventricular ejection fraction; MCF, Myocardial contraction fraction; PAP, Pulmonary artery pressure; PCI, Percutaneous coronary intervention. Multicollinearity was assessed using variance inflation factors and tolerance statistics. Independent predictors were identified using backward stepwise (Wald) Cox regression. For MCF, the univariable estimate refers to MCF $\leq 24\%$ as a binary variable, whereas the multivariable estimate refers to MCF as a continuous variable per 1% increase.

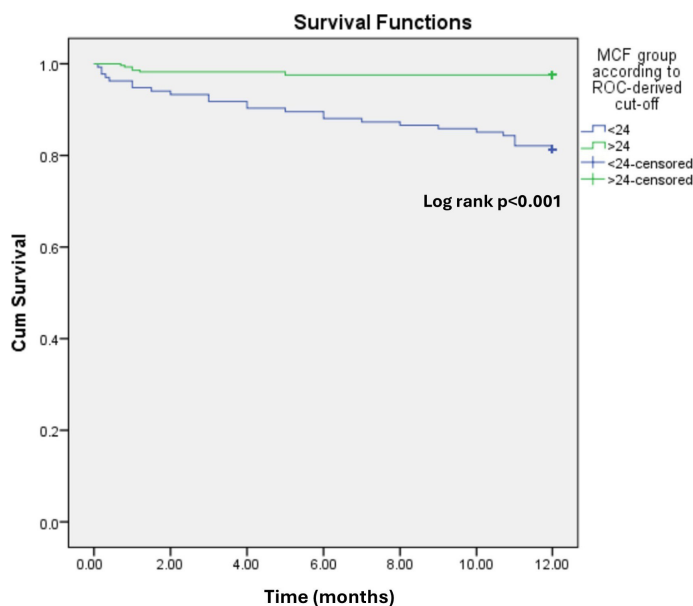


Figure 2. Kaplan-Meier survival curves for 1-year cumulative survival according to the receiver operating characteristic-derived myocardial contraction fraction (MCF) cutoff value of 24%. Patients with MCF $\leq 24\%$ had significantly lower 1-year survival than those with MCF $> 24\%$ (log-rank $P < 0.001$).

study is supported by its association with 1-year cardiac death and its independent relationship with the outcome in Cox regression analysis.

Discussion

In this retrospective, single-center study of patients undergoing TAVI for severe AS, reduced MCF was associated with adverse myocardial remodeling and a significantly increased risk of 1-year cardiac death. The principal findings were as follows: first, patients

with MCF $\leq 24\%$ had lower LVEF, lower stroke volume index, higher myocardial mass index, larger left ventricular diameters, greater left atrial diameter, and higher pulmonary artery pressure; second, an MCF threshold of 24% demonstrated good discriminatory ability for 1-year cardiac death; third, the rate of 1-year cardiac death was markedly higher in patients with MCF $\leq 24\%$ than in those with MCF $> 24\%$; and fourth, higher MCF, analyzed as a continuous variable, remained independently associated with a lower risk of 1-year cardiac death after adjustment for conventional clinical and echocardiographic variables.

These findings are pathophysiologically plausible. Severe AS causes chronic left ventricular pressure overload, leading to concentric hypertrophy, increased myocardial mass, impaired relaxation, interstitial fibrosis, and, eventually, systolic dysfunction and low-flow physiology.^{5,6} In this setting, LVEF may remain preserved despite substantial myocardial disease. MCF may partly overcome this limitation because it relates stroke volume to myocardial volume rather than to left ventricular cavity volume.^{9,10} Therefore, a lower MCF indicates that a larger myocardial mass generates a relatively smaller forward stroke volume, reflecting inefficient myocardial performance (Figure 3).

The concept of MCF was first described as a volumetric index of myocardial shortening using three-dimensional echocardiography.⁹ Subsequent studies showed that MCF may provide prognostic information in apparently healthy individuals and may identify abnormal myocardial performance in conditions associated with left ventricular hypertrophy.^{11,12} Cardiovascular magnetic resonance studies have demonstrated that MCF is reduced in hypertensive heart disease, hypertrophic cardiomyopathy, and cardiac amyloidosis despite varying degrees of LVEF preservation.¹² More recent three-dimensional echocardiographic studies have also supported the diagnostic utility of MCF in hypertrophic phenotypes with normal LVEF.¹⁰ These observations support the concept that MCF is not simply

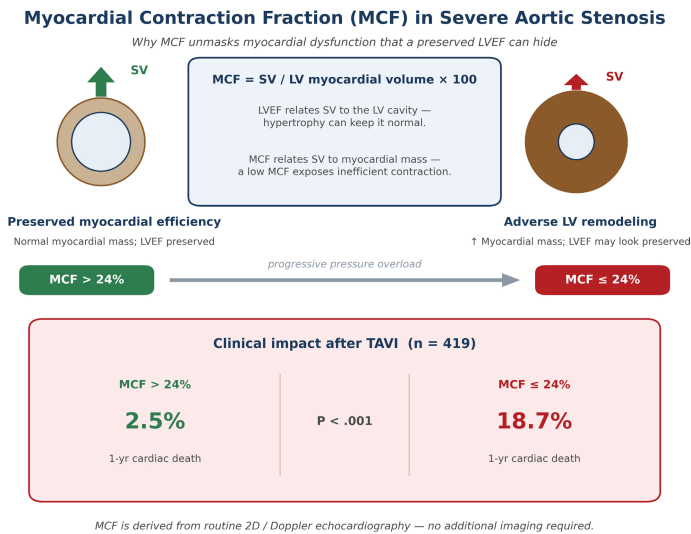


Figure 3. Mechanistic relationship between left ventricular remodeling and myocardial contraction fraction (MCF) in severe aortic stenosis. Chronic pressure overload causes concentric hypertrophy and increased myocardial mass, such that a larger myocardium generates a relatively smaller forward stroke volume (SV). Because left ventricular ejection fraction relates SV to cavity volume, it may remain preserved despite this remodeling, whereas MCF relates SV to myocardial volume and therefore reveals reduced myocardial efficiency. In this cohort, patients with $MCF \leq 24\%$ had a markedly higher rate of 1-year cardiac death than those with $MCF > 24\%$ (18.7% vs. 2.5%; $P < 0.001$). MCF is derived from routine two-dimensional and Doppler echocardiography without additional imaging.

another expression of LVEF but rather an integrated measure of myocardial shortening relative to myocardial mass. Consistent with this concept, LVEF lost its independent association with 1-year cardiac death after multivariable adjustment ($P = 0.602$), whereas higher MCF, analyzed as a continuous variable, remained independently associated with a lower risk. This dissociation suggests that MCF captures prognostic information beyond that conveyed by LVEF alone, which may be particularly relevant in severe AS, in which LVEF is frequently preserved despite advanced myocardial remodeling.

The clinical implications of these findings are important. MCF can be calculated using routinely available echocardiographic variables and does not require additional imaging, contrast administration, or advanced postprocessing. In contemporary real-world TAVI cohorts, residual mortality remains substantial despite high procedural success rates, underscoring the value of additional preprocedural risk markers.¹⁵ Moreover, although TAVI itself induces favorable changes in left ventricular function that can be monitored echocardiographically, baseline myocardial efficiency may better reflect the extent of preexisting remodeling than postprocedural changes alone.¹⁶ Therefore, MCF may be incorporated into preprocedural TAVI assessment as a simple marker of myocardial reserve and ventricular remodeling. Patients with $MCF \leq 24\%$ may represent a high-risk subgroup requiring closer postprocedural follow-up, optimization of heart failure therapy, and careful surveillance for adverse outcomes. Moreover, reduced MCF may identify patients in whom valve intervention occurs after advanced or partially irreversible myocardial damage

has already developed, emphasizing the importance of timely referral before advanced myocardial remodeling occurs. Because MCF is derived entirely from standard two-dimensional and Doppler echocardiographic measurements, the proposed 24% threshold can be applied at the bedside without additional cost or expertise, which may facilitate its adoption into routine practice. Whether automated MCF calculation within echocardiography platforms could reduce measurement variability remains a question for future study; any such application would require prospective validation before clinical adoption.

Another strength of MCF is that it integrates two key components of AS pathophysiology: forward flow and myocardial hypertrophy. Conventional parameters such as LVEF, mean gradient, valve area, and stroke volume index each describe a single dimension of disease severity. MCF combines myocardial structure and function into a single index, making it particularly attractive in patients with discordant AS parameters, low-flow states, or preserved LVEF despite symptoms. In this respect, MCF may complement rather than replace existing risk scores such as EuroSCORE II. Similarly, adjunctive markers reflecting extracardiac organ dysfunction have been shown to improve mortality prediction beyond conventional risk scores in patients with severe AS, supporting a multiparametric approach to risk stratification.¹⁷

Study Limitations

This study has several limitations. First, it was a retrospective, single-center study; therefore, selection bias and unmeasured confounding cannot be excluded. Second, although the cohort included 419 patients, the number of 1-year cardiac deaths was limited, which may have affected the stability of the multivariable model. Third, MCF was calculated using echocardiographic measurements, which are subject to interobserver variability, particularly in the estimation of stroke volume and left ventricular mass. Fourth, the ROC-derived MCF cutoff value of 24% was obtained from this cohort and requires external validation before routine clinical use.

Fifth, several echocardiographic parameters with established prognostic value after TAVI, including global longitudinal strain, the E/e' ratio, left ventricular diastolic function grade, tricuspid annular plane systolic excursion, right ventricular function, and right ventricular-pulmonary artery coupling, were not systematically available and therefore could not be included in the analysis. Advanced imaging markers, such as cardiac magnetic resonance-derived fibrosis, extracellular volume fraction, and computed tomography-derived myocardial mass, were also unavailable. Sixth, frailty, nutritional status, postdilatation, residual transvalvular gradient, paravalvular leak severity, procedural complications, and postprocedural hemodynamic improvement may influence 1-year cardiac death but were not fully incorporated into the final multivariable model. Although valve type and vascular access were recorded and included in the descriptive analysis, the present study was primarily designed to evaluate the prognostic value of a preprocedural echocardiographic index of myocardial performance. Finally, cardiac death was determined retrospectively using available clinical records and follow-up sources, and events were not independently adjudicated by a clinical events committee.

Conclusion

Reduced MCF was associated with adverse left ventricular remodeling and a significantly increased risk of 1-year cardiac death in patients undergoing TAVI for severe AS. An MCF cutoff value of 24% identified a high-risk subgroup with markedly poorer survival. MCF remained independently associated with 1-year cardiac death after adjustment for conventional clinical and echocardiographic variables. As a simple echocardiographic index that integrates stroke volume and myocardial mass, MCF may improve preprocedural risk stratification in patients undergoing TAVI. Prospective multicenter studies are needed to externally validate the 24% threshold and determine whether incorporating MCF into preprocedural risk assessment improves clinical decision-making and outcomes.

Ethics Committee Approval: Ethics committee approval was obtained from Kocaeli University Non-Interventional Clinical Research Ethics Committee Approval Number: KÜ GOKAEK-2026/06/21; Project No. 2026/170; Date: 12.03.2026).

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