Cite as: Cui SY, Hu Y, Xu X, Li Y, Zhao H, Zhu XW. Association of muscle-to-fat ratio with renal impairment in type 2 diabetic nephropathy patient [J]. Chin J Clin Res, 2026, 39(1):28-32, 37.

DOI: 10.13429/j.cnki.cjcr.2026.01.005

Association of muscle-to-fat ratio with renal impairment in type 2 diabetic nephropathy patients

CUI Siyuan, HU Yun, XU Xiang, LI Yang, ZHAO Han, ZHU Xiaowei

Department of Endocrinology, Wuxi People's Hospital Affiliated to Nanjing Medical University, Wuxi, Jiangsu 214000, China

Corresponding author: ZHU Xiaowei, E-mail: zhuxw@njmu.edu.cn

Abstract: **Objective** To investigate the relationship between the muscle-to-fat ratio (MFR) and renal function impairment in patients with type 2 diabetic kidney disease (DKD), and to provide an objective basis for assessing the risk of DKD progression. **Methods** A cross-sectional study was conducted, involving 277 hospitalized DKD patients at Wuxi People"s Hospital Affiliated to Nanjing Medical University from May 2023 to May 2025. Body composition indicators, including body mass index (BMI), MFR, and skeletal muscle mass index (SMMI), were measured using bioelectrical impedance analysis (BIA). Simultaneously, serum creatinine (Scr), blood urea nitrogen (BUN), and urinary microalbumin-to-creatinine ratio (UACR) were detected, and the estimated glomerular filtration rate (eGFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula. According to the CGA (cause-eGFR-albuminuria) staging system recommended by the Kidney Disease: Improving Global Outcomes (KDIGO) 2022 guidelines, patients were stratified into risk groups based on combined eGFR and UACR levels: Group G1-2A2 [eGFR≥60 mL/ (min·1.73 m 2) and UACR 30~299 mg/g], Group G1-2A3[eGFR≥60 mL/ (min·1.73 m^2) and UACR≥ 300 mg/g], Group G3A2 [eGFR 30~59 mL/ (min·1.73 m^2) and UACR 30~299 mg/g], Group G3A3[eGFR 30~59 mL/ (min·1.73 m^2) and UACR≥300 mg/g], and Group G4-5A2-3 [eGFR < 30 mL/ (min·1.73 m^2) and UACR≥30 mg/g]. Spearman correlation analysis was used to examine the relationship between body composition indicators and renal function parameters. Binary logistic regression analysis was applied to identify independent influencing factors for renal impairment[eGFR<60 mL/ (min·1.73 m^2)], and multivariate logistic regression was used to evaluate the predictive value of MFR for DKD progression risk based on CGA staging. Receiver operating characteristic (ROC) curves were plotted to assess the predictive efficacy of MFR for renal function decline. **Results** As renal function worsened (from the G1-2A2 group to the G4-5A2-3 group), MFR showed a decline trend (P<0.01). MFR was positively correlated with eGFR (r=0.547, P<0.01) and negatively correlated with Scr, BUN, and UACR (r=-0.341, -0.328, -0.136, P<0.05). Multivariate logistic regression analysis indicated that standardized MFR (ZMFR) was an independent protective factor for renal impairment (OR=0.166, 95%CI: 0.098-0.280, P<0.01) after adjusting for confounding factors. Additionally, ZMFR was identified as an independent protective factor against the progression to high-risk DKD stages (OR=0.621, 95%CI: 0.426-0.904, P=0.013). ROC analysis revealed that the area under the curve (AUC) for MFR in predicting renal function decline[eGFR<60 mL/ (min·1.73 m^2)]was 0.813 (P<0.01). **Conclusion** A decrease in MFR is independently associated with renal function impairment in DKD patients. An increase in MFR is an independent protective factor for renal impairment and the progression of CGA staging. Monitoring changes in MFR may help identify high-risk patients early and provide new insights into muscle-fat metabolism interventions for DKD.

Keywords: Muscle-to-fat ratio; Diabetic kidney disease; Renal function impairment; Cross-sectional study; Bioelectrical impedance analysis

Fund program: Youth Project of the National Natural Science Foundation of China (82000685)

Diabetic kidney disease (DKD) is the leading cause of end-stage renal disease (ESRD) worldwide. Approximately 40% of patients with type 2 diabetes mellitus (T2DM) are affected by DKD, and their risk of cardiovascular mortality is significantly elevated [1]. The early pathological changes of DKD are insidious. At present, the diagnosis of DKD relies on the urinary microalbumin-to-creatinine ratio (UACR) and estimated glomerular filtration rate (eGFR). However, both indicators exhibit obvious abnormalities only when renal injury is relatively severe [2], resulting in insufficient sensitivity for the detection of early renal function impairment. Therefore, the development of more effective risk warning tools and intervention targets has become an urgent need for the current clinical management and treatment of DKD.

Recent studies have revealed that the imbalance between muscle and adipose metabolism is a key pathological mechanism driving the progression of DKD. Sarcopenia can activate inflammatory factor-mediated protein catabolism, exacerbating insulin resistance, renal microinflammation, and oxidative stress. In contrast, visceral fat accumulation promotes lipid deposition in nephrons through lipotoxic reactions, increasing the lipid load of nephrons and forming a vicious cycle of the

"muscle-kidney axis" [3-4]. Nevertheless, traditional body composition indicators such as the skeletal muscle mass index (SMMI) and visceral fat area only evaluate a single tissue. They fail to quantify the interaction between muscle and adipose tissue, are easily interfered with by edema and body weight fluctuations, and thus cannot accurately assess the comprehensive metabolic status of the body [5-6].

The muscle-to-fat mass ratio (MFR) is defined as the ratio of total body skeletal muscle mass to fat mass. It can simultaneously quantify muscle reserve and fat load, and assess bidirectional metabolic processes. In addition, MFR can identify and warn of "normal-weight obesity" and quantify the synergistic effects of muscle gain and fat loss [7]. Previous studies have reported the association between MFR and cardiovascular diseases in diabetic patients, suggesting that a low MFR is significantly correlated with coronary artery calcification and cardiac function decline in T2DM patients [8]. However, its specific predictive value for renal function impairment and disease progression in DKD remains unclear.

Accordingly, this cross-sectional study was designed to investigate the association between MFR and renal function impairment, as well as the comprehensive risk of disease progression in DKD patients, in combination with the Cause-eGFR-Albuminuria (CGA) staging system recommended by the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines. The study aims to provide a new basis for DKD risk stratification and offer evidence for metabolic interventions targeting the muscle-adipose axis in the treatment of T2DM complications.

1 Participants and Methods

1.1 Study Population

This study adopted a cross-sectional design. A total of 277 DKD patients hospitalized in the Department of Endocrinology, Wuxi People's Hospital Affiliated to Nanjing Medical University, were enrolled between May 2023 and May 2025.

Inclusion criteria: (1) Meeting the World Health Organization (WHO) diagnostic criteria for diabetes in 1999 [fasting blood glucose (FBG) ≥ 7.0 mmol/L or 2-hour plasma glucose during an oral glucose tolerance test ≥ 11.1 mmol/L] [9]; (2) Meeting the diagnostic criteria for DKD in the KDIGO guidelines [diabetes duration ≥ 5 years, and UACR ≥ 30 mg/g and/or eGFR < 60 mL/(min·1.73 m²)] [10].

Exclusion criteria: (1) Presence of factors interfering with body composition measurement (e.g., edema, pacemaker implantation, severe visual, auditory or cognitive impairment); (2) Presence of severe systemic diseases (hemoglobin ≤ 90 g/L, alanine transaminase > 120 U/L, active tumors, severe infections, etc.); (3) Presence of neuropsychiatric disorders (Parkinson's disease, epilepsy, dementia, schizophrenia, etc.).

Among the 400 initially screened T2DM patients, 123 were excluded for the following reasons: thyroid dysfunction (n = 12), anemia (n = 6), hepatic insufficiency (n =7), tumors (n=8), failure to complete body composition testing (n=52), neuropsychiatric disorders (n=2), dementia (n =4), and missing evaluation data (n =32). Finally, 277 patients were included in the analysis. This study was approved by the Ethics Committee of Wuxi People's Hospital [No. (2023) 67].

1.2 Methods

1.2.1 Collection of Baseline Data

Baseline data of the patients were uniformly collected from the hospital's electronic medical record system. (1) Demographic characteristics, including gender, age, height, body weight, waist circumference, hip circumference, and diabetes duration; (2) Biochemical indicators, including glycosylated hemoglobin (HbA1c), FBG, triglyceride (TG), and low-density lipoprotein cholesterol (LDL-C); (3) Calculated indices: body mass index (BMI) and waist-to-hip ratio (WHR). All data were independently entered by two researchers. Discrepant data were verified and corrected by a third researcher against the original records.

1.2.2 Measurement of Renal Function Indicators and Grouping

Renal function was evaluated using standardized testing procedures. Serum creatinine (Scr), urinary creatinine, and blood urea nitrogen (BUN) were measured via enzymatic methods using the Roche Cobas 8000 analyzer. Urinary microalbumin was measured via immunoturbidimetry using the Siemens ADVIA 2400 analyzer. The UACR was calculated, and eGFR was computed using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation [11]. DKD patients were stratified for risk using the CGA (Cause-eGFR-Albuminuria) staging system recommended by the 2022 KDIGO guidelines [10]. Considering that all enrolled patients were diagnosed with DKD and thus classified into the moderate-to-high risk strata of the CGA staging system, the patients were divided into 5 clinical subgroups representing different stages of disease progression based on the combination of eGFR stages (G stages) and albuminuria stages (A stages). (1) G1-2A2 group (moderate risk): Normal renal function [eGFR≥60 mL/(min·1.73 m²)] combined with microalbuminuria (UACR 30–299 mg/g). (2) G1-2A3 group (high risk): Normal renal function [eGFR ≥ 60 mL/(min·1.73 m²)] combined with macroalbuminuria (UACR ≥ 300 mg/g). (3) G3A2 group (high risk): Reduced renal function [eGFR 30–59 mL/(min·1.73 m²)] combined with microalbuminuria (UACR 30–299 mg/g). (4) G3A3 group (very high risk): Reduced renal function [eGFR 30–59 mL/(min·1.73 m²)] combined with macroalbuminuria (UACR ≥ 300 mg/g). (5) G4-5A2-3 group (very high risk/renal failure): Severe renal impairment or renal failure [eGFR < 30 mL/(min·1.73 m²)] combined with albuminuria (UACR ≥ 30 mg/g).

1.2.3 Body Composition Measurement

Body composition was measured using a bioelectrical impedance analysis (BIA) device (InBody 720,

BIOSPACE) under standardized protocols. Before measurement, patients were required to fast for 8 hours, empty their bladders, wear light clothing, and remove all metal objects. During measurement, patients stood barefoot on the electrode plates, with both feet in close contact with the foot electrodes, held the electrode handles with both hands, and remained stationary for 5 minutes. The device directly output body weight, fat mass (FM), and skeletal muscle mass (SMM). Derived indices were calculated as follows: SMMI = SMM (kg) / height² (m²); MFR = SMM / FM.

1.3 Statistical Analysis

Statistical analyses were performed using SPSS 26.0 software. Measurement data conforming to a normal distribution were expressed as $\bar{x}\pm s$, while non-normally distributed data were presented as median (interquartile range, M (Q_1, Q_3). Enumeration data were reported as cases (percentage). One-way analysis of variance (ANOVA) was used for comparisons among multiple groups if the data were normally distributed and with homogeneity of variance; otherwise, the Kruskal-Wallis H test was applied. The chi-square test was used for comparisons of categorical variables. Spearman's rank correlation was employed to analyze the correlations between body composition indicators and renal function parameters. To eliminate the influence of dimensionality and compare effect sizes, the MFR variable was standardized using the Z-score method. Binary logistic regression analysis was conducted to assess the independent effect of standardized MFR (ZMFR) on renal function impairment. Multivariate logistic regression models were applied to evaluate the predictive value of ZMFR for the comprehensive progression risk of DKD. Receiver operating characteristic (ROC) curve analysis was performed to determine the predictive efficacy of MFR for renal function decline [eGFR < 60 mL/(min • 1.73 m²)], with the area under the curve (AUC) and optimal cutoff value calculated. All statistical tests were two-sided, and a P value < 0.05 was considered statistically significant.

2 Results

2.1 Comparison of Baseline Characteristics

Among the 277 DKD patients, 144 were male and 133 were female. The number of patients in each subgroup was as follows: 53 (19.13%) in the G1-2A2 group, 57 (20.58%) in the G1-2A3 group, 49 (17.69%) in the G3A2 group, 63 (22.74%) in the G3A3 group, and 55 (19.86%) in the G4-5A2-3 group. The comparisons of baseline characteristics among the groups are presented in **Table 1**.

The median diabetes duration was the longest in the G4-5A2-3 group [16.0 (13.0, 21.0) years]. With the progression of renal dysfunction, Scr and BUN levels gradually increased, while eGFR gradually decreased. Scr and BUN levels were the highest, and eGFR was the lowest in the G4-5A2-3 group, with statistically significant differences among the groups (P < 0.01).

In terms of body composition, MFR showed a decreasing trend with the decline of renal function (P < 0.01), dropping from 0.77 (0.64, 0.98) in the G1-2A2 group to 0.61 (0.48, 0.66) in the G4-5A2-3 group. BMI, WHR, and FM exhibited an overall increasing trend (P < 0.01).

Regarding metabolic indicators, statistically significant differences were observed in FBG levels among the five groups (P =0.047). Specifically, FBG levels were significantly lower in the G3A2 and G4-5A2-3 groups than in the G1-2A2 group, while they were significantly higher in the G3A3 group than in the G1-2A2 and G1-2A3 groups (P < 0.01). No statistically significant differences were found in HbA1c and TG levels among the groups (P > 0.05).

Tab.1 Comparison of baseline characteristics among different patient groups ［M（Q_1, Q_3）］

Indicators	G1-2A2 (n=53)	G1-2A3 (n=57)	G3A2 (n=49)	G3A3 (n=63)	G4-5A2-3 (n=55)	χ^2/H/F value	P value
Male [case (%)]	30(56.60)	32(56.14)	22(44.90)	31(49.21)	29(52.73)	2.040	0.728
Age (years)	53.0 (45.5 57.0)	55.0 (46.5 63.5)	52.0 (44.5 58.5)	44.0 (38.0 58.0) [abc]	51.0 (45.0 58.0)	11.751	0.019
Diabetes duration (years)	9.0 (6.5 12.0)	11.0 (7.5 14.5)	11.0 (7.0 14.5)	12.0 (7.0 15.0)	16.0 (13.0 21.0) [abc]	48.867	<0.001
HbA1c (%, $\bar{x}\pm s$)	9.05 ± 2.16	8.89 ± 2.09	8.48 ± 2.01	9.55 ± 1.97	9.08 ± 2.12	1.951	0.102
FBG (mmol/L, $\bar{x}\pm s$)	8.76 ± 2.10	8.51 ± 2.62	8.02 ± 2.10 [a]	9.27 ± 2.46 [ab]	8.27 ± 2.25 [a]	2.449	0.047
TG (mmol/L)	2.39 (1.26 4.57)	3.41 (1.62 5.23)	2.19 (1.16 4.97)	2.24 (1.65 4.17)	3.08 (2.58 3.86)	5.645	0.227
LDL-C (mmol/L)	2.69 (2.28 3.24)	3.10 (2.58 3.55)	2.86 (2.37 3.42)	3.15 (2.60 3.68) [ab]	3.27 (2.73 3.75) [ab]	15.984	0.003
Height (cm)	170 (163.0 174.0)	169 (164.0 175.0)	166 (157.5 175.5) [a]	163 (155.0 171.0) [ab]	169 (158.0 173.0)	15.589	0.004
Weight (kg)	66 (56 73)	58 (52 64) [a]	62 (53 74)	66 (59 71) [b]	65 (60 81) [b]	39.108	<0.001
BMI (kg/m²)	21.89 (19.44 26.39)	20.69 (17.94 22.30) [a]	22.89 (19.60 26.27)	24.28 (21.85 27.73) [ab]	23.81 (22.21 28.63) [b]	39.108	<0.001
Waist circumference (cm)	77.0 (72.0 86.5)	87.0 (80.5 93.0) [a]	87.0 (80.0 92.5) [a]	86.0 (77.0 95.0) [ab]	86.0 (75.0 92.0) [ab]	18.425	0.001
Hip circumference (cm)	89.0 (86.0 95.0)	94.0 (90.0 98.0) [a]	91.0 (87.5 95.5)	92.0 (88.0 97.0) [ab]	90.0 (85.0 95.0) [ab]	12.895	0.012
WHR	0.87 (0.82 0.91)	0.90 (0.86 0.99) [a]	0.92 (0.89 1.01) [a]	0.91 (0.85 0.99) [ab]	0.95 (0.87 1.00) [abcd]	20.297	<0.001
SMM（kg）	26.0 (24.4 27.6)	23.2 (21.0 27.2) [a]	21.3 (19.8 23.1) [ab]	21.9 (20.1 25.1) [ab]	22.0 (20.9 23.5) [ab]	52.098	<0.001
SMMI（kg/m²）	8.8 (8.4 9.7)	8.2 (7.4 9.1)	7.7 (7.1 8.7) [a]	8.3 (7.5 9.0)	7.9 (7.1 8.6) [a]	29.447	<0.001

Indicators	G1-2A2 (*n*=53)	G1-2A3 (*n*=57)	G3A2 (*n*=49)	G3A3 (*n*=63)	G4-5A2-3 (*n*=55)	χ^2/H/F value	*P* value
FM（kg）	33.6 (24.3 40.3)	27.0 (23.0 34.2) [a]	33.6 (27.0 41.3) [b]	35.9 (31.1 39.7) [ab]	36.9 (32.4 47.6) [ab]	37.141	<0.001
MFR（kg/kg）	0.77 (0.64 0.98)	0.88 (0.69 1.01) [a]	0.71 (0.50 0.79) [ab]	0.66 (0.54 0.71) [abc]	0.61 (0.48 0.66) [abcd]	64.019	<0.001
Scr（μmol/L）	83.4 (75.5 95.9)	90.0 (80.5 102.6)	109.2 (89.1 126.9) [a]	139.1 (124.6 153.2) [ab]	197.1 (143.1 269.8) [abcd]	166.65	<0.001
BUN（mmol/L）	5.61 (4.57 6.95)	5.85 (4.86 7.12)	7.79 (5.98 9.27) [a]	8.48 (6.86 9.35) [ab]	9.39 (7.30 10.91) [abcd]	97.606	<0.001
UACR（mg/g）	76.5 (57.3 122.9)	508.2 (406.8 681.4) [a]	163.2 (98.6 198.6) [ab]	593.0 (500.6 635.1) [abc]	386.3 (282.3 564.4) [abcd]	200.815	<0.001
eGFR [mL/(min·1.73m²)]	85.0 (78.0 92.5)	84.0 (77.0 89.5)	50.0 (45.0 55.0) [ab]	43.0 (39.0 48.0) [abc]	17.0 (14.0 21.0) [abcd]	209.056	<0.001

Note: Compared with G1-2A2 group: [a]*P*<0.05; Compared with G1-2A3 group, [b]*P*<0.05; Compared with G3A2 group, [c]*P*<0.05; Compared with G3A3 group, [d]*P*<0.05

2.2 Correlation Analysis Between Body Composition Indicators and Renal Function Parameters

Spearman's correlation analysis demonstrated that MFR was positively correlated with eGFR ($P < 0.01$) and negatively correlated with Scr, BUN, and UACR ($P < 0.05$). SMMI was positively correlated with eGFR ($P = 0.020$) and negatively correlated with BUN ($P = 0.020$), but showed no correlation with Scr and UACR ($P > 0.05$). WHR was negatively correlated with eGFR ($P < 0.01$) and positively correlated with Scr, BUN, and UACR ($P < 0.05$). BMI and FM were negatively correlated with eGFR ($P < 0.01$) and positively correlated with Scr and BUN ($P < 0.01$), but had no significant correlation with UACR ($P = 0.267$). The detailed results are shown in **Table 2**.

2.3 Multivariate Logistic Regression Analysis of Factors Associated With Renal Function Impairment

Binary logistic regression analysis was performed with the occurrence of renal function decline [eGFR < 60 mL/(min·1.73 m²)] as the dependent variable (0 = no, 1 = yes). ZMFR, age, diabetes duration, standardized WHR (ZWHR), HbA1c, TG, and LDL-C were included as independent variables.

The results revealed that after adjusting for confounding factors including age, diabetes duration, and TG, elevated ZMFR was an independent protective factor against renal function impairment (*P*<0.01). Each 1-standard deviation increase in ZMFR was associated with an 83.4% reduction in the risk of renal function impairment in patients. The detailed results are presented in **Table 3**.

2.4 Multivariate Logistic Regression Analysis of Factors Associated With DKD Progression

According to the CGA staging system of the 2022 KDIGO guidelines, patients were divided into a moderate-risk group (G1-2A2 group) and a high-progression-risk group (G1-2A3 group and above). Multivariate logistic regression models were constructed with the comprehensive progression risk of DKD as the dependent variable (0 =moderate risk, 1 = high progression risk). Indicators directly related to staging definitions, such as Scr and UACR, were excluded to avoid multicollinearity. ZMFR, age, diabetes duration, HbA1c, TG, LDL-C, and ZWHR were incorporated into the models.

The results indicated that elevated ZMFR was independently associated with a reduced risk of high-progression DKD (*P*=0.013). Each 1-standard deviation increase in ZMFR was associated with a 37.9% reduction in the risk of high-risk progression in DKD patients. In addition, diabetes duration, LDL-C, and ZWHR were independent influencing factors for DKD progression ($P < 0.05$). The detailed results are shown in **Table 4**.

2.5 Predictive Efficacy of MFR for Renal Function Decline in DKD Patients

ROC curve analysis was conducted to evaluate the predictive value of MFR for renal function decline [eGFR < 60 mL/(min·1.73 m²)] in DKD patients. As shown in **Figure 1**, the AUC of MFR for predicting renal function decline was 0.813 (95% *CI*: 0.760–0.866, $P < 0.01$), indicating that MFR had favorable predictive efficacy. The optimal cutoff value determined by the Youden index was 0.534, with a corresponding sensitivity of 0.643 and specificity of 0.891.

Tab.2 Correlation analysis between body composition indicators and renal function indicators

Indicators		MFR	SMMI	WHR	BMI	FM
eGFR	*r* value	0.547	0.139	-0.323	-0.426	-0.396
	P value	<0.001	0.020	<0.001	<0.001	<0.001
UACR	*r* value	-0.136	-0.082	0.153	0.064	0.067
	P value	0.023	0.174	0.011	0.289	0.267
SCr	*r* value	-0.341	-0.022	0.215	0.312	0.284
	P value	<0.001	0.711	<0.001	<0.001	<0.001
BUN	*r* value	-0.328	-0.140	0.150	0.214	0.238
	P value	<0.001	0.020	0.012	<0.001	<0.001

Tab.3 Multivariate logistic regression analysis of renal impairment in DKD patients

Factors	*β*	*SE*	*Wald* χ^2	*P value*	*OR (95% CI)*
Age	-0.043	0.015	7.002	0.008	0.958 (0.929–0.987)
Diabetes duration	0.098	0.030	9.586	0.002	1.103 (1.037–1.174)
HbA1c	0.050	0.082	0.392	0.531	1.051 (0.899–1.230)
TG	0.181	0.075	5.859	0.016	0.834 (0.720–0.966)
LDL-C	-0.169	0.196	0.743	0.389	1.184 (0.807–1.738)
ZWHR	0.310	0.218	2.029	0.154	1.364 (0.890–2.090)
ZMFR	-1.798	0.269	44.823	<0.001	0.166 (0.098–0.280)
(Constant)	-1.534	2.295	0.447	0.504	-

Tab.4 Multivariate logistic regression analysis of high-risk DKD progression based on CGA staging

Factors	*β*	*SE*	*Wald* χ^2	*P value*	*OR (95% CI)*
Age	0.002	0.016	0.016	0.892	1.002 (0.971–1.034)
Diabetes duration	0.085	0.036	5.550	0.018	1.089 (1.014–1.168)
HbA1c	-0.034	0.084	0.167	0.670	0.966 (0.825–1.131)
TG	-0.116	0.075	2.481	0.116	0.890 (0.770–1.029)
LDL-C	0.082	0.257	9.708	0.002	2.229 (1.346–3.691)
ZWHR	0.098	0.251	0.156	0.692	1.099 (1.088–1.768)
ZMFR	-0.476	0.312	6.177	0.013	0.621 (0.426–0.904)
(Constant)	-0.911	1.313	0.482	0.488	-

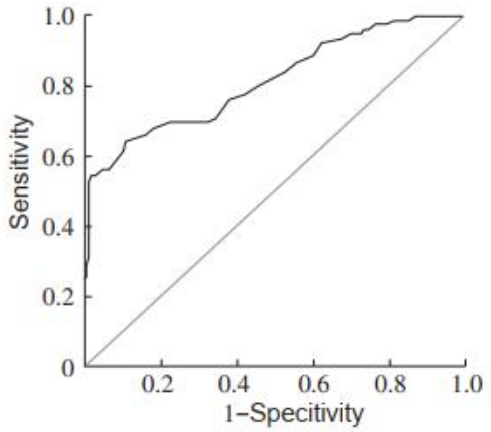

Fig.1 ROC curve of MFR for predicting renal function decline in patients with DKD

3 Discussion

The development of the MFR indicator aims to address the limitations of traditional body composition parameters (such as SMMI and visceral fat area) in evaluating the dynamic relationship between muscle and adipose tissue, as well as their sensitivity to edema [5]. By simultaneously considering muscle mass and fat content, this ratio reflects the metabolic advantages gained by the body during muscle gain and fat loss, and is informative for identifying individuals with normal body weight but concurrent sarcopenia and excessive visceral fat [12].

It should be emphasized that the imbalance between muscle and fat mass promotes DKD development through two mechanisms: on the one hand, sarcopenia induces inflammatory responses and stimulates renal fibrosis pathways; on the other hand, excessive fat triggers lipotoxicity, thereby damaging the structure and function of podocytes [13]. Thus, MFR not only integrates comprehensive information on muscle and adipose tissue but also establishes a critical link between metabolic disorders and renal damage.

The results of this study demonstrate that elevated MFR is an independent protective factor against renal function impairment and disease progression in DKD patients, which supports the hypothesis of the muscle-adipose axis [14]. Previous studies by Heitman et al. [15] also found that a lower MFR exacerbates renal damage through nuclear factor-κB-related inflammatory responses and mitochondrial dysfunction, and the conclusions of this study are consistent with these findings.

Different from previous research, this study employed binary logistic regression models. After adjusting for confounding factors such as age, diabetes duration, and TG, it was found that each 1-standard deviation increase in ZMFR was associated with an 83.4% reduction in the risk of renal function impairment. Further analysis based on the CGA staging system revealed that even after excluding Scr and UACR, which are directly related to staging definitions to avoid circular reasoning, elevated MFR still significantly reduced the risk of DKD progression to high/very high-risk stages. This finding suggests that MFR is not only independently associated with eGFR levels but also has early warning value for aggravated proteinuria and comprehensive disease deterioration, thereby expanding the application scope of this indicator from cardiovascular risk assessment to renal prognosis evaluation. In addition, through ROC curve analysis, this study further proposed an MFR cutoff value for identifying renal function decline, providing a basis for clinical hierarchical management.

From a practical application perspective, MFR can play a role in the following three aspects: first, taking MFR $\leqslant$ 0.534 as the warning threshold for renal dysfunction can facilitate early screening and risk stratification; second, personalized intervention plans, such as resistance training and controlled protein intake, can be formulated for patients with low MFR to restore muscle-adipose balance and delay DKD progression; third, a dynamic monitoring and follow-up system can be established by combining KDIGO staging with MFR values. Intensive management should be implemented for patients in stages G1-2A2 and G1-2A3 with MFR below 0.534 to restore muscle-adipose balance and delay disease progression to stage G3 or macroalbuminuria.

However, this study has several limitations. (1) As a single-center cross-sectional study, it is difficult to infer the causal relationship between MFR and DKD progression, which needs to be verified by subsequent prospective studies. (2) The study sample was derived from hospitalized patients with relatively severe conditions, so caution should be exercised when extrapolating the conclusions to community-based DKD populations. More primary care data should be included in future research. (3) The BIA method has low measurement accuracy in patients with severe edema, and cross-validation using dual-energy X-ray absorptiometry and other methods can be conducted in the future [16]. Intervention studies based on MFR stratification are planned to be carried out next to explore the impact of metabolic modulation on renal prognosis.

In conclusion, elevated MFR is an independent protective factor against renal function impairment and CGA staging progression in DKD patients, and its underlying mechanism may be related to the dysregulation of the muscle-adipose axis. In the future, long-term follow-up and intervention trials are needed to integrate

MFR into individualized DKD management strategies, which is expected to provide new strategies and evidence for the individualized precision treatment of DKD.

Conflict of interest None

Reference

[1] Han YN, Li L, Li YH, et al. Research progress of oxidative stress in the pathogenesis and treatment of diabetic complications[J]. Chin J Clin Res, 2025, 38(4): 515-519.**[In Chinese]**

[2] Drexler Y, Fornoni A. Adenine crosses the biomarker bridge: from 'omics to treatment in diabetic kidney disease[J]. J Clin Invest, 2023, 133(20): e174015.

[3] Cleveland KH, Schnellmann RG. Pharmacological targeting of mitochondria in diabetic kidney disease[J]. Pharmacol Rev, 2023, 75(2): 250-262.

[4] Fu Y, Sun Y, Wang M, et al. Elevation of JAML promotes diabetic kidney disease by modulating podocyte lipid metabolism[J]. Cell Metab, 2020, 32(6): 1052-1062.e8.

[5] Kim JA, Shin C, Jung I, et al. Impact of muscle quality on muscle strength and physical performance beyond muscle mass or diabetes status[J]. J Cachexia Sarcopenia Muscle, 2025, 16(2): e13760.

[6] Faria G, Gonçalves A, Cunha R, et al. Beyond central adiposity: Liver fat and visceral fat area are associated with metabolic syndrome in morbidly obese patients[J]. Int J Surg, 2015, 14: 75-79.

[7] do A H Souza AC, Troschel AS, Marquardt JP, et al. Skeletal muscle adiposity, coronary microvascular dysfunction, and adverse cardiovascular outcomes[J]. Eur Heart J, 2025, 46(12): 1112-1123.

[8] Yu PC, Hsu CC, Lee WJ, et al. Muscle-to-fat ratio identifies functional impairments and cardiometabolic risk and predicts outcomes: biomarkers of sarcopenic obesity[J]. J Cachexia Sarcopenia Muscle, 2022, 13(1): 368-376.

[9] Aguilar-Ramirez D, Alegre-Díaz J, Gnatiuc L, et al. Changes in the diagnosis and management of diabetes in Mexico City between 1998-2004 and 2015-2019[J]. Diabetes Care, 2021, 44(4): 944-951.

[10] de Boer IH, Khunti K, Sadusky T, et al. Diabetes management in chronic kidney disease: a consensus report by the American diabetes association (ADA) and kidney disease: improving global outcomes (KDIGO)[J]. Diabetes Care, 2022, 45(12): 3075-3090.

[11] Levey AS, Tighiouart H, Titan SM, et al. Estimation of glomerular filtration rate with vs without including patient race[J]. JAMA Intern Med, 2020, 180(5): 793.

[12] Chen X, Duan MR, Hou R, et al. Prevalence of abdominal obesity in Chinese middle-aged and older adults with a normal body mass index and its association with type 2 diabetes mellitus: a nationally representative cohort study from 2011 to 2018[J]. Diabetes Metab Syndr Obes, 2021, 14: 4829-4841.

[13] Tang GY, Li S, Zhang C, et al. Clinical efficacies, underlying mechanisms and molecular targets of Chinese medicines for diabetic nephropathy treatment and management[J]. Acta Pharm Sin B, 2021, 11(9): 2749-2767.

[14] Yang JK, Vamvini M, Nigro P, et al. Single-cell dissection of the obesity-exercise axis in adipose-muscle tissues implies a critical role for mesenchymal stem cells[J]. Cell Metab, 2022, 34(10): 1578-1593.e6.

[15] Heitman K, Alexander MS, Faul C. Skeletal muscle injury in chronic kidney disease-from histologic changes to molecular mechanisms and to novel therapies[J]. Int J Mol Sci, 2024, 25(10): 5117.

[16] Slart RHJA, Punda M, Ali DS, et al. Updated practice guideline for dual-energy X-ray absorptiometry (DXA)[J]. Eur J Nucl Med Mol Imag, 2025, 52(2): 539-563.

Submission Received: 2025-09-03 / **Revised:** 2025-09-28

·论　著·

2型糖尿病肾病患者肌肉脂肪质量比与肾功能损伤的相关性

崔思远，胡蕴，徐湘，李洋，赵涵，朱晓巍
南京医科大学附属无锡人民医院内分泌科，江苏 无锡 214000

摘要：目的　探讨肌肉脂肪质量比(MFR)与2型糖尿病肾病(DKD)患者肾功能损伤的关系，为评估DKD进展风险提供客观依据。**方法**　采用横断面研究方法，纳入2023年5月至2025年5月南京医科大学附属无锡人民医院277例住院DKD患者。使用生物电阻抗法测量体成分指标，包括身体质量指数、MFR和骨骼肌质量指数；同步检测血肌酐(Scr)、血尿素氮(BUN)及尿微量白蛋白/肌酐比值(UACR)，并依据慢性肾脏病流行病学协作组(CKD-EPI)公式计算估算肾小球滤过率(eGFR)。根据改善全球肾脏病预后组织(KDIGO)2022指南的CGA(病因-eGFR-白蛋白尿)分期系统，结合eGFR和UACR水平对患者进行风险分层分组：G1-2A2组[eGFR≥60 mL/(min·1.73 m²)且UACR 30~299 mg/g]、G1-2A3组[eGFR≥60 mL/(min·1.73 m²)且UACR≥300 mg/g]、G3A2组[eGFR 30~59 mL/(min·1.73 m²)且UACR 30~299 mg/g]、G3A3组[eGFR 30~59 mL/(min·1.73 m²)且UACR≥300 mg/g]及G4-5A2-3组[eGFR<30 mL/(min·1.73 m²)且UACR≥30 mg/g]。采用Spearman相关分析体成分指标与肾功能参数的相关性。二元logistic回归分析肾功能受损[eGFR<60 mL/(min·1.73 m²)]的独立影响因素，并进一步建立多因素logistic回归模型评估MFR对DKD综合进展风险(基于CGA分期高危/极高危)的预测价值，并绘制受试者工作特征(ROC)曲线评估MFR对肾功能下降的预测效能。**结果**　随着肾功能恶化(从G1-2A2组至G4-5A2-3组)，MFR呈降低趋势(P<0.01)。MFR与eGFR呈正相关(r=0.547，P<0.01)，与Scr、BUN及UACR均呈负相关(r=−0.341、−0.328、−0.136，P<0.05)。多因素logistic回归分析显示，在校正年龄、病程及血脂等混杂因素后，标准化MFR(ZMFR)升高是肾功能受损的独立保护因素(OR=0.166，95%CI：0.098~0.280，P<0.01)；同时，ZMFR亦是DKD进展至高危阶段的独立保护因素(OR=0.621，95%CI：0.426~0.904，P=0.013)。ROC曲线分析显示，MFR预测肾功能下降[eGFR<60 mL/(min·1.73 m²)]的曲线下面积为0.813(P<0.01)。**结论**　MFR下降与DKD患者肾功能损伤独立相关，MFR升高是肾功能受损及CGA分期进展的独立保护因素，监测MFR变化有助于早期识别高危患者，并为DKD肌肉-脂代谢干预提供新思路。

关键词：肌肉脂肪质量比；糖尿病肾病；肾功能损伤；横断面研究；生物电阻抗分析法
中图分类号：R587.2　**文献标识码：**A　**文章编号：**1674-8182(2026)01-0028-06

Association of muscle-to-fat ratio with renal impairment in type 2 diabetic nephropathy patients

CUI Siyuan, HU Yun, XU Xiang, LI Yang, ZHAO Han, ZHU Xiaowei
Department of Endocrinology, Wuxi People's Hospital Affiliated to Nanjing Medical University, Wuxi, Jiangsu 214000, China
Corresponding author: ZHU Xiaowei, E-mail: zhuxw@njmu.edu.cn

Abstract: Objective　To investigate the relationship between the muscle-to-fat ratio (MFR) and renal function impairment in patients with type 2 diabetic kidney disease (DKD), and to provide an objective basis for assessing the risk of DKD progression. **Methods**　A cross-sectional study was conducted, involving 277 hospitalized DKD patients at Wuxi People's Hospital Affiliated to Nanjing Medical University from May 2023 to May 2025. Body composition indicators, including body mass index (BMI), MFR, and skeletal muscle mass index (SMMI), were measured using bioelectrical impedance analysis (BIA). Simultaneously, serum creatinine (Scr), blood urea nitrogen (BUN), and

DOI：10.13429/j.cnki.cjcr.2026.01.005
基金项目：国家自然科学基金青年项目(82000685)
通信作者：朱晓巍，E-mail：zhuxw@njmu.edu.cn
出版日期：2026-01-20

QR code for English version

urinary microalbumin-to-creatinine ratio (UACR) were detected, and the estimated glomerular filtration rate (eGFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula. According to the CGA (cause-eGFR-albuminuria) staging system recommended by the Kidney Disease: Improving Global Outcomes (KDIGO) 2022 guidelines, patients were stratified into risk groups based on combined eGFR and UACR levels: Group G1-2A2 [eGFR≥60 mL/(min·1.73 m^2) and UACR 30~299 mg/g], Group G1-2A3 [eGFR≥60 mL/(min·1.73 m^2) and UACR≥300 mg/g], Group G3A2 [eGFR 30~59 mL/(min·1.73 m^2) and UACR 30~299 mg/g], Group G3A3 [eGFR 30~59 mL/(min·1.73 m^2) and UACR≥300 mg/g], and Group G4-5A2-3 [eGFR < 30 mL/(min·1.73 m^2) and UACR≥30 mg/g]. Spearman correlation analysis was used to examine the relationship between body composition indicators and renal function parameters. Binary logistic regression analysis was applied to identify independent influencing factors for renal impairment [eGFR<60 mL/(min·1.73 m^2)], and multivariate logistic regression was used to evaluate the predictive value of MFR for DKD progression risk based on CGA staging. Receiver operating characteristic (ROC) curves were plotted to assess the predictive efficacy of MFR for renal function decline. **Results** As renal function worsened (from the G1-2A2 group to the G4-5A2-3 group), MFR showed a decline trend (P<0.01). MFR was positively correlated with eGFR (r=0.547, P<0.01) and negatively correlated with Scr, BUN, and UACR (r=−0.341, −0.328, −0.136, P<0.05). Multivariate logistic regression analysis indicated that standardized MFR (ZMFR) was an independent protective factor for renal impairment (OR=0.166, 95%CI: 0.098−0.280, P<0.01) after adjusting for confounding factors. Additionally, ZMFR was identified as an independent protective factor against the progression to high-risk DKD stages (OR=0.621, 95%CI: 0.426−0.904, P=0.013). ROC analysis revealed that the area under the curve (AUC) for MFR in predicting renal function decline [eGFR<60 mL/(min·1.73 m^2)] was 0.813 (P<0.01). **Conclusion** A decrease in MFR is independently associated with renal function impairment in DKD patients. An increase in MFR is an independent protective factor for renal impairment and the progression of CGA staging. Monitoring changes in MFR may help identify high-risk patients early and provide new insights into muscle-fat metabolism interventions for DKD.

Keywords: Muscle-to-fat ratio; Diabetic kidney disease; Renal function impairment; Cross-sectional study; Bioelectrical impedance analysis

Fund program: Youth Project of the National Natural Science Foundation of China (82000685)

糖尿病肾病(diabetic kidney disease, DKD)是全球终末期肾病(end-stage renal disease, ESRD)的主要病因,约40%的2型糖尿病(type 2 diabetes mellitus, T2DM)患者受累,且其心血管死亡风险显著升高[1]。DKD的早期病理改变隐匿,目前DKD诊断依赖尿微量白蛋白/肌酐比值(urinary microalbumin-to-creatinine ratio, UACR)和估算肾小球滤过率(estimated glomerular filtration rate, eGFR),但二者仅在肾损伤相对严重时出现明显异常[2],对早期肾功能损伤敏感性不足。因此,开发更有效的风险预警工具及干预靶点,已成为当前DKD临床管理与治疗的迫切需求。

近年研究发现,肌肉-脂肪代谢失衡是DKD进展的关键病理机制。肌肉减少可激活炎症因子介导的蛋白质分解代谢,加剧胰岛素抵抗、肾脏微炎症和氧化应激,而内脏脂肪堆积则通过脂毒性反应促进肾单位脂质沉积,加重肾单位脂质负荷,形成“肌-肾轴”恶性循环[3-4]。然而,传统体成分指标如骨骼肌质量指数(skeletal muscle mass index, SMMI)和内脏脂肪面积仅评估单一组织,无法量化肌肉与脂肪的交互作用,且易受水肿及体质量波动干扰,难以准确评估机体的综合代谢状态[5-6]。

肌肉脂肪质量比(muscle-to-fat mass ratio, MFR)为全身肌肉质量与脂肪质量的比值,它能够同时量化肌肉储备和脂肪负荷,同步评估双向代谢,MFR还能识别和预警“正常体重型肥胖”,量化增肌/减脂的协同效应[7]。已有研究报道MFR与糖尿病患者心血管疾病的关联,并提示低MFR与T2DM患者冠状动脉钙化及心功能下降显著相关[8],但其在DKD肾功能损伤及疾病进展中的具体预警价值尚未明确。

基于此,本研究拟通过横断面调查,结合KDIGO指南推荐的CGA分期系统,探讨MFR与DKD肾功能受损及综合疾病进展风险的关联,旨在为DKD风险分层提供新依据,并为肌肉-脂肪轴代谢干预治疗T2DM并发症提供依据。

1 对象与方法

1.1 对象来源 本研究采用横断面设计,于2023年5月至2025年5月纳入南京医科大学附属无锡人民医院内分泌科住院的DKD患者277例。纳入标准:(1)符合1999年WHO糖尿病诊断标准[空腹血糖

(fasting blood glucose, FBG)≥7.0 mmol/L 或葡萄糖耐量试验 2 h 血糖≥11.1 mmol/L][9];(2) 符合改善全球肾脏病预后组织(Kidney Disease: Improving Global Outcomes, KDIGO)指南中 DKD 诊断[糖尿病病程≥5 年且 UACR≥30 mg/g 和/或 eGFR<60 mL/(min·1.73 m²)][10]。排除标准:(1) 存在体成分测量干扰因素(水肿、起搏器植入及严重视听/认知障碍等);(2) 患有严重疾病(血红蛋白≤90 g/L,丙氨酸氨基转移酶>120 u/L,活动性肿瘤及重症感染等);(3) 患有神经精神疾病(帕金森病、癫痫、痴呆及精神分裂症等)。初筛 400 例 T2DM 患者中,因甲状腺功能异常(n=12)、贫血(n=6)、肝功能不全(n=7)、肿瘤(n=8)、未完成体成分检测(n=52)、精神疾病(n=2)、痴呆(n=4)及评估缺失(n=32)共排除 123 例,最终纳入 277 例。本研究通过无锡市人民医院科研伦理委员会审核批准[(2023)67 号]。

1.2　*方法*

1.2.1　基线资料收集　本研究通过医院电子病历系统统一采集患者的基线资料。(1)患者的人口学特征数据(包括性别、年龄、身高、体质量、腰围、臀围及糖尿病病程);(2)患者的生化指标,包括糖化血红蛋白(glycosylated hemoglobin, HbA1c)、FBG、三酰甘油(triglyceride, TG)及低密度脂蛋白胆固醇(low density lipoprotein cholesterol, LDL-C);(3)计算身体质量指数(body mass index, BMI)、腰臀比(waist to hip ratio, WHR)。所有数据由两名研究员独立录入,不一致数据经第三方复核原始记录后校正。

1.2.2　肾功能指标测定及分组　肾功能评估采用标准化检测流程:血肌酐(serum creatinine, Scr)、尿肌酐、血尿素氮(blood urea nitrogen, BUN)通过酶法测定(罗氏 Cobas 8000),尿微量白蛋白采用免疫比浊法(西门子 ADVIA 2400),计算 UACR,并基于慢性肾脏病流行病学协作组(Chronic Kidney Disease Epidemiology Collaboration, CKD-EPI)公式计算 eGFR[11]。参照 KDIGO 2022 指南推荐的 CGA(病因-eGFR-白蛋白尿)分期系统对 DKD 患者进行风险分层[10]。考虑到本研究纳入对象均为已确诊 DKD 患者,即已处于 CGA 分期的中高风险区域,本研究依据 eGFR 分期(G 期)和白蛋白尿分期(A 期)的组合,将患者分为 5 个代表不同疾病进展阶段的临床亚组。(1) G1-2A2 组(中风险):肾功能[eGFR≥60 mL/(min·1.73 m²)]合并微量白蛋白尿(UACR 30~299 mg/g)。(2) G1-2A3 组(高风险):肾功能[eGFR≥60 mL/(min·1.73 m²)]合并大量白蛋白尿(UACR≥300 mg/g)。(3) G3A2 组(高风险):肾功能下降[eGFR 30~59 mL/(min·1.73 m²)]合并微量白蛋白尿(UACR 30~299 mg/g)。(4) G3A3 组(极高风险):肾功能下降[eGFR 30~59 mL/(min·1.73 m²)]且合并大量白蛋白尿(UACR≥300 mg/g)。(5) G4-5A2-3 组(极高风险/肾衰竭):肾功能严重受损或肾衰竭[eGFR<30 mL/(min·1.73 m²)]合并白蛋白尿(UACR≥30 mg/g)。

1.2.3　人体体成分测量　使用生物电阻抗分析(bioelectrical impedance analysis, BIA)仪器(InBody 720, BIOSPACE)进行标准化体成分测量。操作前要求患者禁食 8 h、排空膀胱、着轻便衣物并移除金属物品;测量时患者赤足站立于电极板,两脚与足电极分别紧密接触,双手握持电极柄,保持静止 5 min。设备直接输出体质量、脂肪质量(fat mass, FM)及骨骼肌质量(skeletal muscle mass, SMM),计算衍生指标:SMMI=SMM(kg)/身高²(m²);MFR=SMM/FM。

1.3　*统计学方法*　采用 SPSS 26.0 软件进行统计分析。符合正态分布的计量资料以 $\bar{x}\pm s$ 表示,非正态分布资料以 $M(Q_1, Q_3)$ 描述;计数资料以例(%)表示。多组间比较符合正态分布且方差齐性时采用单因素方差分析,否则采用 Kruskal-Wallis H 检验;分类变量比较采用 χ^2 检验。Spearman 秩相关用于分析体成分指标与肾功能参数的相关性。为消除量纲影响并比较效应强度,对 MFR 变量进行 Z-score 标准化处理。采用二元 logistic 回归分析标准化 MFR(ZMFR)对肾功能受损的独立影响。采用多因素 logistic 回归模型评估 ZMFR 对 DKD 综合进展风险的预测价值。采用受试者工作特征(receiver operating characteristic, ROC)曲线分析 MFR 对肾功能下降[eGFR<60 mL/(min·1.73 m²)]的预测效能,计算曲线下面积(area under the curve, AUC)和最佳截断值。所有统计检验均为双侧,α=0.05。

2　结　果

2.1　*基线特征资料比较*　277 例 DKD 患者中男性 144 例,女性 133 例。G1-2A2 组 53 例(19.13%),G1-2A3 组 57 例(20.58%),G3A2 组 49 例(17.69%),G3A3 组 63 例(22.74%)及 G4-5A2-3 组 55 例(19.86%)。各组患者基线特征比较结果见表 1。G4-5A2-3 组患者的糖尿病病程中位时间最长[16.0(13.0, 21.0)年]。随着肾功能进展,Scr、BUN 水平逐渐升高,eGFR 逐渐下降,G4-5A2-3 组 Scr、BUN 最高,eGFR 最低,组间比较差异有统计学意义(P<0.01)。人体成分方面,MFR 随肾功能下降呈降低趋势(P<0.01),从 G1-2A2 组的

0.77(0.64,0.98)降至G4-5A2-3组的0.61(0.48,0.66)。BMI、WHR和FM整体均呈上升趋势(P<0.01)。代谢指标中,5个组别FBG水平整体比较差异有统计学意义(P=0.047),其中G3A2组、G4-5A2-3组显著低于G1-2A2组,而G3A3组显著高于G1-2A2组及G1-2A3组(P<0.01),而HbA1c及TG在各组间的差异无统计学意义(P>0.05)。

2.2 人体体成分指标与肾功能指标的相关性分析 Spearman相关分析结果显示,MFR与eGFR呈正相关(P<0.01),与Scr、BUN及UACR呈负相关(P<0.05);SMMI与eGFR呈正相关(P=0.020),与BUN呈负相关(P=0.020),与Scr、UACR无相关性(P>0.05);WHR与eGFR呈负相关(P<0.01),与Scr、BUN及UACR呈正相关(P<0.05);BMI、FM与eGFR呈负相关(P<0.01),与Scr和BUN呈正相关(P<0.01),与UACR无显著相关性(P=0.267)。见表2。

2.3 肾功能受损影响因素的多因素logistic回归分析 以是否发生肾功能下降[eGFR<60 mL/(min·1.73 m^2)]为因变量(0=否,1=是),纳入ZMFR及年龄、病程、标准化WHR(ZWHR)、HbA1c、TG、LDL-C等进行二元logistic回归分析。结果显示,在校正了年龄、病程及TG等混杂因素后,ZMFR升高是肾功能受损的独立保护因素(P<0.01)。ZMFR每增加1个标准差,患者发生肾功能受损的风险降低83.4%。见表3。

2.4 肾功能受损影响因素的多因素logistic回归分析 依据KDIGO 2022指南CGA分期系统,将患者分为中风险组(G1-2A2组)与高危进展组(G1-2A3及以上组别)。以DKD综合进展风险为因变量(0=中风险,1=高危进展),剔除Scr、UACR等与分期定义直接相关的指标以避免多重共线性,将ZMFR及年龄、病程、HbA1c、TG、LDL-C、ZWHR纳入多因素logistic回归模型。结果显示,ZMFR升高与DKD高危进展风险降低独立相关(P=0.013)。ZMFR每增加1个标准差,DKD患者病情高危进展的风险降低37.9%。此外,病程、LDL-C、ZWHR均为DKD疾病进展的独立影响因素(P<0.05)。见表4。

2.5 MFR对DKD肾功能下降的预测效能 通过ROC曲线分析评估MFR对DKD患者肾功能下降[eGFR<60 mL/(min·1.73 m^2)]的预测价值。如图1所示,MFR预测肾功能下降的AUC为0.813(95%CI:0.760~0.866,P<0.01),表明MFR具有良好的预测效能。根据Youden指数确定最佳截断值为0.534,此时对应的敏感度和特异度分别为0.643和0.891。

表1 各组患者基线特征资料比较 [$M(Q_1,Q_3)$]

Tab.1 Comparison of baseline characteristics among different patient groups [$M(Q_1,Q_3)$]

指标	G1-2A2组(n=53)	G1-2A3组(n=57)	G3A2组(n=49)	G3A3组(n=63)	G4-5A2-3组(n=55)	$\chi^2/H/F$值	P值
男性[例(%)]	30(56.60)	32(56.14)	22(44.90)	31(49.21)	29(52.73)	2.040	0.728
年龄(岁)	53.0(45.5,57.0)	55.0(46.5,63.5)	52.0(44.5,58.5)	44.0(38.0,58.0)^{abc}	51.0(45.0,58.0)	11.751	0.019
病程(年)	9.0(6.5,12.0)	11.0(7.5,14.5)	11.0(7.0,14.5)	12.0(7.0,15.0)	16.0(13.0,21.0)^{abc}	48.867	<0.001
HbA1c(%,$\bar{x}\pm s$)	9.05±2.16	8.89±2.09	8.48±2.01	9.55±1.97	9.08±2.12	1.951	0.102
FBG(mmol/L,$\bar{x}\pm s$)	8.76±2.10	8.51±2.62	8.02±2.10^{a}	9.27±2.46^{ab}	8.27±2.25^{a}	2.449	0.047
TG(mmol/L)	2.39(1.26,4.57)	3.41(1.62,5.23)	2.19(1.16,4.97)	2.24(1.65,4.17)	3.08(2.58,3.86)	5.645	0.227
LDL-C(mmol/L)	2.69(2.28,3.24)	3.10(2.58,3.55)	2.86(2.37,3.42)	3.15(2.60,3.68)^{ab}	3.27(2.73,3.75)^{ab}	15.984	0.003
身高(cm)	170(163.0,174.0)	169(164.0,175.0)	166(157.5,175.5)^{a}	163(155.0,171.0)^{ab}	169(158.0,173.0)	15.589	0.004
体重(kg)	66(56,73)	58(52,64)^{a}	62(53,74)	66(59,71)^{b}	65(60,81)^{b}	39.108	<0.001
BMI(kg/m^2)	21.89(19.44,26.39)	20.69(17.94,22.30)^{a}	22.89(19.60,26.27)	24.28(21.85,27.73)^{ab}	23.81(22.21,28.63)^{b}	39.108	<0.001
腰围(cm)	77.0(72.0,86.5)	87.0(80.5,93.0)^{a}	87.0(80.0,92.5)^{a}	86.0(77.0,95.0)^{ab}	86.0(75.0,92.0)^{ab}	18.425	0.001
臀围(cm)	89.0(86.0,95.0)	94.0(90.0,98.0)^{a}	91.0(87.5,95.5)	92.0(88.0,97.0)^{ab}	90.0(85.0,95.0)^{ab}	12.895	0.012
WHR	0.87(0.82,0.91)	0.90(0.86,0.99)^{a}	0.92(0.89,1.01)^{a}	0.91(0.85,0.99)^{ab}	0.95(0.87,1.00)^{abcd}	20.297	<0.001
SMM(kg)	26.0(24.4,27.6)	23.2(21.0,27.2)^{a}	21.3(19.8,23.1)^{ab}	21.9(20.1,25.1)^{ab}	22.0(20.9,23.5)^{ab}	52.098	<0.001
SMMI(kg/m^2)	8.8(8.4,9.7)	8.2(7.4,9.1)	7.7(7.1,8.7)^{a}	8.3(7.5,9.0)	7.9(7.1,8.6)^{a}	29.447	<0.001
FM(kg)	33.6(24.3,40.3)	27.0(23.0,34.2)^{a}	33.6(27.0,41.3)^{b}	35.9(31.1,39.7)^{ab}	36.9(32.4,47.6)^{ab}	37.141	<0.001
MFR	0.77(0.64,0.98)	0.88(0.69,1.01)^{a}	0.71(0.50,0.79)^{ab}	0.66(0.54,0.71)^{abc}	0.61(0.48,0.66)^{abcd}	64.019	<0.001
Scr(μmol/L)	83.4(75.5,95.9)	90.0(80.5,102.6)	109.2(89.1,126.9)^{a}	139.1(124.6,153.2)^{ab}	197.1(143.1,269.8)^{abcd}	166.65	<0.001
BUN(mmol/L)	5.61(4.57,6.95)	5.85(4.86,7.12)	7.79(5.98,9.27)^{a}	8.48(6.86,9.35)^{ab}	9.39(7.30,10.91)^{abcd}	97.606	<0.001
UACR(mg/g)	76.5(57.3,122.9)	508.2(406.8,681.4)^{a}	163.2(98.6,198.6)^{ab}	593.0(500.6,635.1)^{abc}	386.3(282.3,564.4)^{abcd}	200.815	<0.001
eGFR[mL/(min·1.73 m^2)]	85.0(78.0,92.5)	84.0(77.0,89.5)	50.0(45.0,55.0)^{ab}	43.0(39.0,48.0)^{abc}	17.0(14.0,21.0)^{abcd}	209.056	<0.001

注:与G1-2A2组比较,^{a}P<0.05;与G1-2A3组比较,^{b}P<0.05;与G3A2组比较,^{c}P<0.05;与G3A3组比较,^{d}P<0.05。

表2　人体体成分指标与肾功能指标的相关性分析

Tab.2　Correlation analysis between body composition indicators and renal function indicators

指标		MFR	SMMI	WHR	BMI	FM
eGFR	*r*值	0.547	0.139	−0.323	−0.426	−0.396
	*P*值	<0.001	0.020	<0.001	<0.001	<0.001
UACR	*r*值	−0.136	−0.082	0.153	0.064	0.067
	*P*值	0.023	0.174	0.011	0.289	0.267
Scr	*r*值	−0.341	−0.022	0.215	0.312	0.284
	*P*值	<0.001	0.711	<0.001	<0.001	<0.001
BUN	*r*值	−0.328	−0.140	0.150	0.214	0.238
	*P*值	<0.001	0.020	0.012	<0.001	<0.001

表3　DKD患者肾功能受损的多因素logistic回归分析

Tab.3　Multivariate logistic regression analysis of renal impairment in DKD patients

因素	*β*	标准误	Wald χ^2	*P*值	*OR*(95%*CI*)
年龄	−0.043	0.015	7.702	0.006	0.958(0.929~0.987)
病程	0.098	0.032	9.586	0.002	1.103(1.037~1.174)
HbA1c	0.050	0.080	0.392	0.531	1.051(0.899~1.230)
TG	−0.181	0.075	5.859	0.016	0.834(0.720~0.966)
LDL-C	0.169	0.196	0.743	0.389	1.184(0.807~1.738)
ZWHR	0.310	0.218	2.029	0.154	1.364(0.890~2.090)
ZMFR	−1.798	0.269	44.823	<0.001	0.166(0.098~0.280)
(常量)	−1.534	2.295	0.447	0.504	

表4　基于CGA分期的DKD高危进展风险的多因素logistic回归分析

Tab.4　Multivariate logistic regression analysis of high-risk DKD progression based on CGA staging

因素	*β*	标准误	Wald χ^2	*P*值	*OR*(95%*CI*)
年龄	0.002	0.016	0.018	0.892	1.002(0.971~1.034)
病程	0.085	0.036	5.550	0.018	1.089(1.014~1.168)
HbA1c	−0.034	0.080	0.181	0.670	0.966(0.825~1.131)
TG	−0.116	0.074	2.467	0.116	0.890(0.770~1.029)
LDL-C	0.802	0.257	9.708	0.002	2.229(1.346~3.691)
ZWHR	1.098	0.281	15.220	<0.001	2.998(1.727~5.025)
ZMFR	−0.476	0.192	6.177	0.013	0.621(0.426~0.904)
(常量)	−0.911	1.313	0.482	0.488	

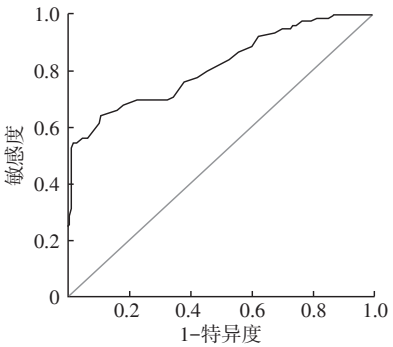

图1　MFR预测DKD患者肾功能下降的ROC曲线

Fig.1　ROC curve of MFR for predicting renal function decline in patients with DKD

3　讨　论

MFR这一指标的发展，旨在解决传统人体成分参数（如SMMI和内脏脂肪面积）在评估肌肉与脂肪动态关系时的不足，以及其对水肿状态的敏感性问题[5]。该比值通过同时考量肌肉量与脂肪含量，体现机体在肌肉增加和脂肪减少过程中所获得的代谢优势，并对识别体质量正常但实际存在肌肉减少和内脏脂肪过多的人群具有提示意义[12]。需强调的是，肌肉与脂肪比例失调可通过两种机制促进DKD发展：一方面，肌肉减少会诱发炎症反应并刺激肾脏纤维化路径；另一方面，过多脂肪可引发脂毒性，进而损害足细胞结构及功能[13]。由此可见，MFR不仅整合了肌肉与脂肪的综合信息，还在代谢紊乱与肾脏损害之间建立起重要联系。

本研究结果显示，MFR升高是DKD患者肾功能受损及疾病进展的独立保护因素，这一发现支持了肌肉-脂肪轴相关假说[14]。既往如Heitman等[15]也发现较低的MFR会通过核因子-κB相关炎症反应与线粒体功能异常而加重肾脏损害，本研究得出的结论与其一致。与以往研究不同的是，本研究采用二元logistic回归模型，在校正年龄、病程及TG等混杂因素后，发现ZMFR每增加1个标准差，患者发生肾功能受损的风险降低83.4%。进一步对CGA分期的分析显示，即便剔除了与分期定义直接相关的Scr和UACR以避免循环论证，MFR升高仍可显著降低DKD向高危/极高危阶段进展的风险。这一结果提示，MFR不仅与eGFR水平独立相关，更对蛋白尿加重和综合病情恶化具有早期预警价值，从而拓展了该指标从心血管风险评估至肾脏病预后判断的适用范畴。另外，通过ROC曲线分析，本研究进一步提出了用于判断肾功能下降的MFR切点值，为临床分级管理提供了依据。

从实践应用角度，MFR可在以下三个方面发挥作用：其一，将MFR≤0.534视为肾功能减退的预警界值，助力早期筛查与分层；其二，为MFR偏低者定制个性化方案，如力量训练与可控蛋白摄入，以恢复肌肉-脂肪平衡并延缓DKD进展；其三，联合KDIGO分期与MFR数值建立动态监测随访体系，对已处于G1-2A2、G1-2A3期但MFR低于0.534的患者实行强化管理以恢复肌肉-脂肪平衡，延缓疾病向G3期或大量蛋白尿阶段进展。

然而，本研究仍存在若干局限：（1）本研究为单中心横断面设计，难以推断MFR与DKD进展间的因果联系，需后续前瞻性研究加以验证；（2）样本来自住院患者，一般病情较重，结论外推至社区DKD人群时需谨慎，今后需纳入更多基层数据；（3）BIA方法

（下转第37页）

者肾脏进展的关系[J]. 中华医学杂志, 2025, 105(12):888-893.
[9] Xie J, Wang ZH, Zhang ZJ, et al. Adjusted glycated albumin is a novel indicator of glycemic control in patients with macroalbuminuria in diabetic kidney disease[J]. Sci Rep, 2025, 15(1):13812.
[10] Butler AE, Hunt SC, Kilpatrick ES. Using nephropathy as an outcome to determine the HbA1c diagnostic threshold for type 2 diabetes[J]. Diabetes Metab Syndr, 2024, 18(4):103005.
[11] Dwivedi S, Sikarwar MS. Diabetic nephropathy: pathogenesis, mechanisms, and therapeutic strategies[J]. Horm Metab Res, 2025, 57(1):7-17.
[12] Hu SW, Hang X, Wei Y, et al. Crosstalk among podocytes, glomerular endothelial cells and mesangial cells in diabetic kidney disease: an updated review[J]. Cell Commun Signal, 2024, 22(1):136.
[13] Zhang ZT, Qi YL, Chen P, et al. Dang-Gui-Bu-Xue decoction against diabetic nephropathy via modulating the carbonyl compounds metabolic profile and AGEs/RAGE pathway[J]. Phytomedicine, 2024, 135:156104.
[14] Bahreini N, Saghafi-Asl M, Nikpayam O, et al. Effects of dried okra extract on lipid profile, renal function and some RAGE-related inflammatory genes expression in patients with diabetic nephropathy: a randomized controlled trial[J]. Complement Ther Med, 2024, 81:103027.
[15] 杨录波, 叶韬, 李丽敏, 等. HbA1c、Betatrophin对2型糖尿病肾脏疾病相关性及诊断价值研究[J]. 中国中西医结合肾病杂志, 2024, 25(4):319-322.
[16] Wang JW, Jin CH, Ke JF, et al. GA/HbA1c ratio is a simple and practical indicator to evaluate the risk of metabolic dysfunction-associated fatty liver disease in type 2 diabetes: an observational study[J]. Diabetol Metab Syndr, 2022, 14(1):167.
[17] Yang TY, Qi F, Guo F, et al. An update on chronic complications of diabetes mellitus: from molecular mechanisms to therapeutic strategies with a focus on metabolic memory[J]. Mol Med, 2024, 30(1):71.
[18] Hu LJ, Li J, Wang WZ, et al. Hypertension accelerates the development of a diabetic nephropathy model with more relevant clinical features in Dahl salt-sensitive rat[J]. J Hypertens, 2025, 43(7):1225-1231.
[19] Huang XM, Ma X, Lu JY, et al. Relationship between serum IL-6, VAP-1, Cys C, Scr, BUN, and type 2 diabetic kidney disease[J]. Minerva Endocrinol, 2023, 48(1):121-122.
[20] 王亚玲, 杨芳婷, 王惠丽, 等. 2型糖尿病患者慢性肾脏病风险感知水平及相关因素[J]. 中国临床研究, 2025, 38(9): 1364-1367.
[21] 史曼曼, 马毓华, 郑金鑫, 等. 1990年至2021年全球及中国2型糖尿病导致的慢性肾脏病疾病负担及危险因素分析[J]. 诊断学理论与实践, 2025, 24(3): 268-278.
[22] Zhang ZF, Wang H, Chen MY, et al. L-shaped association between the GA/HbA1c ratio and all-cause mortality in U.S. adults with NAFLD: a cross-sectional study from the NHANES 1999-2004[J]. BMC Endocr Disord, 2024, 24(1):35.

收稿日期:2025-07-14 **修回日期**:2025-09-17 **编辑**:王宇

(上接第32页)

对严重水肿患者测定准确性较低,未来可采用双能X线吸收法等方法进行交叉验证[16]。计划下一步实施基于MFR分层的干预研究,以探讨代谢调整对肾脏预后的影响。

综上所述,MFR升高是DKD肾功能受损及CGA分期进展的独立保护因素,其作用机制或与肌肉-脂肪轴失调有关。今后需通过长期随访与干预试验,将MFR整合进DKD个体化管理策略,有望为DKD的个体化精准治疗提供新的策略与证据支持。

利益冲突 无

参考文献

[1] 韩宇南, 李琳, 李玉洪, 等. 氧化应激在糖尿病并发症发病机制及治疗中的研究进展[J]. 中国临床研究, 2025, 38(4): 515-519.
[2] Drexler Y, Fornoni A. Adenine crosses the biomarker bridge: from 'omics to treatment in diabetic kidney disease[J]. J Clin Invest, 2023, 133(20):e174015.
[3] Cleveland KH, Schnellmann RG. Pharmacological targeting of mitochondria in diabetic kidney disease[J]. Pharmacol Rev, 2023, 75(2):250-262.
[4] Fu Y, Sun Y, Wang M, et al. Elevation of JAML promotes diabetic kidney disease by modulating podocyte lipid metabolism[J]. Cell Metab, 2020, 32(6):1052-1062.e8.
[5] Kim JA, Shin C, Jung I, et al. Impact of muscle quality on muscle strength and physical performance beyond muscle mass or diabetes status[J]. J Cachexia Sarcopenia Muscle, 2025, 16(2):e13760.
[6] Faria G, Gonçalves A, Cunha R, et al. Beyond central adiposity: liver fat and visceral fat area are associated with metabolic syndrome in morbidly obese patients[J]. Int J Surg, 2015, 14:75-79.
[7] Souza ACDAH, Troschel AS, Marquardt JP, et al. Skeletal muscle adiposity, coronary microvascular dysfunction, and adverse cardiovascular outcomes[J]. Eur Heart J, 2025, 46(12):1112-1123.
[8] Yu PC, Hsu CC, Lee WJ, et al. Muscle-to-fat ratio identifies functional impairments and cardiometabolic risk and predicts outcomes: biomarkers of sarcopenic obesity[J]. J Cachexia Sarcopenia Muscle, 2022, 13(1):368-376.
[9] Aguilar-Ramirez D, Alegre-Díaz J, Gnatiuc L, et al. Changes in the diagnosis and management of diabetes in mexico city between 1998-2004 and 2015-2019[J]. Diabetes Care, 2021, 44(4):944-951.
[10] de Boer IH, Khunti K, Sadusky T, et al. Diabetes management in chronic kidney disease: a consensus report by the american diabetes association (ADA) and kidney disease: Improving Global Outcomes (KDIGO)[J]. Diabetes Care, 2022, 45(12):3075-3090.
[11] Levey AS, Tighiouart H, Titan SM, et al. Estimation of glomerular filtration rate with *vs* without including patient race[J]. JAMA Intern Med, 2020, 180(5):793-795.
[12] Chen X, Duan MR, Hou R, et al. Prevalence of abdominal obesity in Chinese middle-aged and older adults with a normal body mass index and its association with type 2 diabetes mellitus: a nationally representative cohort study from 2011 to 2018[J]. Diabetes Metab Syndr Obes, 2021, 14:4829-4841.
[13] Tang GY, Li S, Zhang C, et al. Clinical efficacies, underlying mechanisms and molecular targets of Chinese medicines for diabetic nephropathy treatment and management[J]. Acta Pharm Sin B, 2021, 11(9):2749-2767.
[14] Yang J, Vamvini M, Nigro P, et al. Single-cell dissection of the obesity-exercise axis in adipose-muscle tissues implies a critical role for mesenchymal stem cells[J]. Cell Metab, 2022, 34(10):1578-1593.e6.
[15] Heitman K, Alexander MS, Faul C. Skeletal muscle injury in chronic kidney disease-from histologic changes to molecular mechanisms and to novel therapies[J]. Int J Mol Sci, 2024, 25(10):5117.
[16] Slart RHJA, Punda M, Ali DS, et al. Updated practice guideline for dual-energy X-ray absorptiometry (DXA)[J]. Eur J Nucl Med Mol Imaging, 2025, 52(2):539-563.

收稿日期:2025-09-03 **修回日期**:2025-09-28 **编辑**:许煜晗