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Association between diabetic kidney disease and low muscle mass in patients with type 2 diabetes: a cross-sectional study

Huang Qiuhong*, Cheng Qingfeng, Zeng Qinglian, Ni Xia, Li Xun

Department of Endocrinology, The First Affiliated Hospital of Chongqing Medical University, Chongqing, 400016, China
**Corresponding author: hqiuh0817@yeah.net*

Abstract: *This study included 1241 inpatients with type 2 diabetes mellitus(T2DM) at the First Affiliated Hospital of Chongqing Medical University, China, from October 2013 to October 2018 to investigate the relationship between diabetic kidney disease and low muscle mass. The body composition of patients was measured with dual-energy X-ray-absorptiometry. Social history, medical history, and Clinical indexes were all collected to analyze. In the results, this study found: (1) The percentage of low muscle mass in the population with diabetic complications and cardiovascular and cerebrovascular diseases was far more than the diabetic patients without those; (2) Urinary albumin-to-creatinine ratio (UACR) in patients with low muscle mass was significantly higher than that observed in the cases without [23.3(5.80, 193.35) vs. 16.45(4.80, 76.73), P=0.01], and the prevalence of low muscle mass was positively correlated with stratified UACR (Cramer's V=0.086, P for Cramer's V=0.011); (3) Diabetic kidney disease was one of the independent risk factors for low muscle mass in patients with T2DM. [OR=1.64(1.05, 2.55), P=0.02].*

Keywords: *Diabetes mellitus; Diabetic kidney disease; Low muscle mass; Urinary albumin-to-creatinine ratio; Sarcopenia*

1. Introduction

With the aging of the world population, there is growing concern about sarcopenia. In 1989, Rosenberg first defined sarcopenia as a decline in age-related muscle mass. With a better understanding of sarcopenia, the European Working Group on Low muscle mass in Older People (EWGSOP2) has recently redefined it as a progressive and generalized skeletal muscle disorder associated with an increased likelihood of adverse outcomes, including falls, fractures, physical disability, and mortality[1]. Currently, sarcopenia has become a significant public health problem. In terms of health, it increases the risk of falls, fractures, and mortality [2]. In financial terms, it increases hospitalization rates and costs[3].

Low muscle mass was primarily associated with aging in the past; however, it has been recently found in younger individuals [4] and has been determined to be related to many non-age factors, including daily behavior and disease-related factors[5]. Daily behavior includes daily nutrient intake and daily exercise. Disease-related factors include a series of diseases that cause malnutrition and anabolic disorders, such as Type 2 Diabetes mellitus(T2DM). T2DM is associated with insulin resistance, inflammation, increased oxidative stress, and advanced glycation end-products (AGEs) accumulation, contributing to muscle mass, strength, and function loss. Thus sarcopenia occurs [6]. Sarcopenia can also influence glucose metabolism through complex mechanisms. T2DM and Sarcopenia interact with each other. At the same time, Patients with DKD have a higher relative risk of mortality and heavier economic burden than those with diabetes alone[7]. Here this study was conducted to investigate the association between DKD and sarcopenia in patients with T2DM.

2. Materials and Methods

2.1 Study Design and Population

Adults were included in this retrospective study if they were diagnosed with T2DM based on WHO 1999 standards or previous medical records. Patients with any one of the following conditions were

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excluded: (1) Severe heart failure (New York Heart Association Class III-IV); (2) Severe liver impairment (alanine aminotransferase≥ 3-fold the upper limit of normal range); (3) With Malignant tumor; (4) Long-term stay in bed or with severe malnutrition. The final study population comprised 1,241 patients from the Endocrinology Department at the First Affiliated Hospital of Chongqing Medical University, China, from October 2013 to October 2018. All subjects provided informed consent. The study was approved by the Ethics Committee of the First Affiliated Hospital of Chongqing Medical University, China (2020-236) .

2.2 General Data Collection

General data of the inpatients were collected from the electronic medical record database of the hospital, including age, gender, body mass index (BMI), smoking history, duration of T2DM, and some laboratory examination index. We evaluated Chronic diabetic complications, including diabetic foot (DF), diabetic kidney disease (DKD), diabetic retinopathy (DR), and diabetic peripheral neuropathy (DPN). DKD assessment relied upon eGFR < 60 mL/min/1.73 m2 or UACR >30 mg/g[8]. In addition, cardiovascular and cerebrovascular diseases were collected. This research used a DXA Hologic scanner to measure body composition. The Low muscle mass was diagnosed according to AWGS 2019 Consensus that appendicular skeletal muscle mass (ASM) is below 7.0 kg/m^2 in men and 5.4 kg/m^2 in women[9].

2.3 Statistical Analysis

The data were presented as the Mean ± standard deviation (SD) for normally distributed variables and tested by independent t-tests. While the nonnormally distributed variables were presented as medians (interquartile ranges) and analyzed by the Mann-Whitney U test. Categorical variables were reported as frequencies and proportions, and Chi-square tests or Fisher's exact tests were used for group comparisons. Kruskal-Wills H test was used for grade data. Bonferroni correction was applied to all multiple comparisons. Cramer's V value suggested the correlations. Continuous missing data were handled using the Expectation Maximization (EM) method. We matched the low muscle mass and comparison group based on the propensity score. Multivariable logistic regression was used to test the relationship between low muscle mass and DKD. At last, we performed several sensitivity analyses to check the consistency of the results obtained in the primary analysis. The significance level was predetermined at P <0.05 for all tests. All statistical analyses were conducted using SPSS version 26.0 and software R version 4.0.0.

3. Results

3.1 Statistical Analysis of General Data of the Study Subjects

The study population, 1241 patients with T2DM, was divided into two groups: without low muscle mass (n = 1008) and with low muscle mass (n = 233). Participants' general characteristics are shown in Table 1. Compared to the group without low muscle mass, patients with low muscle mass were older (66.83 ± 10.56 vs. 64.3 ± 9.48, P<0.001), were more likely to have had a prior arteriosclerosis obliterans (15.9% vs. 3.5%), cerebrovascular disease (12.7% vs. 9%), DF (44.2% vs. 15.2%), DPN (87.8% vs. 73.5%), DR (37.5% vs. 29.1%), and men accounted for the main part (77.7% vs. 23.3%). Moreover, the low muscle mass group showed higher HbA1c, WBC, NC, PLT, and UACR and lower levels of BMI, Hb, ALT, AST, TC, TG, LDL-C, eGFR, UA, and Albumin. The baseline analysis of EM-imputed and PS-matched data showed a similar result in supplementary materials.

Additionally, Figure 1(supplementary materials) showed that UACR in patients with low muscle mass was significantly higher than that observed in the subjects without (23.3 (5.80, 193.35) vs. 16.45 (4.80, 76.73), P = 0.01). To go a step further, UACR was stratified (UACR <30mg/g; 30 ≤ UACR < 300mg/g; and UACR ≥300mg/g). As was shown in (supplementary materials), it demonstrated the result of the difference between stratified UACR level and low muscle mass (P = 0.011) tested by the Kruskal-Wallis H test and Bonferroni's multiple comparisons. And the prevalence of low muscle mass was positively correlated with stratified UACR (Cramer's V=0.086, P for Cramer's V=0.011).

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Table 1: General Data of the Study Subjects

Variables	Level	Without low muscle mass (n=1008)	Low muscle mass (n=233)	t/Z/χ^2	*P*
Gender (%)	Male	555 (55.1)	181 (77.7)	40.13	<0.001*
	Female	453 (44.9)	52 (22.3)		
Smoking (%)	No	620 (62.1)	95 (40.8)	35.16	<0.001*
	Yes	379 (37.9)	138 (59.2)		
Cardiovascular and Cerebrovascular Diseases					
Hypertension (%)	No	363 (36.1)	105 (45.1)	6.49	0.01 *
	Yes	643 (63.9)	128 (54.9)		
Arteriosclerosis Obliterans (%)	No	891 (96.5)	191 (84.1)	50.29	<0.001*
	Yes	32 (3.5)	36 (15.9)		
Coronary Heart Disease (%)	No	773 (83.7)	183 (79.9)	1.91	0.17
	Yes	150 (16.3)	46 (20.1)		
Cerebrovascular Disease (%)	No	840 (91.0)	200 (87.3)	2.82	0.09
	Yes	83 (9.0)	29 (12.7)		
Diabetic Complication					
DF (%)	No	855 (84.8)	130 (55.8)	97.39	<0.001*
	Yes	153 (15.2)	103 (44.2)		
DPN (%)	No	250 (26.5)	27 (12.2)	20.24	<0.001*
	Yes	692 (73.5)	194 (87.8)		
DR (%)	No	653 (70.9)	140 (62.5)	5.97	0.02*
	Yes	268 (29.1)	84 (37.5)		
DKD (%)	No	650 (64.5)	145 (62.8)	0.24	0.62
	Yes	358 (35.5)	86 (37.2)		
Age (year)		64.30 ± 9.48	66.83 ± 10.56	-3.36	<0.001*
BMI (kg/m²)		25.32 ± 2.97	21.35 ± 2.39	21.79	<0.001*
Duration of Diabetes (year)		9.48 ± 6.79	9.65 ± 7.57	-0.32	0.75
HbA1c (%)		8.20(6.90, 9.81)	9.00(7.20, 11.00)	-3.74	<0.001*
White Blood Cell(10^9/L)		6.37 (5.44, 7.59)	6.92 (5.45, 8.29)	-3.66	<0.001*
NC (%)		64.74 ± 9.55	68.64 ± 10.13	-5.35	<0.001*
PLT (10^9/L)		179.30 (148.00, 220.00)	198.00(158.0,251.00)	-3.86	<0.001*
Hemoglobin (g/L)		131.69 ± 18.98	126.35 ± 22.22	3.39	<0.001*
ALT (U/L)		21.00 (14.00, 30.00)	19 (12.00, 26.00)	-3.15	0.002*
AST (U/L)		19.96 (15.00, 24.32)	18 (14.00, 23.00)	-3.67	<0.001*
Total Cholesterol (mmol/L)		4.18 ± 1.10	4.03 ± 1.10	1.89	0.06
Triglyceride (mmol/L)		1.45 (1.00, 2.10)	1.25 (0.92, 1.69)	-4.11	<0.001*
LDL-C (mmol/L)		2.44 (1.80, 3.18)	2.3 (1.85, 3.13)	-0.44	0.66
HDL-C (mmol/L)		1.13 ± 0.35	1.11 ± 0.35	0.67	0.50
UACR (mg/g)		16.45 (4.80, 76.73)	23.30 (5.80, 193.35)	-2.68	0.01*
Creatinine (umol/L)		72.00 (59.00, 88.00)	74.00 (59.00, 91.00)	-1.11	0.27
eGFR (ml/min/1.73 m²)		79.02 (59.79, 110.96)	72.59 (53.21, 98.28)	-3.11	0.002*
Uric Acid (umol/L)		312.21 ± 90.39	288.82 ± 95.81	3.52	<0.001*
Albumin (g/L)		40.81 ± 4.77	38.40 ± 5.39	6.27	<0.001*
ASM (kg/m²)		7.02 ± 0.95	5.87 ± 0.73	20.49	<0.001*

Data are presented as mean ± SD, median (interquartile range) or %. BMI: body mass index, HDL-C: high-density lipoprotein cholesterol, LDL-C: low-density lipoprotein cholesterol, eGFR: estimated glomerular filtration rate, UACR: urinary albumin-to-creatinine ratio, UA: uric acid, HbA1c: glycosylated hemoglobin, TC: total cholesterol, TG: triglycerides, ALT: alanine aminotransferase, AST: aspartate aminotransferase, PLT: platelet count, WBC: white blood cell count, and NC: neutrophilic granulocyte percentage, ASM: appendicular skeletal muscle mass. * P<0.05.

3.2 Primary Analysis

The primary analysis was conducted to estimate the risk factors promoting low muscle mass and the relationship between DKD and low muscle mass. Multivariable logistic regression analysis excluded confounding factors, and three different models were developed (see Table 2). It is clear from the outcome that DKD, male, aging, lower BMI, higher WBC, and arteriosclerosis obliterans were independent risk factors for low muscle mass; however, unexpectedly, higher eGFR increased the risk of muscle mass loss. The three models showed similar results regarding the association between DKD and muscle mass loss (Model 1 OR (CI%) = 1.82 (1.22, 2.71), P=0.003; Model 2 OR (CI%) = 1.64 (1.09, 2.48), P=0.02; Model 3 OR (CI%) =1.64 (1.05, 2.55), P=0.02) (Table 2). And the same analysis was conducted in the EM data, illustrating a similar result (supplementary materials).

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Table 2: Multivariable Analysis for Logistic Regression of Low Muscle Mass

Variables	No. of Patients (%)	Model1[a]		Model2[b]		Model3[c]	
		OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*
DKD (%)							
Yes	444 (35.8)	1.82 (1.22, 2.71)	0.003*	1.64 (1.09, 2.48)	0.02*	1.64 (1.05, 2.55)	0.02*
No	796 (64.2)	1		1		1	
Gender (%)							
Female	506 (40.7)	0.22 (0.14, 0.34)	<0.001*	0.20 (0.13, 0.32)	<0.001*	0.2 (0.13, 0.32)	<0.001*
Male	735 (59.3)	1		1		1	
Age (year)		1.04 (1.02, 1.06)	<0.001*	1.03 (1.01, 1.05)	0.01*	1.03 (1.01, 1.05)	<0.001*
BMI (kg/m²)		0.51 (0.46, 0.56)	<0.001*	0.50 (0.46, 0.56)	<0.001*	0.50 (0.46, 0.56)	<0.001*
Arteriosclerosis Obliterans (%)							
Yes	68 (5.9)			5.98 (2.95, 12.11)	<0.001*	5.76 (2.84, 11.68)	<0.001*
No	1082 (94.1)			1		1	
Hypertension (%)							
Yes	771 (62.2)			1.13 (0.74, 1.73)	0.57	1.17 (0.76, 1.79)	0.58
No	468 (37.8)			1		1	
Cerebrovascular Disease (%)							
Yes	112 (9.7)			1.31 (0.73, 2.35)	0.37	1.35 (0.75, 2.43)	0.36
No	1041 (90.3)			1		1	
Hemoglobin (g/L)						1.00 (0.98, 1.01)	0.39
Albumin (g/L)						1.04 (0.99, 1.09)	0.15
HbA1c (%)						1.04 (0.95, 1.14)	0.38
eGFR (ml/min/1.73 m²)						1.01 (1.00, 1.02)	0.01*
WBC (10^9/L)						1.11 (1.03, 1.20)	0.01*
ALT (U/L)						1.00 (0.98, 1.01)	0.54
UA (umol/L)						1.00 (1.00, 1.00)	0.35
TG (mmol/L)						1.03 (0.90, 1.18)	0.67
HDL-C (mmol/L)						0.58 (0.30, 1.12)	0.11
LDL-C (mmol/L)						0.99 (0.97, 1.01)	0.42

*[a]Model1: Adjusted for DKD, Gender, Age, and BMI. [b]Model2: Model1+ adjusted for Arteriosclerosis Obliterans, Hypertension, and Cerebrovascular Disease. [c]Model3: Model2+ adjusted for Hemoglobin, Albumin, HbA1c, eGFR, WBC, ALT, UA, TG, HDL-C, LDL-C. *P<0.05.*

3.3 Sensitivity Analysis

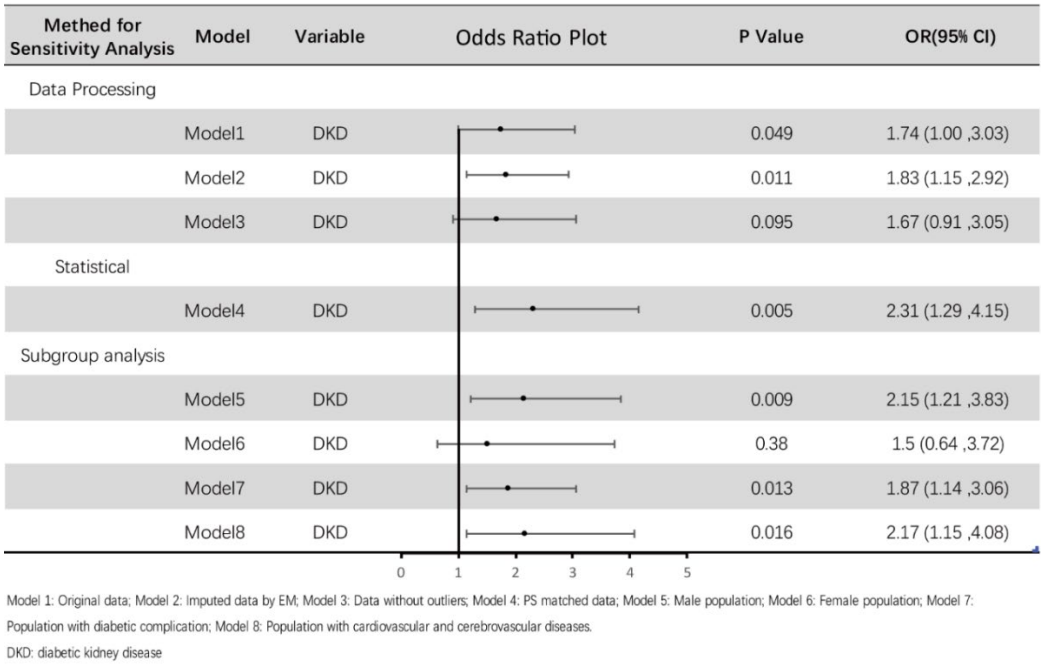

Figure 1: Forest plot: Different Methods for Sensitivity Analysis in Logistic Analysis.

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In Figure 1, several sensitivity analyses were performed to verify the relationship between DKD and sarcopenia. From a data standpoint, the primary analysis of original data, imputed data by EM, and outliers removed data were carried out. From a statistical perspective, PSM data was conducted. For the subgroup analysis, logistic regression was performed in a population stratified by gender in people with diabetic complications or cardiovascular and cerebrovascular diseases. The sensitivity analysis yielded consistent results.

4. Discussion

Diabetic patients are more likely to suffer from sarcopenia. DKD is one of the most common and critical complications of diabetes. Previous studies have explored the relationship between DKD and sarcopenia[10, 11], involving different ethnic regions and adopting different diagnostic criteria for sarcopenia. According to AWGS 2019 Consensus, this study further discussed the relationship between DKD and Low muscle mass in the Asian T2DM population.

This retrospective study found that the prevalence of low muscle mass was positively correlated with stratified UACR, and DKD was an independent risk factor for low muscle mass in patients with T2DM. This result may be explained by chronic inflammation and protein metabolism. Inflammation is considered an essential factor in the event of low muscle mass. There is always chronic inflammation in DKD patients, which promotes the occurrence and development of low muscle mass. From the perspective of protein metabolism, protein is the primary component of muscle. DKD can cause proteinuria, which results in the loss of protein in the body and leads to malnutrition, increasing the risk of low muscle mass[12, 13]. In addition, reduced vitamin D synthesis, mitochondrial dysfunction and metabolic acidosis are thought to contribute to low muscle mass[6].

UACR is commonly used to screen for albuminuria and detect the stage of DKD due to its ease and convenience[14]. Nevertheless, UACR is not stable enough. Several conditions may elevate UACR, including marked hypertension, marked hyperglycemia, infection, etc. [14, 15]. In this study, many parameters and diseases were taken into regression statistics to exclude the confounders and get a reliable consequence.

Besides, eGFR is also an essential index for evaluating renal function. In our study, compared with the non-low muscle mass group, patients with low muscle mass had lower eGFR levels. However, multivariate logistic regression analysis showed that increased eGFR would increase the risk of muscle loss, which was different from the expected results. Yang et al. found that, regardless of whether they had T2DM, patients without sarcopenia showed higher eGFR than those with sarcopenia. Still, in logistic regression analysis, it was found that there was a correlation between ASMI (ASM/HT2) and eGFR only in the non-diabetic group but not in the diabetic group, which may be due to the effects of many factors on the renal function of diabetic patients; moreover, the study observed that patients in the higher quartiles of ASM/HT2 performed greater risk for abnormal renal function when compared to patients in the lowest quartiles in the diabetes group, which could be explained for high muscle mass associated with elevated serum creatinine level[16]. In addition, the eGFR in this study was calculated according to the MDRD equation, and it has been reported that muscle mass may affect MDRDeGFR[17], which may lead to deviation of the research results. Therefore, more studies are needed to verify the relationship between eGFR and muscle mass.

Regarding gender, men have a higher risk of muscle mass loss than women in T2DM patients; in sensitivity analysis, the relationship between DKD and muscle mass loss was significant in men but not in women, which may be due to higher fatty tissue content in women. In contrast, individuals with higher fat content may have higher protein intake and thus resist muscle loss[18]. It is also possible that the decrease of testosterone secretion in older men leads to a faster reduction of skeletal muscle rate in males than in females and is more prone to muscle loss. However, with estrogen levels declining in postmenopausal women, muscle strength will also decrease; therefore, whether male or female patients with diabetic nephropathy, active screening for sarcopenia is necessary.

Currently, the primary treatment of sarcopenia is nonpharmacologic treatment, such as exercise and enhanced nutrition. For patients with DKD, a low-protein diet is recommended to reduce urinary protein and delay disease progression. However, the measure may aggravate the negative protein balance and increase patients' long-term mortality[19]. Therefore, it's necessary to explore a new dietary strategy for patients with DKD. Moreover, DKD represents more proteinuria during the initial period than other chronic kidney diseases so that sarcopenia may appear earlier in DKD patients.

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In addition to the inherent limitations of a cross-sectional study, there are some other problems in our research. First, when selecting the population, the subjects were hospitalized in the same hospital with relatively severe conditions, resulting in selection bias. Second, in terms of DKD diagnosis in this study, one cannot accurately distinguish DKD from chronic kidney disease caused by other causes, perhaps resulting in a specific deviation in the research results. Third, the causal relationship between low muscle mass and DKD could not be determined, which requires more well-designed prospective studies to verify.

5. Conclusion

This research observed that the prevalence of low muscle mass was positively correlated with stratified UACR, and DKD was an independent risk factor of low muscle mass in patients with T2DM. Clinical researchers can attempt to combine the management of DKD to explore more appropriate prevention and treatment strategies for sarcopenia.

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