Risk Management and Healthcare Policy

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Association of the Triglyceride-Glucose Index with Body Composition and Laboratory Parameters in Chronic Kidney Disease Stages 3–5

Shan-Shan Xu, Yan-Meng Guan, Hong-Yun Xuan, Xiu-Fang Fan, Ping Lu, Li-Hai Hao

Department of Nephrology, Weifang People's Hospital, Weifang, Shandong Province, 261041, People's Republic of China

Correspondence: Li-Hai Hao, Department of Nephrology, Weifang People's Hospital, Weifang, Shandong Province, 261041, People's Republic of China, Tel +86 13583690680, Email haolihai_li@126.com; Ping Lu, Department of Nephrology, Weifang People's Hospital, Weifang, Shandong Province, 261041, People's Republic of China, Tel +86 13853636296, Email pinglulp6@126.com

Objective: This study aimed to evaluate the association of the triglyceride-glucose index (TyG index) with body composition and laboratory parameters in individuals with chronic kidney disease (CKD) stage 3–5.

Methods: A total of 89 individuals with CKD stages 3–5 were classified into two groups based on glomerular filtration rate (GFR): the CKD stages 3 to 4 group (n = 53) and the CKD stage 5 group (n = 36). Body composition parameters, including body fat mass, lean body mass, skeletal muscle mass, and body mass index, were measured. Laboratory indices, including hemoglobin, albumin, cholesterol, and the TyG index were analyzed. The correlations between the TyG index and these parameters were analyzed using Pearson correlation, and the factors of the TyG index were analyzed using linear regression.

Results: (1) Hemoglobin levels, lymphocyte counts, the TyG index, and low-density lipoprotein cholesterol concentrations were higher in patients with CKD stages 3 to 4 compared to those with CKD stage 5. (2) Measures of body composition, including body fat mass, lean body mass, skeletal muscle mass, and BMI were significantly higher in patients with CKD stages 3 to 4 compared to those with CKD stage 5. (3) The TyG index exhibited positive correlations with cholesterol, lymphocyte and monocyte counts, fasting blood glucose, triglycerides, low-density lipoprotein cholesterol, and BMI, while showing a negative correlation with serum creatinine levels. (4) Multivariate linear regression suggested that creatinine, blood glucose, GFR, triglycerides, low-density lipoprotein, and monocyte-lymphocyte ratio may be the influencing factors of TYG index.

Conclusion: TYG index was positively correlated with BMI. The TyG index, an indicator of insulin resistance, is closely linked to chronic inflammation, impaired renal function, and alterations in blood glucose and lipid profiles. These findings underscore the potential utility of the TyG index in assessing metabolic and inflammatory changes in CKD.

Keywords: bioelectrical impedance, body composition, chronic kidney disease, insulin resistance, triglyceride glucose index

Introduction

Chronic kidney disease (CKD) has a major impact on global health. The 2017 Disease Research Report shows that the number of people with CKD worldwide reached 697.5 million in 2017, accounting for 9.1% of the global population.[1] Cardiovascular disease is a prevalent complication in patients with chronic kidney disease (CKD). CKD patients have a higher risk of cardiovascular death and end-stage renal disease than non-CKD patients.[2] Insulin resistance (IR) has been recognized as a critical factor in the pathogenesis of cardiovascular disease in this population.[3] Even during the early stages of CKD, disruptions in glucose and insulin homeostasis contribute to the development of IR.[4] The triglyceride-glucose (TyG) index has been identified as a reliable biochemical surrogate marker for IR and has been linked to an increased risk of cardiovascular events in patients with CKD.[5,6] A large cross-sectional study in China found that the TyG index in CKD group was significantly higher than that in non-CKD group.[7]

As CKD advances, changes occur in the structure and function of body volume, fat, and skeletal muscle. Body composition analysis can more effectively detect these changes and monitor disease progression. IR can cause multiple

Received: 16 December 2024
Accepted: 28 February 2025
Published: 14 March 2025

metabolic disorders, such as hyperglycemia, dyslipidemia and hypertension. IR is a potential pathophysiological factor in the progression of CKD. A large study found a consistent correlation between TyG and serum creatinine and eGFR levels.[8] IR can affect the appearance of various metabolic disorders in the body, and human composition analysis can reveal changes in human composition and the progress of the disease in a more subtle manner, however, no studies have yet investigated the relationship between the TyG index and body composition in this population.

This study aims to explore the correlation between the TyG index and body composition parameters, as well as other laboratory markers, in individuals with varying stages of CKD. A recent high-value publication reported that a higher TyG index is associated with increased hospitalizations and 1-year mortality in patients with CKD.[9] This study hopes to detect and pay attention to insulin resistance in the early stage of CKD patients, and improve insulin resistance through corresponding treatment. This can delay the progression of CKD and reduce the incidence and mortality of cardiovascular disease in patients with CKD.

Participants and Methods

Study Participants

This cross-sectional study included individuals with CKD who were admitted to the Department of Nephrology at Weifang People's Hospital between February 2022 and August 2022.

Inclusion Criteria

Participants were eligible if they met the following criteria:

(1) Estimated glomerular filtration rate (eGFR) levels consistent with CKD stages 3a, 3b, 4, or 5, as defined by the Kidney Disease Improving Global Outcomes (KDIGO) guidelines: CKD 3a: eGFR 45–59 mL/ (min·1.73 m^2); CKD 3b: eGFR 30–44 mL/ (min·1.73 m^2); CKD 4: eGFR 15–29 mL/min·1.73 m^2); CKD 5: eGFR < 15 mL/ (min·1.73 m^2).

(2) Male or female participants aged 18–80 years.

(3) Patients who had not undergone hemodialysis, peritoneal dialysis, or kidney transplantation.

Exclusion Criteria

Participants were excluded if they met any of the following conditions:

(1) History of severe cardiovascular and cerebrovascular events or malignant tumors.

(2) Presence of severe hypoproteinemia (albumin < 25 g/L) or moderate to severe edema.

(3) History of severe infection, immune system diseases, or severe metabolic disorders within the three months preceding the study.

(4) Severe cognitive impairment or mental health disorders.

(5) Pregnant or lactating.

Finally, a total of 89 patients entered the study, as shown in Figure 1 for details.

This study was approved by the Ethics Committee of Weifang People's Hospital (Approval No. KYLL20200513-2).

Body Composition Data Collection

Measurement of Body Parameters

Height and body mass were measured directly to obtain baseline anthropometric data.

Bioelectrical Impedance Analysis of Human Body Composition

Bioelectrical impedance analysis is based on the principle that different body components have different water content and conductivity. A small electrical current of a certain frequency is passed through the body, and its resistance is measured to indirectly estimate the biological composition of the body. In this study, body composition was evaluated using the Inbody770 bioelectrical impedance analyzer (Korea). The procedure required all participants to fast for at least four hours prior to the test. Additionally, they were asked to empty their bladder and bowels 30 minutes before the test, wear light clothing, and avoid tight or heavy garments. During the test, participants stood barefoot on the electrodes, ensuring close contact with both foot electrodes, while their arms hung naturally at their sides to maintain proper

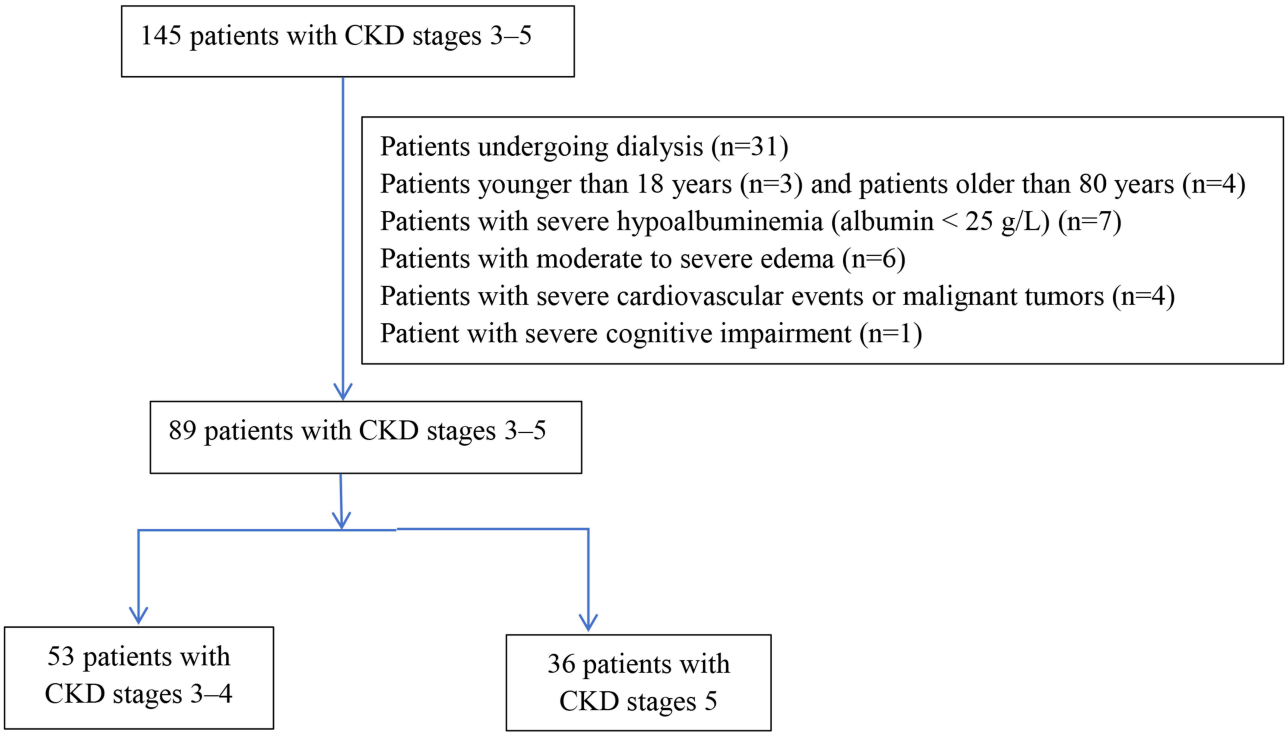

Figure 1 Study flowchart.

electrode contact. Information such as height, gender, and age was entered into the device, and the measurement was initiated by pressing the button. The instrument automatically analyzed the data and displayed the results.

The parameters measured included: total body water (TBW); intracellular water (ICW) extracellular water (ECW); body fat mass (BFM); percent body fat (PBF); visceral fat area (VFA); fat-free mass (FFM); skeletal muscle mass (SMM); soft lean mass (SLM): the sum of total body water and protein weight; body mass index (BMI): calculated as BMI = body mass (kg)/height (m).[2]

Clinical Data Collection

Demographic information, including age, gender, and medical history (eg, hypertension, diabetes mellitus, and primary disease), was collected. All patients were fasted for 8–12 hours within 48 hours after admission for fasting blood samples for laboratory analysis of hematology and biochemical parameters.

Blood routine analyses were conducted using an automatic five-part differential hematology analyzer (Mindray XN9000), while blood biochemical indicators were evaluated with an automatic biochemical analyzer (Siemens ADVIA Centaur XP). The following parameters were measured and documented: hemoglobin; albumin; monocyte and lymphocyte counts; fasting blood glucose; cholesterol and triglycerides; serum creatinine and urea nitrogen; monocyte-to-lymphocyte ratio (MLR).

The TyG index was calculated using the formula: TyG index = Ln [triglyceride (mmol/L) × fasting blood glucose (mmol/L)/2].[10]

Statistical Analysis

Statistical analyses were performed using SPSS software version 25.0. The sample size was calculated using the finite population sample size formula for cross-sectional studies. The parameters were set as follows: Type I error α (two-sided) = 0.05, allowable error ∂ = 0.1, standard deviation σ = 0.5, and a finite population size N = 600. Based on these parameters, the calculated required sample size was n = 84. The final sample size in this study was 89, which meets the required sample size and ensures statistical significance. Normally distributed measurement data are expressed as mean ±

https://doi.org/10.2147/RMHP.S511635

standard deviation ($\overline{X} \pm S$). The independent *t*-test was used to compare data between two groups. Categorical data are expressed as n (%), and the chi-squared (χ^2) test was applied to assess differences between the groups.

Pearson's correlation analysis was performed to examine the relationship between the TyG index and body composition as well as laboratory parameters. Multivariate linear regression analysis was performed to identify the factors affecting the TyG index. A *P*-value < 0.05 was considered statistically significant.

Results

Comparison of General Clinical Data Between the Two Groups

A total of 89 individuals with CKD, aged 19 to 80 years, with a mean age of (57.78 ± 14.03), were included in the study. Of these, 50 were males, and 39 were females. Participants were categorized into two groups: CKD stages 3–4 (n = 53) and CKD stage 5 (n = 36).

The primary diseases identified included diabetic nephropathy in 21 cases, hypertensive nephropathy in 20 cases, chronic glomerulonephritis in 43 cases, ischemic renal injury in 1 case, interstitial nephropathy in 1 case, and other conditions in 3 cases. No statistically significant differences were observed between the two groups in terms of gender, age, or distribution of primary diseases ($P > 0.05$), confirming the comparability of the groups. Detailed information is presented in Table 1.

Table 1 Comparison of Demographic and Laboratory Parameters Between the Two Groups (%, $\overline{x} \pm s$)

Item	CKD 3–4 Group	CKD5 Group	t/χ^2	p
Age	57.43± 14.11	58.28± 14.09	−0.277	0.782
Gender [n (%)]				
Male	34 (64.2%)	16 (44.4%)	3.382	0.066
Female	19 (35.8%)	20 (55.6%)		
Hypertension [n (%)]	32 (60.4%)	21(58.3%)	0.037	0.847
Diabetes mellitus [n (%)]	25 (47.2%)	11 (30.6%)	2.457	0.117
Serum creatinine	223.17±67.93	472.89±116.27	−12.772	<0.001
Urea nitrogen	18.09±7.34	27.10±9.34	−4.930	<0.001
GFR	28.82±10.38	10.67±2.41	10.289	<0.001
Hemoglobin	114.13±19.91	89.29±17.91	5.899	<0.001
Albumin	39.85±6.33	37.29±6.26	1.878	0.064
Lymphocyte count	1.70±0.89	1.27± 0.52	2.619	0.010
Monocyte count	0.47±0.52	0.40± 0.17	0.834	0.406
Fasting blood glucose	6.29± 2.30	5.51± 1.58	1.781	0.078
Triglyceride	2.11±1.21	1.68±0.74	1.893	0.062
Cholesterol	5.27± 1.75	4.75± 1.42	1.488	0.140
TyG index	9.08±0.64	8.76±0.47	2.541	0.013
Low-density lipoprotein cholesterol	3.20±1.20	2.71±0.99	2.032	0.045
MLR	0.28±0.16	0.37±0.27	−1.945	0.055

Notes: Values are shown as mean ± SD for continuous variables or n (%) for categorical variables.
Abbreviations: CKD, chronic kidney disease; GFR, glomerular filtration rate; TyG index, triglyceride-glucose index; MLR, monocyte lymphocyte ratio.

Comparison of Laboratory Parameters Between the Two Groups

There were no statistically significant differences in albumin levels, monocyte counts, fasting blood glucose, triglycerides, cholesterol, and MLR between the two groups ($P > 0.05$).

However, hemoglobin levels, GFR, lymphocyte counts, TyG index, and low-density lipoprotein cholesterol were significantly higher in the CKD stage 3–4 group compared to the CKD stage 5 group ($P < 0.05$). Serum creatinine and urea nitrogen levels were significantly lower in the CKD stage 3–4 group compared to the CKD stage 5 group, and the difference was statistically significant ($P < 0.05$). These findings are summarized in Table 1.

Comparison of Body Composition Analysis Between the Two Groups

Analysis using a two-sample *t*-test revealed no statistically significant differences in TBW, ICW, ECW, PBF, or SLM between individuals in the CKD stage 3–4 group and those in the CKD stage 5 group ($P > 0.05$). However, the VFA was higher in the CKD stage 3–4 group; this difference was not statistically significant ($P > 0.05$). Nevertheless, BFM, FFM, SMM, and BMI were significantly higher in the CKD stage 3–4 group compared to the CKD stage 5 group ($P < 0.05$). Detailed results are presented in Table 2.

Correlation Analysis Between the TyG Index and Other Indicators

The TyG index demonstrated positive correlations with cholesterol, lymphocyte counts, monocyte counts, blood glucose, triglycerides, low-density lipoprotein cholesterol, and BMI ($P < 0.05$). A negative correlation was observed between the TyG index and serum creatinine levels ($P < 0.05$). No statistically significant correlations were identified between the TyG index and other laboratory parameters or body composition measures. See Table 3 for detailed results.

Factors Influencing TyG Index

A multivariate linear regression analysis was conducted using the TyG index as the dependent variable. Independent variables included laboratory parameters such as age, gender, MLR, creatinine, hemoglobin, and albumin, as well as body composition parameters. The analysis identified the following significant factors influencing the TyG index: creatinine (b = −0.001, $t = -2.690$, $P = 0.009$); blood glucose (b = 0.121, $t =$

Table 2 Comparison of Body Composition Parameters Between the Two Groups ($\bar{x} \pm s$)

Item	CKD 3–4 Group	CKD 5 Group	t/χ^2	p
TBW	37.35±8.17	37.88±6.17	−0.326	0.745
ICW	22.56±4.94	22.89±3.89	−0.344	0.731
ECW	14.76±2.91	15.04±2.63	−0.454	0.651
PBF	24.92±8.70	22.53±5.44	1.467	0.146
VFA	80.00±34.38	68.34±16.98	1.883	0.063
BFM	14.77± 7.21	12.07±3.71	2.070	0.041
SLM	50.41± 9.30	49.06± 8.14	0.708	0.481
FFM	53.70±8.57	48.75±6.53	2.931	0.004
SMM	31.31±6.57	24.79± 5.23	4.976	<0.001
BMI	24.45± 3.51	22.57± 2.88	2.661	0.009

Note: Values are shown as mean ± SD.

Abbreviations: CKD, chronic kidney disease; TBW, Total Body Water; ICW, Intracellular Water; ECW, Extracellular Water; PBF, Percent Body Fat; VFA, Visceral Fat Area; BFM, Body Fat Mass; SLM, Soft Lean Mass; FFM, Fat Free Mass; SMM, Skeletal Muscle Mass; BMI, Body Mass Index.

https://doi.org/10.2147/RMHP.S511635

Table 3 Correlation Analysis of the TyG Index With Laboratory and Body Composition Parameters

TyG Index	Creatinine	Cholesterol	Lymphocyte Count	Monocyte Count	Blood Glucose	Triglyceride	Low-density Lipoprotein Cholesterol	BMI
r	−0.22	0.407	0.318	0.277	0.5	0.324	0.41	0.296
p	0.038	<0.001	0.002	0.008	<0.001	<0.001	<0.001	0.005

Abbreviations: TyG index, Triglyceride glucose index; BMI, body mass index.

Table 4 Factors Influencing the TyG Index Based on Linear Regression Analysis

Variable	b value	b-value Standard Error	t value	*P* value
Creatinine	0.00	0.00	−2.690	0.009
Lymphocyte count	0.14	0.06	2.189	0.033
Fasting blood glucose	0.12	0.01	9.370	<0.001
Triglyceride	0.39	0.03	15.185	<0.001
Low-density lipoprotein cholesterol	0.13	0.05	2.720	0.009
MLR	0.41	0.18	2.227	0.030
GFR	−0.01	0.01	−2.230	0.030

Abbreviations: TyG index, Triglyceride glucose index; MLR, monocyte lymphocyte ratio; GFR, glomerular filtration rate.

9.370, $P < 0.001$); GFR (b = − 0.010, $t = -2.230$, $P = 0.030$); triglycerides (b = 0.393, $t = 15.185$, $P < 0.001$); low-density lipoprotein cholesterol (b = 0.129, $t = 2.720$, $P = 0.009$); and MLR (b = 0.406, $t = 2.227$, $P = 0.030$).

All these factors were statistically significant ($P < 0.05$). Detailed regression outcomes are summarized in Table 4.

Discussion

The global incidence of chronic kidney disease is currently high. Cardiovascular disease is a prevalent complication in individuals with CKD. IR has been identified as an important factor in the pathogenesis of cardiovascular disease in this population.[3] The Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) is a widely utilized method for evaluating pancreatic β-cell function and IR. However, its applicability is limited in patients receiving insulin therapy or those with non-functioning β-cells.[11] In contrast, the TyG index, a non-insulin-based measure, offers broader applicability across individuals, regardless of their insulin treatment status.[12] The TyG index has demonstrated greater utility compared to HOMA-IR in evaluating IR in both diabetic and non-diabetic populations.[13] It serves as a reliable surrogate biochemical marker for IR.[5] Xiao et al's study showed that an increase in the TyG index was associated with an increase in the prevalence of CKD.[14] A meta-analysis showed that an increase in TyG index levels was positively correlated with an increase in the relative risk of coronary heart disease.[15] Paying attention to IR and improving IR in a timely manner will have certain benefits for controlling the progression of CKD and reducing the incidence of cardiovascular disease.

This study demonstrated variations in the TyG index across different stages of CKD, indicating changes in IR levels associated with CKD progression. These findings align with the observations of Rabbani et al, who reported alterations in IR and blood lipid levels with advancing CKD.[4] The current study revealed a significantly higher TyG index in patients with CKD stages 3–4 compared to those with CKD stage 5. Similarly, Yildirim et al reported an increase in the TyG index with greater proteinuria, particularly among patients with a urinary albumin-to-creatinine ratio exceeding

300 mg/g.[16] This correlation may explain the higher TyG index observed in CKD stages 3–4, as patients at these stages often exhibit elevated urinary albumin-to-creatinine ratios compared to those at stage 5.

A prospective multicenter cohort study by Quiroga et al found that the TyG index was significantly higher in patients with CKD stages 3–5 compared to normal controls, and the TyG index was independently associated with the occurrence of serious cardiovascular events in non-diabetic individuals with CKD.[6] Although the TyG index was lower in CKD stage 5 compared to CKD stages 3–4, its median value in CKD stage 5 may still exceed that of the general population. A large cross-sectional study found that the TyG index in the CKD group was significantly higher than that in the non-CKD group.[7] Other studies have found that TyG variability can be a useful tool for identifying individuals at risk of CKD progression.[17] However, the absence of a control group in the present study precluded a direct comparison of the TyG index between patients with CKD stages 3–5 and the general population.

Bioimpedance analysis facilitates the real-time evaluation of human body composition, including TBW, muscle mass, fat content, and nutritional status. This method can more sensitively detect changes in various components and indicators. In the present study, no significant differences were observed in TBW, ICW, ECW, PBF, or SLM between groups. However, BFM, FFM, SMM, and BMI were significantly higher in individuals with CKD stages 3–4 compared to those with CKD stage 5.

Previous research has demonstrated that male patients with CKD stages 3–5 have significantly higher SMM compared to female patients. Furthermore, both male and female patients in CKD stages 3–4 have higher SMM compared to those in CKD stage 5. This study also revealed a positive correlation between SMM and GFR, indicating that SMM progressively decreases as GFR declines.[18] While sex-specific analyses were not conducted in the current study, these findings align with existing literature. Skeletal muscle is the primary site for glucose uptake and utilization, responsible for approximately 80% of insulin-stimulated glucose uptake, making it the body's largest insulin target organ.[19] Therefore, skeletal muscle is among the earliest and most critical sites affected by IR. Thus, SMM decreases progressively with the development of IR in CKD. Recent studies have shown that reduced SMM may be linked to IR and could contribute to the development of diabetes mellitus and metabolic syndrome.[20,21] Other studies have found that in different adjustment models, TyG index remains independently and positively correlated with muscular atrophy.[8] Furthermore, the relationship between SMM reduction and IR may be bidirectional. The mechanisms underlying the reduction in SMM associated with IR are multifaceted. Insulin stimulates glucose uptake mainly in skeletal muscle.[22] A decline in skeletal muscle mass reduces glucose uptake, thereby impairing insulin sensitivity and promoting the development of IR. Additionally, skeletal muscle releases various myokines, including irisin, which is produced during exercise. Irisin can activate the peroxisome proliferator-activated receptor α (PPARα) signaling pathway, essential for fatty acid β-oxidation in the liver. This process aids in improving hepatic steatosis and enhancing insulin sensitivity.

A decrease in skeletal muscle mass leads to reduced exercise capacity and lower energy expenditure, contributing to obesity. This cycle can further exacerbate IR and hepatic steatosis. The current study observed that the VFA in CKD stages 3–4 was significantly higher than in stage 5, although the difference was not statistically significant. Visceral adipose tissue, primarily located in the mesentery and omentum, has a higher concentration of inflammatory and immune cells, along with an increased number of glucocorticoid and androgen receptors. Moreover, visceral adipocytes are metabolically more active, more sensitive to lipolysis, and resistant to insulin compared to subcutaneous adipocytes.

A cross-sectional survey conducted by Renee de et al in obese individuals in the Netherlands demonstrated an association between VFA and IR.[23] This finding aligns with the observation in this study that VFA was significantly higher in CKD stages 3–4 compared to stage 5. However, the absence of statistical significance in the results may be due to the limited sample size. Additionally, correlation analysis showed no significant relationship between the TyG index and other body components, such as SMM and VFA. This lack of correlation may suggest a nonlinear relationship or could be influenced by factors such as the limited sample size, history of hyperlipidemia, and medication use.

This study identified significantly lower FFM, BFM, and BMI in patients with CKD stage 5 compared to those in CKD stages 3–4. Additionally, a previous study suggested that FFM is an independent risk factor influencing the survival prognosis of non-dialysis patients with CKD stages 4–5. Furthermore, FFM serves as a prognostic indicator for both non-dialysis and dialysis patients with CKD.[24] As CKD progresses, there is a gradual decline in both muscle structure and function. The pathological mechanisms contributing to the loss of lean body mass may be linked to the disease process of

CKD, along with the effects of aging. Additionally, factors such as reduced appetite, impaired nutrient absorption, and decreased muscle synthesis, which are common in advanced CKD, may contribute to declines in BFM and BMI.

This study found that except for a positive correlation between TYG index and BMI, there was no correlation between TYG index and other human composition indicators. Some studies will divide participants into four groups based on the TyG index quartiles. It is found that people with higher TyG index values tend to belong to the obese group with higher BMI and waist circumference, and the proportion of individuals with dyslipidemia in the high TyG index group is significantly higher.[8] This is consistent with the results of this study. Other studies have found that TyG index, mean arterial pressure and uric acid mediate the relationship between BMI and ESKD in middle-aged people.[25,26] It also emphasized that in addition to weight loss, controlling metabolic risk factors may be crucial to reducing the adverse effects of BMI on renal function.[27]

This study also found that creatinine, GFR, blood sugar, triglycerides, low-density lipoprotein cholesterol, and MLR are influencing factors on the TyG index. TyG index was positively correlated with cholesterol, lymphocytes, monocytes, blood sugar, triglycerides, and low-density lipoprotein, and negatively correlated with creatinine. First, studies have found that TyG variability can be a useful tool for identifying individuals at risk of developing CKD, elevated changes in the TYG index can lead to the progression of CKD.[17] In the kidney, multiple insulin-sensitive cell types express insulin receptors, and specific knockdown of these receptors in podocytes or proximal ducts leads to proteinuria, renal pathology, and hyperglycemia.[28–30] Other studies have found that TyG index and TYG-related indicators are significantly positively correlated with blood creatinine and significantly negatively correlated with eGFR, and suggest that these indicators have potential application value in early screening and management of chronic kidney disease risk.[8]

In addition, this study found that blood sugar, triglycerides, and low-density lipoprotein cholesterol are also influential factors in the TYG index. In patients with severe CKD, hypertriglyceridemia and reduced HDL cholesterol levels are prevalent lipid abnormalities, while LDL cholesterol levels often remain within normal ranges. Notably, LDL cholesterol is clearly associated with major atherosclerotic events in the general population. LDL cholesterol is inversely linked to adverse outcomes when levels are below average in patients with CKD. Conversely, elevated LDL cholesterol levels show a slight positive association with mortality.[31] Dysregulation of lipid metabolism in CKD is complex, emphasizing the importance of lipid management in improving prognosis.[16] Lipid levels may fluctuate as the disease progresses or in response to management strategies.[32] Notably, reducing LDL cholesterol levels in patients not on dialysis has been shown to lower the incidence of major atherosclerotic events.[31,32] Therefore, adjusting lipid metabolism and maintaining LDL levels within a reasonable range may improve IR and reduce the risk of atherosclerosis in patients with CKD.

This study also identified correlations between the TyG index and lymphocyte and monocyte levels, with MLR emerging as an influencing factor for the TyG index. Patients with CKD, especially those with end-stage renal disease, often exhibit chronic inflammatory responses marked by changes in C-reactive protein, tumor necrosis factor, and leukocyte subsets. This chronic inflammation is a well-established risk factor for cardiovascular disease.

Recent studies have shown that leukocytes and their subsets are associated with the progression of renal disease. Increased monocytosis and monocyte activation are associated with severe complications in CKD. Furthermore, an elevated MLR has been identified as a strong and independent predictor of all-cause and cardiovascular mortality in patients who are dependent on dialysis.[33] Furthermore, Kanbay et al found that the proportion of monocytes to HDL increased as GFR decreased and was associated with cardiovascular events.[34] Correlation analysis from studies shows that all TyG-related indicators are related to obesity, blood pressure, lipids, inflammation and renal function.[7] These findings suggest that in CKD, chronic inflammatory conditions, IR, and lipid metabolism are interconnected and can contribute independently or collectively to the development of cardiovascular disease.

Limitations

This study has several limitations that should be acknowledged.

1. It is a single-center, cross-sectional study that lacks a control group from the general population, necessitating confirmation through larger, prospective studies.

2. The sample size is limited, and no further stratification was conducted based on age or gender among individuals with chronic kidney disease. Additionally, the study population was limited to hospitalized patients, which may introduce selection bias.
3. Factors such as medication dosage and patient compliance were not considered, potentially influencing the findings.
4. Other markers of inflammation or IR were not measured in this study, which could provide further insights.

Conclusion

In conclusion, TyG index was positively correlated with BMI, but no significant correlation was found with other human components. Significant reductions in skeletal muscle mass and lean body mass are observed as CKD advances. IR may be associated with chronic inflammation and declining renal function, in addition to conventional factors such as elevated blood glucose and blood lipids. Future research should focus on early detection of insulin resistance, and through corresponding treatment to improve insulin resistance, protect skeletal muscles, further delay the progression of CKD and reduce the incidence of cardiovascular disease in patients with CKD. Additionally, large-scale longitudinal studies are needed to confirm the benefits of early insulin resistance management on long-term outcomes in CKD patients, particularly in preventing muscle wasting and cardiovascular complications.

Abbreviations

CKD, Chronic kidney disease; GFR, Glomerular filtration rate; TyG index, Triglyceride Glucose Index; IR, Insulin resistance; TBW, Total Body Water; ICW, Intracellular Water; ECW, Extracellular Water; BFM, Body Fat Mass; PBF, Percent Body Fat; VFA, Visceral Fat Area; FFM, Fat Free Mass; SMM, Skeletal Muscle Mass; SLM, Soft Lean Mass; BMI, Body Mass Index; MLR, Monocyte Lymphocyte ratio; LDL, Low-density lipoprotein.

Data Sharing Statement

All data generated or analysed during this study are included in this article. Further enquiries can be directed to the corresponding author (Li-Hai Hao: E-mail: haolihai_li@126.com).

Ethics Approval and Consent to Participate

The study was conducted in accordance with the Declaration of Helsinki (as was revised in 2013). The study was approved by Ethics Committee of the Weifang People's Hospital (No.KYLL20200513-2). Written informed consent was obtained from all participants.

Funding

1. Weifang Health and Family Planning Commission Research Project, No. WFWSJK-2020-326. 2. Weifang Health and Family Planning Commission Research Project, No. WFWSJK-2024-065.

Disclosure

The authors declare that they have no competing interests.

References

1. Nelson RG, Grams ME, Ballew SH, et al. Development of risk prediction equations for incident chronic kidney disease. *JAMA*. 2019;322 (21):2104–2114. doi:10.1001/jama.2019.17379
2. Zhang L, Wang F, Wang L, et al. Prevalence of chronic kidney disease in China: a cross-sectional survey. *Lancet*. 2012;379(9818):815–822. doi:10.1016/S0140-6736(12)60033-6
3. Zhou M, Wang H, Zeng X, et al. Mortality, morbidity, and risk factors in China and its provinces, 1990-2017: a systematic analysis for the global burden of disease study 2017. *Lancet*. 2019;394(10204):1145–1158. doi:10.1016/S0140-6736(19)30427-1
4. Rabbani N, Thornalley PJ. Advanced glycation end products in the pathogenesis of chronic kidney disease. *Kidney Int*. 2018;93(4):803–813. doi:10.1016/j.kint.2017.11.034
5. Sánchez-García A, Rodríguez-Gutiérrez R, Mancillas-Adame L, et al. Diagnostic accuracy of the triglyceride and glucose index for insulin resistance: a systematic review. *Int J Endocrinol*. 2020;2020:4678526. doi:10.1155/2020/4678526

6. Quiroga B, Muñoz Ramos P, Sánchez Horrillo A, Ortiz A, Valdivieso JM, Carrero JJ. Triglycerides-glucose index and the risk of cardiovascular events in persons with non-diabetic chronic kidney disease. *Clin Kidney J*. 2022;15(9):1705–1712. doi:10.1093/ckj/sfac073
7. Chen T, Liu Y, Wu S, et al. Comparison of TyG and newly TyG related indicators for chronic kidney diseases estimation in a Chinese population. *Diabetes Metab Syndr Obes*. 2024;17:3063–3075. doi:10.2147/DMSO.S469260
8. Li L, Xu Z, Jiang L, et al. Triglyceride-glucose index and its correlates: associations with serum creatinine and estimated glomerular filtration rate in a cross-sectional study from CHARLS 2011-2015. *Metab Syndr Relat Disord*. 2024;22(3):179–189. doi:10.1089/met.2023.0188
9. Chen Y, Li S, Yang K, et al. Triglyceride-glucose index and prognosis in individuals afflicted with heart failure and chronic kidney disease. *ESC Heart Fail*. 2024;11(5):3120–3132. doi:10.1002/ehf2.14898
10. Wang L, Cong HL, Zhang JX, et al. Triglyceride-glucose index predicts adverse cardiovascular events in patients with diabetes and acute coronary syndrome. *Cardiovasc Diabetol*. 2020;19(1):80. doi:10.1186/s12933-020-01054-z
11. Minh HV, Tien HA, Sinh CT, et al. Assessment of preferred methods to measure insulin resistance in Asian patients with hypertension. *J Clin Hypertens*. 2021;23(3):529–537. doi:10.1111/jch.14155
12. Placzkowska S, Pawlik-Sobecka L, Kokot I, Piwowar A. Indirect insulin resistance detection: current clinical trends and laboratory limitations. *Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub*. 2019;163(3):187–199. doi:10.5507/bp.2019.021
13. Simental-Mendía LE, Rodríguez-Morán M, Guerrero-Romero F. The product of fasting glucose and triglycerides as surrogate for identifying insulin resistance in apparently healthy subjects. *Metab Syndr Relat Disord*. 2008;6(4):299–304. doi:10.1089/met.2008.0034
14. Ren X, Jiang M, Han L, Zheng X. Association between triglyceride-glucose index and chronic kidney disease: a cohort study and meta-analysis. *Nutr, Metab Cardiovasc Dis*. 2023;33(6):1121–1128. doi:10.1016/j.numecd.2023.03.026
15. Liang S, Wang C, Zhang J, et al. Triglyceride-glucose index and coronary artery disease: a systematic review and meta-analysis of risk, severity, and prognosis. *Cardiovasc Diabetol*. 2023;22(1):170. doi:10.1186/s12933-023-01906-4
16. Yildirim F, Yildiz AB, Kanbay M. A promising tool: triglyceride-glucose index to stratify the risk of cardiovascular events in chronic kidney disease. *Clin Kidney J*. 2022;15(9):1653–1656. doi:10.1093/ckj/sfac084
17. Yu C, Shi Y, Wang T, et al. Triglyceride-glucose index change and chronic kidney disease progression in a Chinese hypertensive population. *Front Endocrinol*. 2024;15:1342408. doi:10.3389/fendo.2024.1342408
18. Sun J, Wang J, Liu H, et al. Bioelectrical impedance analysis of body composition in non-dialysis chronic kidney disease patients. *Chin J Integr Med Nephrol*. 2024;25(2):164–166.
19. Richter EA, Hargreaves M. Exercise, GLUT4, and skeletal muscle glucose uptake. *Physiol Rev*. 2013;93(3):993–1017. doi:10.1152/physrev.00038.2012
20. Al-Ozairi E, Alsaeed D, Alroudhan D, et al. Skeletal muscle and metabolic health: how do we increase muscle mass and function in people with type 2 diabetes. *J Clin Endocrinol Metab*. 2021;106(2):309–317. doi:10.1210/clinem/dgaa835
21. Nomura K, Eto M, Ogawa S, et al. Association between low muscle mass and metabolic syndrome in elderly Japanese women. *PLoS One*. 2020;15(12):e0243242. doi:10.1371/journal.pone.0243242
22. Bhanji RA, Narayanan P, Allen AM, Malhi H, watt KD. Sarcopenia in hiding: the risk and consequence of underestimating muscle dysfunction in nonalcoholic steatohepatitis. *Hepatology*. 2017;66(6):2055–2065. doi:10.1002/hep.29420
23. de Mutsert R, Gast K, Widya R, et al. Associations of abdominal subcutaneous and visceral fat with insulin resistance and secretion differ between men and women: the Netherlands epidemiology of obesity study. *Metab Syndr Relat Disord*. 2018;16(1):54–63. doi:10.1089/met.2017.0128
24. Vega A, Abad S, Macías N, et al. Low lean tissue mass is an independent risk factor for mortality in patients with stages 4 and 5 non-dialysis chronic kidney disease. *Clin Kidney J*. 2017;10(2):170–175. doi:10.1093/ckj/sfw126
25. Tarabeih M, Qaddumi J, Hamdan Z, Bahar A, Sawalmeh O. Worsening of diabetes control measures and decreased kidney function in pre-diabetic kidney donors compared to non-diabetic donors whose BMI before kidney donation was above 30. *Transplant Proc*. 2024;56(6):1332–1340. doi:10.1016/j.transproceed.2024.05.038
26. Tarabeih M, W N. Comparison between female kidney donors with prediabetes and without diabetes in blood pressure measurements, kidney and diabetes biomarkers: a prospective cohort study. *J Nephrol*. 2024. doi:10.1007/s40620-024-02168-3
27. Fritz J, Brozek W, Concin H, et al. The association of excess body weight with risk of ESKD is mediated through insulin resistance, hypertension, and hyperuricemia. *J Am Soc Nephrol*. 2022;33(7):1377–1389. doi:10.1681/ASN.2021091263
28. Tarabeih M, Qaddumi J, Hamdan Z, et al. Increasing overnight fluid intake and kidney function during ramadan fasting: a randomized controlled trial. *Transplant Proc*. 2023;55(1):80–86. doi:10.1016/j.transproceed.2022.10.059
29. Hinden L, Kogot-Levin A, Tam J, Leibowitz G. Pathogenesis of diabesity-induced kidney disease: role of kidney nutrient sensing. *FEBS J*. 2022;289(4):901–921. doi:10.1111/febs.15790
30. Tiwari S, Singh RS, Li L, et al. Deletion of the insulin receptor in the proximal tubule promotes hyperglycemia. *J Am Soc Nephrol*. 2013;24(8):1209–1214. doi:10.1681/ASN.2012060628
31. Ferro CJ, Mark PB, Kanbay M, et al. Lipid management in patients with chronic kidney disease. *Nat Rev Nephrol*. 2018;14(12):727–749. doi:10.1038/s41581-018-0072-9
32. Suh SH, Kim SW. Dyslipidemia in patients with chronic kidney disease: an updated overview. *Diabetes Metab J*. 2023;47(5):612–629. doi:10.4093/dmj.2023.0067
33. Xiang F, Chen R, Cao X, et al. Monocyte/lymphocyte ratio as a better predictor of cardiovascular and all-cause mortality in hemodialysis patients: a prospective cohort study. *Hemodial Int*. 2018;22(1):82–92. doi:10.1111/hdi.12549
34. Kanbay M, Solak Y, Unal HU, et al. Monocyte count/HDL cholesterol ratio and cardiovascular events in patients with chronic kidney disease. *Int Urol Nephrol*. 2014;46(8):1619–1625. doi:10.1007/s11255-014-0730-1