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Correlation between visceral fat area and diabetic kidney disease in type 2 diabetes mellitus patients with normal body mass index

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Abstract: Objective To investigate the correlation between visceral fat area (VFA) and diabetic kidney disease (DKD) in type 2 diabetes mellitus (T2DM) patients with normal body mass index (BMI) . **Methods** A cross-sectional study was conducted on 309 patients with normal BMI and T2DM in the Department of Endocrinology of the Second Affiliated Hospital of Zhejiang University School of Medicine from August 18, 2022 to April 8, 2024. The patients were divided into DKD group (n=90) and non-DKD group (n=219) according to whether DKD was merged or not. Baseline data, laboratory indicators between the two groups were compared. Spearman correlation analysis, logistic regression analysis, and the drawing of receiver operating characteristic (ROC) curves were used to study the correlation. **Results** VFA in DKD group was higher significantly than that in the non-DKD group[(70.16±25.79) cm^2 *vs* (61.93±22.17) cm^2, t=2.823, P=0.005]. The results of Spearman correlation analysis showed that DKD was positively correlated with age (r=0.218, P< 0.01), disease duration (r=0.202, P<0.01), VFA (r=0.157, P=0.006), triglycerides (r=0.170, P=0.003), serum creatinine (r=0.499, P<0.01) , serum uric acid (r=0.318, P<0.01) , and urine microalbumin (r=0.532, P<0.01). The results of logistic regression analysis showed that VFA was a risk factor for DKD (OR=1.015, 95%CI: 1.004-1.025, P=0.006) . After adjusting for confounding factors such as gender, BMI, and disease duration, VFA remained a risk factor for DKD (OR=1.024, 95%CI: 1.008-1.040, P=0.003) . The ROC curve analysis results showed that the cut-off value of VFA for predicting DKD was 71.05 cm^2,the sensitivity was 0.467,the specificity was 0.726, and the area under the curve (AUC) was 0.600 (95%CI: 0.529-0.670). **Conclusion** In T2DM patients with normal BMI, VFA is positively correlated with DKD, and VFA is a risk factor for DKD. The risk of DKD increases with the increase of VFA.

Keywords: Type 2 diabetes; Diabetic kidney disease; Visceral fat area; Body mass index

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Diabetic kidney disease (DKD) has now become the leading cause of end-stage renal disease (ESRD) globally. Among patients with type 2 diabetes mellitus (T2DM) over a 10-year disease course, approximately 40% will develop DKD [1]. It is estimated that nearly 39 million T2DM patients in China have chronic kidney disease (about one-third), a significant number [2]. Visceral fat impacts insulin resistance and metabolic disorders [3], which, in turn, lead to systemic inflammation, oxidative stress, and the activation of the renin-angiotensin-aldosterone system, resulting in kidney damage [4-5]. Additionally, the lipotoxicity of visceral fat can affect the kidneys, leading to increased proteinuria, renal fibrosis, and dysfunction [6]. In clinical practice, patients with DKD have both obese and normal body types, but there is limited research on whether visceral fat in patients with normal body types is related to DKD. If a relationship between visceral fat area (VFA) and DKD can be identified in T2DM patients with a normal body mass index (BMI), it may provide valuable insights for the prevention and management of DKD in BMI-normal T2DM patients.

1 Subjects and Methods

1.1 Research Subjects

This study was a retrospective cross-sectional analysis, collecting clinical data from 309 T2DM patients with normal BMI (18.5-<24.0 kg/m²) who visited the Department of Endocrinology at the Second Affiliated Hospital of Zhejiang University School of Medicine from August 18, 2022, to April 8, 2024. The study was approved by the Ethics Committee of the Second Affiliated Hospital of Zhejiang University School of Medicine [Ethical Approval No.: 〔2025〕 Lunshen Yandi (0430)]. All the patients signed informed consent forms.

Inclusion Criteria:

(1) A clear diagnosis of T2DM;

(2) BMI between 18.5 kg/m² and <24 kg/m²;

(3) Complete clinical data, including VFA and other relevant information, during hospitalization.

Exclusion Criteria:

(1) Type 1 diabetes mellitus, other special types of diabetes, or gestational diabetes;

(2) Acute diabetic complications, such as diabetic ketoacidosis, hyperglycemic hyperosmolar state, or hypoglycemia;

(3) Severe infection;

(4) Complicated with malignancies;

(5) Other endocrine diseases affecting blood sugar, such as acromegaly, Cushing syndrome, or thyroid disorders;

(6) Severe cardiopulmonary dysfunction;

(7) Underwent dialysis treatment for DKD;
(8) Complicated with other kidney diseases (such as lupus nephritis).

1.2 Disease Diagnosis and Definition

(1) Diagnosis of T2DM was referred to the *Chinese Type 2 Diabetes Prevention and Treatment Guidelines (2017 Edition)*[7]: typical diabetes symptoms (such as excessive thirst, frequent urination, excessive hunger, and unexplained weight loss) combined with one of the following results: random blood glucose ≥ 11.1 mmol/L, fasting blood glucose (FBG) ≥ 7.0 mmol/L, or 2-hour oral glucose tolerance test blood glucose ≥ 11.1 mmol/L. If typical diabetes symptoms are absent, the above results should be rechecked on another day for confirmation. Diagnosis is further confirmed and classified into T2DM based on factors like the patient's age of onset, family history, the rapidity of onset, symptoms, C-peptide levels, and diabetes-related antibodies. (2)Diagnosis of DKD was referred to the *Chinese Type 2 Diabetes Prevention and Treatment Guidelines (2017 Edition)* [7]: A history of T2DM with exclusion of other potential causes of kidney injury, estimated glomerular filtration rate (eGFR) <60 mL/(min·1.73 m²), urine protein/creatinine ratio ≥ 30 mg/g lasting for more than 3 months, and diabetic retinopathy can assist in diagnosing DKD.

1.3 Research Indicators

1.3.1 Baseline Data

Age, gender, weight, height, BMI, VFA, smoking history, alcohol consumption history, duration of diabetes, history of DKD, history of metformin use, history of insulin use, history of sodium-dependent glucose transporter 2 inhibitor (SGLT-2i) use, and history of glucagon-like peptide-1 receptor agonist (GLP-1RA) use.

1.3.2 Laboratory Indicators

FBG, fasting C-peptide (FCP), glycated hemoglobin (HbA_{1C}), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), serum creatinine (SCr), eGFR, UACR, serum uric acid (SUA), alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), and alkaline phosphatase (ALP).

1.4 Statistical Analysis

Data were organized using Excel and analyzed using SPSS 25.0 software. Normally distributed continuous variables were expressed as $\bar{x} \pm s$, and comparisons were made using the t-test. Non-normally distributed continuous variables were expressed as $M(P_{25},P_{75})$, and comparisons were made using the rank sum test. Categorical data were expressed as n (%), and comparisons were made using the Chi-square test. Spearman's correlation coefficient was used to analyze the relationship between various factors and DKD. Logistic regression models were used to analyze the relationship between VFA and DKD. Receiver operating characteristic (ROC) curves were used to assess the diagnostic value of VFA for DKD. A P-value of <0.05 was considered statistically significant for all tests.

2 Results

2.1 Comparison of Baseline Data Between Two Groups

There was no statistically significant difference between the two groups in terms of gender, BMI, insulin use, smoking history, and alcohol consumption (P>0.05). However, there were statistically significant differences in age, disease duration, VFA, metformin use, SGLT2i use, and GLP-1RA use (P<0.01). See **Table 1**.

2.2 Comparison of Laboratory Indicators Between Two Groups

There were significant differences between the two groups in terms of FCP, TG, SUA, and eGFR (P<0.05). See **Table 2**.

Tab.1 Comparison of baseline data between two groups

Indicators	DKD Group (n=90)	Non-DKD group(n=219)	$\chi^2/t/Z$ value	P value
Gender[a]				
Male	61(67.78)	132(60.27)	1.532	0.216
Female	29(32.22)	87(39.73)		
Age(year, $\bar{x} \pm s$)	65.4±11.56	59.0±13.29	4.001	0.001
BMI (kg/m²) [b]	21.3(19.9,22.6)	20.9(19.6,22.3)	1.648	0.102
VFA (cm², $\bar{x} \pm s$)	70.16±25.79	61.93±22.17	2.823	0.005
Medication[b]				
Metformin	42(46.67)	134(61.19)	5.486	0.019
Insulin	66(73.33)	142(64.84)	2.091	0.148
GGLT2i	50(55.56)	82(37.44)	8.552	0.003
GLP1 GA	12(13.33)	11(5.02)	6.394	0.011
Duration of diabetes (year , $\bar{x} \pm s$)	13.0±8.55	9.4±7.82	3.268	0.001
Smoking history[a]	30(33.33)	67(30.59)	0.157	0.692
Drinking history[a]	16(17.78)	54(24.66)	1.878	0.171

Note: [a] meant the data was represented by the form of [case(%)]; [b] meant the data was represented by the form of $M(P_{25},P_{75})$.

Tab.2 Comparison of laboratory indicators between two groups of patients ($\bar{x} \pm s$)

Indicators	DKD group (n=90)	Non-DKD group (n=219)	t/Z value	P value
HbA_{1C}(%)	9.70±2.72	9.28±2.35	1.371	0.171
FCP(nmol/L) [a]	0.44(0.20,0.65)	0.33(0.21,0.52)	1.957	0.050
FBG(mmol/L)	8.26±3.86	8.24±3.32	0.033	0.973
ALT(u/L) [a]	17.00(13.00,26.25)	17.00(13.00,26.00)	0.102	0.919
AST(u/L) [a]	20.00(16.75,26.00)	19.00(16.00,24.00)	1.247	0.213
GGT(u/L) [a]	20.50(14.75,34.25)	19.00(14.00,26.00)	1.474	0.140
ALP(u/L) [a]	77.00(64.75,92.00)	75.00(62.00,94.00)	0.409	0.683
TG(mmol/L) [a]	1.36(0.97,1.89)	1.17(0.80,1.59)	2.991	0.003
LDL-C (mmol/L)	2.39±1.00	2.34±0.92	0.469	0.639
HDL-C (mmol/L)	1.29±0.39	1.33±0.41	0.725	0.469
SCr (μmol/L) [a]	90.05(70.08,125.95)	61.30(54.45,71.30)	8.764	<0.001
eGFR	68.06±24.72	99.08±15.04	13.479	<0.001
SUA (μmol/L)	363.24±104.41	295.01±82.50	6.095	<0.001
UACR(mg/g) [a]	117.44 (39.57,681.49)	12.50 (12.50,24.74)	9.305	<0.001

Note: [a] meant the data was represented by the form of $M(P_{25},P_{75})$. The eGFR was measured by mL/(min·1.73 m²).

2.3 Correlation Analysis Between DKD and Indicators

Spearman correlation analysis showed that DKD was positively correlated with age (r=0.218, P<0.01), duration of diabetes (r=0.202, P<0.01), VFA (r=0.157, P=0.006), TG (r=0.170, P=0.003), SCr (r=0.499, P<0.01), SUA (r=0.318, P<0.01), and UACR (r=0.532, P<0.01). DKD was negatively correlated with eGFR (r=-0.550, P<0.01).

2.4 Logistic Regression Analysis of DKD

A univariate logistic regression analysis with DKD as the dependent variable and various indicators as independent variables showed that age, duration of diabetes, VFA, FCP, SCr, eGFR, SUA, and the use of metformin, SGLT2i, and GLP-1RA were all risk factors for DKD. See **Table 3.**

Tab.3 Results of univariate logistic regression analysis

Indicators	*β* value	SE	*OR* value	95%*CI*	Wald value	*P* value
Female	0.327	0.264	1.386	0.826-2.368	1.526	0.217
Age	0.042	0.011	1.043	1.021-1.066	14.552	0.001
BMI	0.130	0.079	1.139	0.975-1.331	2.691	0.101
Duration of diabetes	0.054	0.015	1.055	1.024-1.088	12.103	0.001
VFA	0.014	0.005	1.015	1.004-1.025	7.506	0.006
HbA_{1C}	0.069	0.050	1.071	0.971-1.182	1.869	0.172
FCP	1.329	0.409	3.778	1.695-8.423	10.558	0.001
FBG	0.001	0.036	1.001	0.933-1.074	0.001	0.973
SCr	0.064	0.008	1.066	1.048-1.084	56.490	0.001
eGFR	-0.081	0.010	0.922	0.905-0.940	68.545	0.001
SUA	0.008	0.002	1.008	1.005-1.011	27.935	0.001
UACR	0.013	0.003	1.013	1.008-1.018	26.190	0.001
Metformin	0.589	0.253	1.802	1.098-2.957	5.426	0.020
Insulin	0.384	0.278	1.469	0.852-2.531	1.916	0.166
SGLT2i	-.0736	0.254	0.479	0.291-0.788	8.408	0.004
GLP1RA	-1.068	0.438	0.344	0.146-0.811	5.943	0.015

In the model without adjusting for confounding factors, high VFA was identified as a risk factor for DKD (*OR*=1.015, 95%*CI:* 1.004–1.025, *P*=0.006). In Model 1, which adjusted for gender and BMI, the significance of high VFA as a risk factor for DKD remained, with an increased *OR* value compared to the unadjusted model (*OR*=1.023, 95%*CI:* 1.007–1.038, *P*=0.004). In Model 2, which further adjusted for duration of diabetes, high VFA remained a significant risk factor for DKD, with a higher *OR* value compared to the previous model (*OR*=1.024, 95%*CI:* 1.008–1.040, *P*=0.003). In Model 3, after adjusting for additional factors such as BMI, HbA_{1C}, PBG, FCP, Scr, SUA, age, duration of diabetes, metformin use, insulin use, SGLT2i use, and GLP-1RA use, the significance of VFA as a risk factor for DKD remained, though the *OR* value was somewhat attenuated (*OR*=1.021, 95%*CI:* 1.001–1.042, *P*=0.035).

2.5 Predictive Value of VFA for DKD

ROC curve analysis showed that the optimal cutoff value for predicting DKD using VFA was 71.05 cm² (Youden index 0.233; sensitivity 0.467, specificity 0.726). The area under the curve (AUC) was 0.600 (95%*CI:* 0.529–0.670, *P*=0.006). See **Figure 1**.

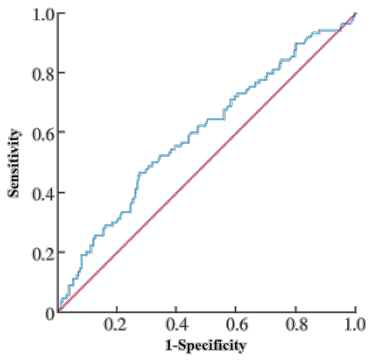

Fig.1 ROC curve for VFA in the prediction of DKD

3 Discussion

The pathogenesis of DKD (diabetic kidney disease) related to VFA (visceral fat area) is complex. Most studies suggest that the development and progression of DKD and VFA are associated with oxidative stress, inflammation, lipotoxicity [8], mitochondrial dysfunction [9], and changes in hemodynamics [10]. Oxidative stress can directly damage podocytes, mesangial cells, and endothelial cells, leading to damage to the mechanical filtration structures of the kidneys [11]. Inflammatory factors, through the development of hyperglycemic memory, generate advanced glycation end-products that harm the kidney's target cells. Additionally, certain inflammatory factors, such as nuclear factor (NF)-κB, nucleotide-binding oligomerization domain (NOD)-like receptor protein 3 (NLRP3), interleukin (IL)-1β, IL-6, tumor necrosis factor-alpha (TNF-α), and monocyte chemoattractant protein-1 (MCP-1), participate in immune-inflammatory responses, leading to kidney damage [12]. Lipotoxicity is triggered by improper accumulation of lipids, which causes organelle dysfunction, chronic inflammation, cellular damage, and cell death [8,13]. Excess fat can promote the attachment of pro-inflammatory factors such as IL-6 and TNF-α, exacerbating oxidative stress in the kidneys and promoting kidney damage [14]. The accumulation of visceral fat can also lead to changes in renal hemodynamics, causing the kidneys to remain in a state of high filtration rate, thereby contributing to kidney damage [10,15].

Currently, the treatment for DKD primarily focuses on controlling blood glucose and blood pressure, reducing the disease's progression [16]. Common medications include angiotensin-converting enzyme inhibitors (ACEI), angiotensin receptor blockers (ARB), and SGLT-2i [17-18]. Recent studies emphasize the protective role of non-steroidal mineralocorticoid receptor antagonists (such as finerenone and esaxerenone) in alleviating proteinuria and repairing renal function in DKD patients [19]. Imeglimin, a mitochondrial-mediated renal protection drug, has also shown promise [18]. Endothelin receptor antagonists like atrasentan have been proven to improve renal microvascular circulation [20]. In addition, stem cell therapy and Chinese medicine provide new directions for future DKD treatment.

An increasing number of researchers have explored the impact of VFA on DKD. Studies by Huang [21], Yang [22] and He [23] have found that VFA is positively correlated with the incidence of early DKD, and VFA is an independent risk factor for DKD in diabetic patients. Our study found that the VFA in the DKD group was higher than in the non-DKD group, and VFA showed a positive correlation with DKD. Unlike the aforementioned studies, the T2DM patients included in this study had a BMI between 18.5 and <24 kg/m². Excluding the influence of obesity further highlights the role of visceral fat in the risk of DKD. There is limited research on the classification of VFA both domestically and internationally, but most researchers define VFA>100 cm² as increased visceral fat [24]. Some studies suggest that the VFA threshold is 111 cm² for men and 91 cm² for women [25]. However, some studies suggest that the effect of VFA on DKD follows a

U-shape [26]. In this study, the VFA threshold was 71.05 cm², but the AUC was 0.600, which indicates a relatively low predictive value for DKD. Compared to several cross-sectional studies [27-28], the AUC for prediction is similar. This suggests that the low predictive value of VFA as a single indicator is not due to an insufficient sample size, but likely related to the research methods and design.

This study also identified several other risk factors for DKD, including age, disease duration, fasting C-peptide levels, creatinine, uric acid, urinary microalbumin, and the use of metformin, SGLT-2i, and GLP-1RA. Therefore, in clinical practice, special attention should be given to T2DM patients who are older, have a longer disease duration, elevated uric acid levels, increased VFA, and higher fasting C-peptide levels, as they may be at higher risk for DKD. In clinical practice, attention should be paid to the selection of drugs in the treatment of DKD patients.

There are some limitations to this study. First, this is a cross-sectional study with a relatively small sample size, resulting in weaker statistical correlations and potential selection bias in the population. Therefore, it is necessary to expand the sample size and conduct multi-center studies in the future. Second, the study did not include a history of hypertension, so the potential confounding bias from hypertension cannot be ruled out. Lastly, another limitation in sample selection is the inability to exclude the confounding effect of age on DKD.

In conclusion, our study found that in T2DM patients with normal BMI, those with DKD had higher VFA compared to those without DKD, and VFA was positively correlated with DKD. As VFA increases, the risk of developing DKD also increases. When VFA ≥ 71.05 cm², the risk of DKD in T2DM patients with normal BMI increases. For every 1 cm² increase in VFA, the risk of DKD in these patients increases by 2.1% to 2.4%.

Conflict of Interest None

References

[1] Thipsawat S. Early detection of diabetic nephropathy in patient with type 2 diabetes mellitus: a review of the literature[J]. Diab Vasc Dis Res, 2021, 18(6): 14791641211058856.
[2] Samsu N. Diabetic nephropathy: challenges in pathogenesis, diagnosis, and treatment[J]. Biomed Res Int, 2021, 2021: 1497449.
[3] Bensussen A, Torres-Magallanes JA, Roces de Álvarez-Buylla E. Molecular tracking of insulin resistance and inflammation development on visceral adipose tissue[J]. Front Immunol, 2023, 14: 1014778.
[4] Dhawan D, Sharma S. Abdominal obesity, adipokines and non-communicable diseases[J]. J Steroid Biochem Mol Biol, 2020, 203: 105737.
[5] Kolb H. Obese visceral fat tissue inflammation: from protective to detrimental?[J]. BMC Med, 2022, 20(1): 494.
[6] Kataoka H, Nitta K, Hoshino J. Visceral fat and attribute-based medicine in chronic kidney disease[J]. Front Endocrinol, 2023, 14: 1097596.
[7] Zhong PT, Tan SY, Zhu ZT, et al. Normal-weight central obesity and risk of cardiovascular and microvascular events in adults with prediabetes or diabetes: Chinese and British cohorts[J]. Diabetes Metab Res Rev, 2023, 39(8): e3707.
[8] Chae SY, Kim Y, Park CW. Oxidative stress induced by lipotoxicity and renal hypoxia in diabetic kidney disease and possible therapeutic interventions: targeting the lipid metabolism and hypoxia[J]. Antioxidants, 2023, 12(12): 2083.
[9] Galvan DL, Mise K, Danesh FR. Mitochondrial regulation of diabetic kidney disease[J]. Front Med, 2021, 8: 745279.
[10] Zhao PP, Li Q, Du TQ, et al. Association of different obesity indexes with diabetic kidney disease in patients with type 2 diabetes mellitus: a cross-sectional study[J]. Sci Rep, 2024, 14(1): 21571.
[11] Li X, Lu LH, Hou WT, et al. Epigenetics in the pathogenesis of diabetic nephropathy[J]. Acta Biochim Biophys Sin, 2022, 54(2): 163-172.
[12] Jin Q, Liu TT, Qiao Y, et al. Oxidative stress and inflammation in diabetic nephropathy: role of polyphenols[J]. Front Immunol, 2023, 14: 1185317.
[13] Schelling JR. The contribution of lipotoxicity to diabetic kidney disease[J]. Cells, 2022, 11(20): 3236.
[14] Li CY, Wang G, Zhang JL, et al. Association between visceral adiposity index and incidence of diabetic kidney disease in adults with diabetes in the United States[J]. Sci Rep, 2024, 14(1): 17957.
[15] Zhao QY, Yi XY, Wang ZH. Meta-analysis of the relationship between abdominal obesity and diabetic kidney disease in type 2 diabetic patients[J]. Obes Facts, 2021, 14(4): 338-345.
[16] Elendu C, John Okah M, Fiemotongha KDJ, et al. Comprehensive advancements in the prevention and treatment of diabetic nephropathy: a narrative review[J]. Medicine, 2023, 102(40): e35397.
[17] Yang J, Liu ZS. Mechanistic pathogenesis of endothelial dysfunction in diabetic nephropathy and retinopathy[J]. Front Endocrinol, 2022, 13: 816400.
[18] Mima A. Mitochondria-targeted drugs for diabetic kidney disease[J]. Heliyon, 2022, 8(2): e08878.
[19] Yao L, Liang XH, Liu YM, et al. Non-steroidal mineralocorticoid receptor antagonist finerenone ameliorates mitochondrial dysfunction via PI3K/Akt/ENOS signaling pathway in diabetic tubulopathy[J]. Redox Biol, 2023, 68: 102946.
[20] Sawaf H, Thomas G, Taliercio JJ, et al. Therapeutic advances in diabetic nephropathy[J]. J Clin Med, 2022, 11(2): 378.
[21] Huang JQ, Xu Y, Tian Y, et al. Clinical observation of the effect of visceral fat area on early diabetic nephropathy[J]. J Clin Exp Med, 2023, 22(17): 1843-1846. **[In Chinese]**
[22] Yang L, Shi WJ, Zhao L, et al. Clinical observation on the influence of visceral fat area in type 2 diabetes to early diabetic nephropathy[J]. Med Pharm Yunnan, 2022, 43(1): 40-43. **[In Chinese]**
[23] He F, Niu L, Luo G, et al. Correlation of Chinese visceral adipose index and visceral fat area with diabetic nephropathy and their warning values[J]. Clin Focus, 2022, 37(12): 1089-1093. **[In Chinese]**
[24] Manabe S, Kataoka H, Mochizuki T, et al. Impact of visceral fat area in patients with chronic kidney disease[J]. Clin Exp Nephrol, 2021, 25(6): 608-620.
[25] Lin XP, Chen ZG, Huang HS, et al. Diabetic kidney disease progression is associated with decreased lower-limb muscle mass and increased visceral fat area in T2DM patients[J]. Front Endocrinol, 2022, 13: 1002118.
[26] Shi SM, Ni LH, Tian Y, et al. Association of obesity indices with diabetic kidney disease and diabetic retinopathy in type 2 diabetes: a real-world study[J]. J Diabetes Res, 2023, 2023: 3819830.
[27] Zheng J, Hu Y, Xu HW, et al. Normal-weight visceral obesity promotes a higher 10-year atherosclerotic cardiovascular disease risk in patients with type 2 diabetes mellitus-a multicenter study in China[J]. Cardiovasc Diabetol, 2023, 22(1): 137.
[28] Wu YR, Wan Q, Xu Y, et al. Lower visceral fat area in patients with type 2 diabetic peripheral neuropathy[J]. Diabetes Metab Syndr Obes, 2022, 15: 3639-3654.

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·论 著·

身体质量指数正常的2型糖尿患者内脏脂肪面积与糖尿病肾病的相关性

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摘要：目的 探讨身体质量指数（BMI）正常的2型糖尿病（T2DM）患者中糖尿病肾病（DKD）的发生与内脏脂肪面积（VFA）的相关性。**方法** 收集2022年8月18日至2024年4月8日浙江大学医学院附属第二医院内分泌科收治的309例BMI正常（18.5～<24.0 kg/m^2）T2DM患者的资料进行横断面研究，根据是否有DKD分为DKD组（n=90）和非DKD组（n=219）。比较两组患者的基线资料和实验室指标等数据的差异。利用Spearman相关性分析和logistic回归分析研究VFA与DKD的相关性，绘制受试者工作特征（ROC）曲线评估VFA在DKD诊断中的价值。**结果** DKD组患者VFA高于非DKD组，差异有统计学意义[（70.16±25.79）cm^2 *vs*（61.93±22.17）cm^2，t=2.823，P=0.005]。Spearman相关性分析结果显示DKD与年龄（r=0.218，P<0.01）、病程（r=0.202，P<0.01）、VFA（r=0.157，P=0.006）、三酰甘油（r=0.170，P=0.003）、血肌酐（r=0.499，P<0.01）、血尿酸（r=0.318，P<0.01）、尿白蛋白/尿肌酐比值（r=0.532，P<0.01）呈正相关。单因素logistic回归分析结果显示VFA是DKD发生的危险因素（*OR*=1.015，95%*CI*：1.004~1.025，P=0.006），在调整性别、BMI、病程等混杂因素后VFA仍是DKD的危险因素（*OR*=1.024，95%*CI*：1.008~1.040，P=0.003）。ROC曲线分析结果表明，VFA预测DKD的截断值为71.05 cm^2，敏感度为0.467，特异度为0.726，曲线下面积（AUC）为0.600（95%*CI*：0.529~0.670）。**结论** 在BMI正常的T2DM患者中，VFA与DKD呈正相关，且VFA是DKD的危险因素。随着VFA的增加，DKD的患病风险增高。

关键词： 2型糖尿病；糖尿病肾病；内脏脂肪面积；身体质量指数

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Correlation between visceral fat area and diabetic kidney disease in type 2 diabetes mellitus patients with normal body mass index

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Abstract: Objective To investigate the correlation between visceral fat area (VFA) and diabetic kidney disease (DKD) in type 2 diabetes mellitus (T2DM) patients with normal body mass index (BMI). **Methods** A cross-sectional study was conducted on 309 T2DM patients with normal BMI (18.5-<24.0 kg/m^2) in the Department of Endocrinology of the Second Affiliated Hospital of Zhejiang University School of Medicine from August 18, 2022 to April 8, 2024. The patients were divided into DKD group (n=90) and non-DKD group (n=219) according to whether DKD was merged or not. Baseline data, laboratory indicators between the two groups were compared. Spearman correlation analysis and logistic regression analysis were used to investigate the correlation between VFA and DKD. Receiver operating characteristic (ROC) curve was drawn to evaluate the value of VFA in the diagnosis of DKD. **Results** VFA in DKD group was higher significantly than that in the non-DKD group [(70.16±25.79) cm^2 *vs* (61.93±22.17) cm^2, t=2.823, P=0.005]. The results of Spearman correlation analysis showed that DKD was positively correlated with age (r=0.218, P<

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0.01), disease duration (r=0.202, P<0.01), VFA (r=0.157, P=0.006), triglycerides (r=0.170, P=0.003), serum creatinine (r=0.499, P<0.01), serum uric acid (r=0.318, P<0.01), and urine albumin-to-creatinine ratio (r=0.532, P<0.01), respectively. The result of univariate logistic regression analysis showed that VFA was a risk factor for DKD (OR=1.015, 95% CI: 1.004-1.025, P=0.006). After adjusting for confounding factors such as gender, BMI, and disease duration, VFA remained be a risk factor for DKD (OR=1.024, 95% CI: 1.008-1.040, P=0.003). The ROC curve analysis results showed that the cut-off value of VFA for predicting DKD was 71.05 cm^2, the sensitivity was 0.467, the specificity was 0.726, and the area under the curve (AUC) was 0.600 (95% CI: 0.529-0.670). **Conclusion** In T2DM patients with normal BMI, VFA is positively correlated with DKD, and VFA is a risk factor for DKD. The risk of DKD increases with the increase of VFA.

Keywords: Type 2 diabetes mellitus; Diabetic kidney disease; Visceral fat area; Body mass index

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糖尿病肾病(diabetic kidney disease, DKD)目前已成为全球终末期肾病(end-stage renal disease, ESRD)的主要病因。在有10年病程的2型糖尿病(type 2 diabetes mellitus, T2DM)患者中,约40%会发展成为DKD[1]。据估计,中国有近3 900万T2DM患者合并慢性肾脏病(约占1/3),这是一个不容小觑的数字[2]。内脏脂肪影响胰岛抵抗及代谢紊乱[3],从而使全身炎症、氧化应激和肾素—血管紧张素—醛固酮系统的激活导致肾脏损伤[4-5]。此外,内脏脂肪组织脂毒性可影响肾脏导致蛋白尿增加和肾纤维化及功能障碍[6]。临床工作中,DKD的患者有体型肥胖也有体型正常,体型正常患者的内脏脂肪是否和DKD相关的研究较少。如若能发现内脏脂肪面积(visceral fat area, VFA)与身体质量指数(body mass index, BMI)正常的DKD存在相关性,可为BMI正常的T2DM患者防治DKD带来指导意义。

1　对象与方法

1.1　*研究对象*　本研究为一项回顾性横断面研究,收集了2022年8月18日至2024年4月8日于浙江大学医学院附属第二医院内分泌科就诊的309例BMI正常(18.5～<24.0 kg/m^2)的T2DM患者的临床数据。该研究得到了浙江大学医学院附属第二医院伦理委员会的批准[伦理批件号:〔2025〕伦审研第(0430)号],研究中纳入的患者均已签署了知情同意书。

纳入标准:(1)明确诊断为T2DM;(2) 18.5 kg/m^2≤BMI<24.0 kg/m^2;(3)住院期间VFA及其他临床数据资料齐全。排除标准:(1)1型糖尿病及特殊类型糖尿病、妊娠期糖尿病;(2)合并急性并发症,如酮症酸中毒、高血糖高渗状态、低血糖等;(3)合并严重感染;(4)合并肿瘤;(5)合并其他影响血糖的内分泌疾病,如肢端肥大、库欣病、甲状腺疾病等;(6)合并严重心肺功能不全;(7)目前已进行透析治疗;(8)合并其他肾脏疾病,如狼疮性肾炎。

1.2　*疾病诊断及定义*　(1)T2DM的诊断参照《中国2型糖尿病防治指南(2017版)》[7]:典型的糖尿病症状(口干多饮、多尿、多食以及不明原因体重下降)加上以下结果中任选一项,随机血糖≥11.1 mmol/L、空腹血糖(fasting blood glucose, FBG)≥7.0 mmol/L、口服葡萄糖耐量试验2小时血糖≥11.1 mmol/L。若没有典型的糖尿病症状,需改日复查以上结果再次确认。再根据患者的发病年龄、有无家族史、起病缓急、症状体征、C肽水平、糖尿病自身抗体等联合诊断分型为T2DM。(2)DKD的诊断参照《中国2型糖尿病防治指南(2017版)》[7]:T2DM病史,并且排除其他可能的肾损伤原因,估算肾小球滤过率(estimated glomerular filtration rate, eGFR)<60 mL/(min·1.73m^2);尿白蛋白/尿肌酐比值(urine albumin-to-creatinine ratio, UACR)≥30 mg/g,且时间超过3个月,眼底出现糖尿病视网膜病变可辅助诊断DKD。

1.3　*研究指标*

1.3.1　基线资料　包括年龄、性别、体重、身高、BMI、VFA、吸烟史、饮酒史、糖尿病病程、二甲双胍使用史、胰岛素使用史、钠-葡萄糖协同转运蛋白2抑制剂(sodium-dependent glucose transporters 2 inhibitor, SGLT-2i)使用史、胰高血糖素样肽-1受体激动剂(glucagon-like peptide-1 receptor agonist, GLP-1RA)使用史。

1.3.2　实验室指标　FBG、空腹C肽、糖化血红蛋白(glycated hemoglobin, HbA_{1C})、三酰甘油(triglycerides, TG)、高密度脂蛋白胆固醇(high density lipoprotein cholesterol, HDL-C)、低密度脂蛋白胆固醇(low density lipoprotein cholesterol, LDL-C)、血肌酐、eGFR、UACR、血尿酸、丙氨酸转氨酶(alanine aminotransferase, ALT)、天冬氨酸转氨酶(aspartate aminotransferase, AST)、γ-谷氨酰转移酶(gamma-glutamyl trans-

ferase, GGT)、碱性磷酸酶(alkaline phosphatase, ALP)。

1.4　统计与分析　采用Excel表格整理数据,采用SPSS 25.0软件统计学分析。符合正态分布的计量资料采用$\bar{x}\pm s$表示,比较采用t检验;不符合正态分布的计量资料采用$M(P_{25},P_{75})$表示,比较采用秩和检验。计数资料以例(%)表示,比较采用χ^2检验。采用Spearman相关系数分析各个因素与DKD之间的相关性。使用logistic回归模型分析VFA与DKD的关系。采用受试者工作特征(receiver operating characteristic,ROC)曲线评估VFA诊断DKD的价值。以上统计学检验均以P<0.05为差异有统计学意义。

2　结　果

2.1　两组患者基线资料的比较　两组患者性别、BMI、胰岛素使用、吸烟史、饮酒史差异无统计学意义(P>0.05)。两组患者年龄、病程、VFA、二甲双胍使用、SGLT2i使用、GLP1RA使用情况差异均有统计学意义(P<0.01)。见表1。

2.2　两组实验室指标的比较结果　两组之间的空腹C肽、TG、血尿酸、eGFR差异有统计学意义(P<0.05)。见表2。

2.3　DKD与各指标的相关性分析　Spearman相关性分析结果显示,DKD与年龄(r=0.218,P<0.01)、糖尿病病程(r=0.202,P<0.01)、VFA(r=0.157,P=0.006)、三酰甘油(r=0.170,P=0.003)、血肌酐(r=0.499,P<0.01)、血尿酸(r=0.318,P<0.01)、UACR(r=0.532,P<0.01)呈正相关,与eGFR(r=−0.550,P<0.01)呈负相关,差异均有统计学意义。

2.4　DKD的logistic回归分析　以DKD为因变量,将各指标为自变量进行单因素logistic回归分析,结果显示:年龄、病程、VFA、空腹C肽、血肌酐、eGFR、血尿酸及二甲双胍、SGLT2i、GLP-1RA的使用均与DKD的发生有关(P<0.05)。见表3。

在上述未调整混杂因素的模型中显示高VFA是DKD的危险因素(OR=1.015,95%CI:1.004~1.025, P=0.006)。在调整了性别、BMI的模型1中,高VFA是

表1　两组患者基线资料比较结果

Tab.1　Comparison of baseline data between two groups of patients

项目	DKD组(n=90)	非DKD组(n=219)	$\chi^2/t/Z$值	P值
性别[例(%)]				
男	61(67.78)	132(60.27)	1.532	0.216
女	29(32.22)	87(39.73)		
年龄(岁,$\bar{x}\pm s$)	65.37±11.56	58.95±13.29	4.001	0.001
BMI(kg/m^2)[a]	21.30(19.90,22.63)	20.90(19.60,22.30)	1.648	0.102
VFA(cm^2,$\bar{x}\pm s$)	70.16±25.79	61.93±22.17	2.823	0.005
药物使用情况[例(%)]				
二甲双胍	42(46.67)	134(61.19)	5.486	0.019
胰岛素	66(73.33)	142(64.84)	2.091	0.148
SGLT2i	50(55.56)	82(37.44)	8.552	0.003
GLP-1RA	12(13.33)	11(5.02)	6.394	0.011
糖尿病病程(年)[a]	11.00(7.50,20.00)	9.00(2.00,15.00)	3.542	<0.001
吸烟史[例(%)]	30(33.33)	67(30.59)	0.157	0.692
饮酒史[例(%)]	16(17.78)	54(24.66)	1.878	0.171

注:[a]为数据以$M(P_{25},P_{75})$表示。

表2　两组患者实验室指标比较结果　($\bar{x}\pm s$)

Tab.2　Comparison of laboratory indicators between two groups of patients　($\bar{x}\pm s$)

项目	DKD组(n=90)	非DKD组(n=219)	t/Z值	P值
HbA_{1c}(%)	9.70±2.72	9.28±2.35	1.371	0.171
空腹C肽(nmol/L)[a]	0.44(0.20,0.65)	0.33(0.21,0.52)	1.957	0.050
FBG(mmol/L)	8.26±3.86	8.24±3.32	0.033	0.973
ALT(u/L)[a]	17.00(13.00,26.25)	17.00(13.00,26.00)	0.102	0.919
AST(u/L)[a]	20.00(16.75,26.00)	19.00(16.00,24.00)	1.247	0.213
γ-GT(u/L)[a]	20.50(14.75,34.25)	19.00(14.00,26.00)	1.474	0.140
ALP(u/L)[a]	77.00(64.75,92.00)	75.00(62.00,94.00)	0.409	0.683
TG(mmol/L)[a]	1.36(0.97,1.89)	1.17(0.80,1.59)	2.991	0.003
LDL-C(mmol/L)	2.39±1.00	2.34±0.92	0.469	0.639
HDL-C(mmol/L)	1.29±0.39	1.33±0.41	0.725	0.469
血肌酐(μmol/L)[a]	90.05(70.08,125.95)	61.30(54.45,71.30)	8.764	<0.001
eGFR[mL/(min·1.73 m^2)]	68.06±24.72	99.08±15.04	13.479	<0.001
血尿酸(μmol/L)	363.24±104.41	295.01±82.50	6.095	<0.001
UACR(mg/g)[a]	117.44(39.57,681.49)	12.50(12.50,24.74)	9.305	<0.001

注:[a]为数据以$M(P_{25},P_{75})$表示。

DKD 的危险因素的显著性仍存在，且 *OR* 值较未调整前升高（*OR*=1.023，95%*CI*：1.007~1.038，*P*=0.004）。模型 2 是在模型 1 的基础上继续调整了病程，高 VFA 是 DKD 的危险因素的显著性仍存在，*OR* 值较前升高（*OR*=1.024，95%*CI*：1.008~1.040，*P*=0.003）。在继续调整 BMI、HbA_{1c}、PBG、空腹 C 肽、血肌酐、血尿酸、年龄、糖尿病病程、二甲双胍的使用、胰岛素的使用、SGLT2i 的使用、GLP-1RA 的使用这些因素后的模型 3 中，VFA 仍是 DKD 的危险因素，但 *OR* 值有所减弱（*OR*=1.021，95%*CI*：1.001~1.042，*P*=0.035）。

2.5　VFA 对 DKD 的预测价值　ROC 曲线分析结果表明，VFA 预测 DKD 的截断值为 71.05 cm^2，约登指数 0.233；敏感度为 0.467，特异度为 0.726，曲线下面积（area under curve，AUC）为 0.600（95%*CI*：0.529~0.670）。见图 1。

表 3　单因素 logistic 回归分析结果

Tab.3　Results of univariate logistic regression analysis

项目	*β* 值	*SE*	*OR* 值	95%*CI*	Wald χ^2 值	*P* 值
性别为女	0.327	0.264	1.386	0.826~2.368	1.526	0.217
年龄	0.042	0.011	1.043	1.021~1.066	14.552	0.001
BMI	0.130	0.079	1.139	0.975~1.331	2.691	0.101
病程	0.054	0.015	1.055	1.024~1.088	12.103	0.001
VFA	0.014	0.005	1.015	1.004~1.025	7.506	0.006
HbA_{1c}	0.069	0.050	1.071	0.971~1.182	1.869	0.172
空腹 C 肽	1.329	0.409	3.778	1.695~8.423	10.558	0.001
FBG	0.001	0.036	1.001	0.933~1.074	0.001	0.973
血肌酐	0.064	0.008	1.066	1.048~1.084	56.490	0.001
eGFR	−0.081	0.010	0.922	0.905~0.940	68.545	0.001
血尿酸	0.008	0.002	1.008	1.005~1.011	27.935	0.001
UACR	0.013	0.003	1.013	1.008~1.018	26.190	0.001
使用二甲双胍	0.589	0.253	1.802	1.098~2.957	5.426	0.020
使用胰岛素	−0.400	0.277	0.671	0.389~1.155	0.278	0.149
使用 SGLT2i	0.736	0.254	2.088	1.270~3.435	8.408	0.004
使用 GLP-1RA	1.068	0.438	2.909	1.233~6.865	5.943	0.015

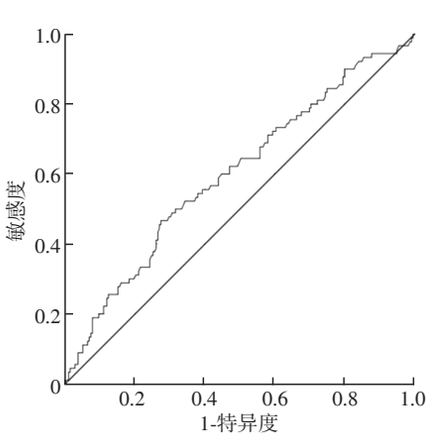

图 1　VFA 预测 DKD 的 ROC 曲线

Fig.1　ROC curve for VFA in the prediction of DKD

3　讨　论

DKD 与 VFA 相关的发病机制复杂。多数研究认为 DKD 与 VFA 的发生和发展和氧化应激反应、炎症、脂毒性[8]、线粒体功能障碍[9]与血流动力学的变化相关[10]。氧化应激可直接损伤足细胞、系膜细胞以及内皮细胞，导致肾脏的机械滤过结构损伤。炎症因子通过参与高血糖记忆的发展产生糖基化终产物对肾脏的靶细胞产生损伤[11]。另外，一些炎症因子如核因子（nuclear factor，NF）-κB、核苷酸结合寡聚化结构域（nucleotide-binding oligomerization domain，NOD）样受体蛋白 3（NOD-like receptor protein3，NLRP3）、白细胞介素（interleukin，IL）-1β、IL-6、肿瘤坏死因子-α（tumor necrosis factor alpha，TNF-α）、单核细胞趋化蛋白-1（monocyte chemoattractant protein-1，MCP-1）等，参与免疫炎症反应，导致肾脏损伤[12]。脂毒性是脂质的不当累积引发细胞器的紊乱、持续炎症、细胞的损伤以及细胞的死亡[8,13]。脂肪过多可促使一些如 IL-6、TNF-α等促炎因子的趋附，加剧肾脏的氧化应激反应，加重肾脏的损伤[14]。内脏脂肪的堆积还可以引起肾脏血流动力学的改变，让肾脏长期处于高率过滤的状态致使肾脏损伤[10,15]。

目前 DKD 的治疗仍以控糖控压，减少疾病的发生进展为主要的手段[16]，药物上以使用血管紧张素转化酶抑制剂（angiotensin converting enzyme inhibitor，ACEI）、血管紧张素受体阻滞剂（angiotensin receptor blocker，ARB）和 SGLT-2i 为主[17-18]。在最近研究中，强调了非甾体盐皮质激素受体拮抗剂（非奈利酮、esaxerenone）的保护作用，缓解 DKD 患者的蛋白尿和修复肾功能[19]。Imeglimin 是一种线粒体介导的肾脏保护机制型的新型口服降糖药物[18]。内皮素拮抗剂阿曲生坦也被证明可以改善肾微血管循环[20]。除此之外，干细胞疗法以及中医药疗法也为未来 DKD 的治疗提供了一个新方向。

目前越来越多的研究者们发现 VFA 对 DKD 的影响。黄佳琪[21]、杨璐[22]、贺枫[23]等研究者发现 VFA 与早期 DKD 的发病率呈正相关，且高 VFA 是糖尿病患者发生 DKD 的独立危险因素。本研究发现 DKD 组的 VFA 高于非 DKD 组，且 VFA 与 DKD 呈正相关。与上述研究不同的是本研究纳入的 T2DM 患者的 BMI 在 18.5~ < 24.0 kg/m^2，除去肥胖的影响，更加凸显了内脏脂肪对 DKD 的风险。目前国内外对于 VFA 的分组研究较少，大多数研究者认为 VFA > 100 cm^2 可定义为内脏脂肪增多[24]。也有研究给出了 VFA 界值是

男性111 cm^2，女性91 cm^2[25]。但也有研究认为VFA对DKD的影响是U型的[26]。本研究的VFA界值为71.05 cm^2，但AUC为0.600，对DKD的预测价值较低。与多项横向研究比较[27-28]AUC的预测值相似。可见，VFA单个预测值指标偏低并不是由样本数量不足造成，可能与研究的方法与设计等相关。本研究还发现，年龄、糖尿病病程、空腹C肽水平、血肌酐、血尿酸、UACR及二甲双胍、SGCT2i、GLP-1RA的使用史都是DKD的影响因素。所以在临床工作中，对于年龄大、病程长、尿酸高、VFA增大、空腹C肽水平升高的T2DM患者要当心其患DKD的风险；要注意在DKD患者治疗中选择的药物。

本研究存在一些局限性。首先，本研究为横断面研究，纳入样本量偏少，结果统计得出的相关性较弱，也存在人群选择偏倚，因此有必要进一步扩展样本量，进行多中心研究。其次，本研究未能纳入高血压病史的情况，不能排除高血压带来的混杂偏倚。最后，在样本选择上无法排除年龄对DKD的混杂影响。

综上所述，本研究发现在BMI正常的T2DM患者中，DKD患者的VFA高于非DKD患者，DKD与VFA呈正相关，随着VFA的增加，DKD的发病风险增高。当VFA≥71.05 cm^2时，BMI正常的T2DM患者患DKD的风险增加。VFA每增加1 cm^2，BMI正常的T2DM患者罹患DKD的风险增加2.1%~2.4%。

利益冲突 无

参考文献

[1] Thipsawat S. Early detection of diabetic nephropathy in patient with type 2 diabetes mellitus: a review of the literature[J]. Diab Vasc Dis Res, 2021, 18(6): 14791641211058856.

[2] Jia WP, Yu R, Wang LM, et al. Prevalence of chronic kidney disease among Chinese adults with diabetes: a nationwide population-based cross-sectional study[J]. Lancet Reg Health West Pac, 2025,55:101463.

[3] Bensussen A, Torres-Magallanes JA, de Álvarez-Buylla ER. Molecular tracking of insulin resistance and inflammation development on visceral adipose tissue[J]. Front Immunol, 2023, 14: 1014778.

[4] Dhawan D, Sharma S. Abdominal obesity, adipokines and non-communicable diseases[J]. J Steroid Biochem Mol Biol, 2020, 203: 105737.

[5] Kolb H. Obese visceral fat tissue inflammation: from protective to detrimental?[J]. BMC Med, 2022, 20(1): 494.

[6] Kataoka H, Nitta K, Hoshino J. Visceral fat and attribute-based medicine in chronic kidney disease[J]. Front Endocrinol (Lausanne), 2023, 14: 1097596.

[7] 中华医学会糖尿病学分会. 中国2型糖尿病防治指南(2017年版)[J].中国实用内科杂志,2018,38(4):292-344.

[8] Chae SY, Kim Y, Park CW. Oxidative stress induced by lipotoxicity and renal hypoxia in diabetic kidney disease and possible therapeutic interventions: targeting the lipid metabolism and hypoxia[J]. Antioxidants (Basel), 2023, 12(12): 2083.

[9] Galvan DL, Mise K, Danesh FR. Mitochondrial regulation of diabetic kidney disease[J]. Front Med (Lausanne), 2021, 8: 745279.

[10] Zhao PP, Li Q, Du TQ, et al. Association of different obesity indexes with diabetic kidney disease in patients with type 2 diabetes mellitus: a cross-sectional study[J]. Sci Rep, 2024, 14(1): 21571.

[11] Li X, Lu LH, Hou WT, et al. Epigenetics in the pathogenesis of diabetic nephropathy[J]. Acta Biochim Biophys Sin (Shanghai), 2022, 54(2): 163-172.

[12] Jin Q, Liu TT, Qiao Y, et al. Oxidative stress and inflammation in diabetic nephropathy: role of polyphenols[J]. Front Immunol, 2023, 14: 1185317.

[13] Schelling JR. The contribution of lipotoxicity to diabetic kidney disease[J]. Cells, 2022, 11(20): 3236.

[14] Li CY, Wang G, Zhang JL, et al. Association between visceral adiposity index and incidence of diabetic kidney disease in adults with diabetes in the United States[J]. Sci Rep, 2024, 14(1): 17957.

[15] Zhao QY, Yi XY, Wang ZH. Meta-analysis of the relationship between abdominal obesity and diabetic kidney disease in type 2 diabetic patients[J]. Obes Facts, 2021, 14(4): 338-345.

[16] Elendu C, Okah MJ, Fiemotongha KDJ, et al. Comprehensive advancements in the prevention and treatment of diabetic nephropathy: a narrative review[J]. Medicine (Baltimore), 2023, 102(40): e35397.

[17] Yang J, Liu ZS. Mechanistic pathogenesis of endothelial dysfunction in diabetic nephropathy and retinopathy[J]. Front Endocrinol (Lausanne), 2022, 13: 816400.

[18] Mima A. Mitochondria-targeted drugs for diabetic kidney disease[J]. Heliyon, 2022, 8(2): e08878.

[19] Wu Y, Lin H, Tao Y, et al. Network meta-analysis of mineralocorticoid receptor antagonists for diabetic kidney disease[J]. Front Pharmacol, 2022,13:967317

[20] Sawaf H, Thomas G, Taliercio JJ, et al. Therapeutic advances in diabetic nephropathy[J]. J Clin Med, 2022, 11(2): 378.

[21] 黄佳琪, 徐莹, 田园, 等. 内脏脂肪面积对早期糖尿病肾病影响的临床观察[J]. 临床和实验医学杂志, 2023, 22(17): 1843-1846.

[22] 杨璐, 施文军, 赵玲, 等. 2型糖尿病患者内脏脂肪面积对早期糖尿病肾病影响的临床观察[J]. 云南医药, 2022, 43(1): 40-43.

[23] 贺枫, 牛璐, 雒杲, 等. 中国人内脏脂肪指数及内脏脂肪面积与糖尿病肾病的相关性及其预警价值[J]. 临床荟萃, 2022, 37(12): 1089-1093.

[24] Manabe S, Kataoka H, Mochizuki T, et al. Impact of visceral fat area in patients with chronic kidney disease[J]. Clin Exp Nephrol, 2021, 25(6): 608-620.

[25] Lin XP, Chen ZG, Huang HS, et al. Diabetic kidney disease progression is associated with decreased lower-limb muscle mass and increased visceral fat area in T2DM patients[J]. Front Endocrinol (Lausanne), 2022, 13: 1002118.

[26] Shi SM, Ni LH, Tian Y, et al. Association of obesity indices with diabetic kidney disease and diabetic retinopathy in type 2 diabetes: a real-world study[J]. J Diabetes Res, 2023, 2023: 3819830.

[27] Zheng J, Hu Y, Xu HW, et al. Normal-weight visceral obesity promotes a higher 10-year atherosclerotic cardiovascular disease risk in patients with type 2 diabetes mellitus-a multicenter study in China[J]. Cardiovasc Diabetol, 2023, 22(1): 137.

[28] Wu YR, Wan Q, Xu Y, et al. Lower visceral fat area in patients with type 2 diabetic peripheral neuropathy[J]. Diabetes Metab Syndr Obes, 2022, 15: 3639-3654.

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