

This author's PDF version corresponds to the article as it appeared upon acceptance. Fully formatted PDF versions will be made available soon.

Associations between weight-adjusted-waist index and serum ferritin in patients with type 2 diabetes

doi: 10.6133/apjcn.202503/PP.0003

Published online: March 2025

Running title: TWI of young male athletes increases with PAEE

Yirong Wang MD^{1,2}, Pingping Zhao MD¹, Yang ting Zhao MD¹, Chongyang Chen MD¹, Xiaoyu Lv MD¹, Liting Wang MD², Jie Gao MD¹, Jingfang Liu MD, PhD¹

¹The First Clinical Medical College, Lanzhou University, Lanzhou, Gansu, China.

²Gansu Provincial Maternity and Child-care Hospital (Gansu Provincial Central Hospital), Lanzhou, Gansu, China

Authors' email addresses and contributions:

Yirong Wang: wangyr2023@lzu.edu.cn

Contribution: conceived the study question, and contributed to the study design, supervision of data collection, data analysis and interpretation, and writing the manuscript.

Pingping Zhao: 1258451661@qq.com

Contribution: commented on previous versions of the manuscript.

Yangting Zhao: sunpeach513@163.com

Contribution: material preparation and data collection.

Chongyang Chen: chenzhy21@lzu.edu.cn

Contribution: material preparation and data collection.

Xiaoyu Lv: lvxyaclo@163.com

Contribution: material preparation and data collection.

Liting Wang: 492929507@qq.com

Contribution: contributed to data interpretation.

Jie Gao: 164880352@qq.com

Contribution: undertook data collection and data analysis.

Jingfang Liu: conceived the study question, and contributed to the study design, supervision of data collection, data analysis and interpretation, and writing the manuscript.

Corresponding Author: Dr Jingfang Liu, Department of Endocrinology, First Hospital of Lanzhou University, No. 1, Donggang West Road, Chengguan District, Lanzhou City, Gansu Province, 730000, PR. China. Tel: +86-931-8356242. Email: ljf824168@126.com

ABSTRACT

Background and Objectives: To investigate the correlation between weight-adjusted-waist index (WWI), a novel obesity index, and serum ferritin (SF) level in patients with type 2 diabetes mellitus (T2DM), and the association between WWI and the prevalence of hyperferritinemia. **Methods and Study Design:** A total of 943 patients with T2DM were divided into three groups based on WWI tertile levels. Disparities in SF levels and the prevalence of hyperferritinemia were compared among these groups. The correlations among WWI, SF levels, and hyperferritinemia were analyzed in patients with T2DM. **Results:** As WWI tertile levels increased, SF levels tended to increase (p for trend <0.01). A statistically significant positive correlation was observed between the WWI and SF levels ($R = 0.263$, $p < 0.001$). After adjusting for confounders by multiple linear regression, a significant positive correlation was maintained between the WWI and SF levels [$\beta = 0.194$, 95% CI (49.914, 112.120)], $p < 0.01$]. Binary logistic regression analysis revealed a positive association between the WWI and the likelihood of hyperferritinemia, with a notably stronger correlation observed in females compared to males [OR = 3.248, 95% CI (2.027, 5.204), $p < 0.01$ vs. OR = 2.091, 95% CI (1.432, 3.054), $p < 0.01$]. **Conclusions:** Along with increasing WWI, SF levels gradually increased in patients with T2DM. The WWI exhibited a positive correlation with SF levels and hyperferritinemia, more significantly in female patients.

Key Words: weight-adjusted-waist index, serum ferritin, hyperferritinemia, type 2 diabetes; obesity

INTRODUCTION

With rapid socioeconomic development and changes in diet and lifestyle, diabetes has become a significant global health issue. Globally, 537 million patients with diabetes account for approximately 10.5% of the population. By 2030, the number of patients with diabetes is projected to increase to 643 million, reaching 783 million by 2045, representing a 46% increase.¹ Obesity increases synchronously with diabetes and exacerbates health threats. From 1990–2021, global obesity contributed to a 24.3% increase in disability-adjusted life years in patients with type 2 diabetes mellitus (T2DM).²

A prospective study among Chinese adults indicated that approximately 24.4% of diabetes risk is attributed to insulin resistance, while only 12.4% is linked to β -cell dysfunction. This suggests that insulin resistance has a greater impact on diabetes occurrence and development in Chinese adults, doubling that of β -cell dysfunction and tripling to 3.5 times in obese

individuals. Scholars have proposed that a chronic inflammatory state is a potential mechanism for insulin resistance development in patients with obesity and T2DM, particularly in individuals with visceral obesity. Fat deposition in the liver and pancreas, along with inflammatory molecules (adipokines) secreted by adipose tissue, negatively affects insulin sensitivity in the liver and other tissues.³

Body mass index (BMI) is commonly used to assess obesity. However, BMI cannot effectively distinguish between the distributions of muscle and fat mass. The weight-adjusted-waist index (WWI) is a new anthropometric index. According to the calculation of waist circumference (cm) divided by the square root of body weight (kg), waist circumference and weight can be standardized to reflect adverse metabolic components, such as high fat mass, low muscle mass, and low bone mass, and to better evaluate central obesity independent of body weight.⁴ Recent studies have reported that an increased WWI is associated with new-onset diabetes.⁵ Furthermore, several studies have revealed that the WWI is positively correlated with the risk of hyperuricemia and a variety of cardiocerebrovascular events, including stroke, coronary heart disease, heart failure left ventricular hypertrophy, and abdominal aortic calcification.⁶⁻¹¹

Serum ferritin (SF), the principal repository of stored iron within the body, plays crucial roles in iron homeostasis, maintaining cell function, and protecting proteins from potential iron toxicity.¹² Iron overload in the body increases the risk of chronic metabolic diseases, such as diabetes, by affecting insulin sensitivity.¹³ Several studies have demonstrated a close association between SF levels and fasting plasma glucose, insulin resistance the risk of diabetes, and nonalcoholic fatty liver disease.¹⁴⁻¹⁷ In clinical practice, ferritin is primarily used as a serum marker to reflect overall iron storage and inflammatory status.¹⁸

With an increased understanding of micronutrients, further research on the role of iron metabolism in glucose and lipid metabolism is required. The WWI is a useful index for evaluating central obesity. To date, no study has investigated the correlation between the WWI and SF levels in patients with T2DM. Therefore, this study aimed to explore the associations among the WWI, SF levels, and the prevalence of hyperferritinemia in patients with T2DM.

MATERIALS AND METHODS

Study population

Initially, 1633 patients with T2DM admitted to the Department of Endocrinology at a university hospital between March 2022 and March 2023 were considered for the study. Upon

application of the predefined inclusion and exclusion criteria, 943 patients with T2DM were enrolled into the study. This study was performed in accordance with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of the First Hospital of Lanzhou University (2024-04-28/LDYLL2024-315). Informed consent Written informed consent was obtained from all participants.

Inclusion criteria

Patients were over eighteen years of age and of Han ethnicity, met the World Health Organization (WHO) T2DM diagnostic criteria, and had complete clinical information and data.

Exclusion criteria

(1) Individuals diagnosed with type 1 diabetes, gestational diabetes, or another distinctive form of diabetes; (2) acute complications related to diabetes, including hyperosmolar hyperglycemia or diabetic ketoacidosis; (3) acute infection; (4) malignant tumors and autoimmune diseases; (5) severe heart disease defined as heart failure; (6) kidney disease defined by an eGFR $<30 \text{ mL}/(\text{min} \cdot 1.73 \text{ m}^2)$, or renal insufficiency necessitating renal replacement therapy; (7) liver disease defined by AST/ALT levels > 3 times the upper limit of normal; (8) hematologic disorders, including anemia (hemoglobin concentration below 120 g/L for adult males and below 110 g/L for adult females); (9) treatment with steroids, iron supplements, or blood transfusions recently; and (10) incomplete clinical data.

General characteristics of the study participants

Demographic and clinical information, including sex, age, diabetes duration, personal habits including smoking and alcohol consumption, menopausal status for female patients, past medical history, and medication usage, was extracted from medical records. Height, weight, waist circumference, hip circumference, and blood pressure were measured and recorded.

$$\text{BMI} = \text{weight (kg)}/\text{height (m)}^2$$

$$\text{Weight-adjusted-waist index (WWI)} = \text{waist circumference (cm)}/\text{weight (kg)}^{1/2}$$

Laboratory examinations

Following an 8-hour fasting period, 5 mL of venous blood was collected from all participants in the morning, and the serum was isolated. The alanine aminotransferase (ALT), aspartate aminotransferase (AST), fasting plasma glucose (FPG), total cholesterol (TC), serum

triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) levels were measured using an automatic biochemical analyzer (BS-220, Shenzhen Miabio, Guangdong Province). Glycated hemoglobin (HbA1c) levels were measured by high-performance liquid chromatography (Bio-Rad-D10; Bio-Rad Laboratories). Fasting insulin (FINS) and fasting C-peptide (FCP) levels were measured using a chemiluminescent immunoassay analyzer (CENTAUR-XP Automated Chemiluminescent Immunoassay Analyzer; Siemens Healthineers).

$$\text{HOMA-IR} = \text{FINS} \times \text{FPG} / 22.5$$

Body composition measurement

The distribution of abdominal fat was assessed by measuring the abdominal visceral fat area (VFA) and abdominal subcutaneous fat area (SFA) using an OsteoSys EXA-3000 dual-energy X-ray device.

Diagnostic criteria of disease

T2DM diagnoses were determined based on the World Health Organization's (WHO) 1999 diagnostic criteria.

Hyperferritinemia was defined as SF levels ≥ 300 ng/mL for adult males or ≥ 150 ng/mL for adult females.^{19,20}

Nonalcoholic fatty liver disease (NAFLD) was diagnosed by abdominal ultrasound examination by an experienced sonographer. The presence of two of the following abdominal ultrasound features suggests diffuse fatty liver: (1) the near-field echo of the liver demonstrates diffuse enhancement, which is more intense than that of the kidneys and spleen; (2) indistinct intrahepatic ductal structures; and (3) gradual weakening of the liver's far-field echo.

Study population grouping

The patients were divided into T1 ($\text{WWI} < 10.77$), T2 ($10.77 \leq \text{WWI} < 11.28$), and T3 ($\text{WWI} \geq 11.28$) groups based on the tertile levels of the WWI. Differences in SF levels and the prevalence of hyperferritinemia were compared among the groups.

Statistical methods

All the data were subjected to statistical analysis using IBM SPSS Statistics for Windows, Version 27.0 (IBM Corp.). The Shapiro–Wilk test was used to test normality. Normally

distributed measurement data are expressed as the mean \pm standard deviation ($\bar{x}\pm s$), while nonnormally distributed measures are expressed as medians and quartiles [M (P25, P75)]. One-way analysis of variance (ANOVA) was used for normally distributed data to analyze independent samples, and the Kruskal–Wallis nonparametric test was used for nonnormally distributed data. Bonferroni correction was applied for pairwise comparisons. The Bonferroni correction was used to compare the two groups. Enumeration data are presented as frequencies and percentages (%). The chi-square test was used to compare the differences between groups, and the Bonferroni correction was used to compare the differences between two groups via the z test to adjust the p values. Linear-by-linear association tests and the Jonckheere-Terpstra test were used to analyze trends between groups. Correlation analyses were performed using Pearson or Spearman's rank correlation analysis. Independent correlations between the WWI and SF levels were explored using multiple linear regression analyses. Additionally, linear trend analysis was performed by incorporating the median values of each WWI category as continuous variables within the model. The association between the WWI and hyperferritinemia was investigated using binary logistic regression analysis. A significance level of $p < 0.05$ was considered to indicate statistical significance.

RESULTS

Baseline characteristics according to WWI tertile group

Significant increasing trends were observed for age, weight, hypertension prevalence, SBP, WC, HC, BMI, SF, HbA1c, HOMA-IR, FCP, FINS, TG, LDL-C, HDL-C, SFA, VFA, and NAFLD prevalence (all p for trend < 0.05), while a decreasing trend in height and HDL-C was found with an increase in WWI tertile levels (p for trend < 0.05). No significant differences were found in the duration of diabetes, menopausal status, smoking history, drinking history, FPG, or TC levels among the groups ($p > 0.05$) (Table 1).

In male patients, the SF level in the T3 group (228.79 ng/mL) was significantly higher than that in the T1 group (147.00 ng/mL, $p < 0.01$). In females, the median SF concentration in the T3 group was 172.50 ng/mL, marking a significant increase relative to the median concentrations observed in the T1 and T2 groups, which were 84.10 ng/mL and 120.00 ng/mL, respectively (both $p < 0.05$). Regardless of sex, as the WWI tertiles increased, the SF levels exhibited an increasing trend (p for trend < 0.01). Hyperferritinemia prevalence in the T3 group (37.3%) was significantly higher than that in the T1 and T2 groups (27.2% and 18.5%, respectively; both $p < 0.01$), and the T2 group also exhibited a higher prevalence of hyperferritinemia than the T1 group ($p < 0.01$) in male patients. In female patients,

hyperferritinemia prevalence in the T3 group (57.7%) was also significantly higher than that in the T1 group (16.9%, $p < 0.01$) (Table 1).

Comparison of glucose and lipid metabolism indicators between the hyperferritinemia and non-hyperferritinemia groups

The glucose and lipid metabolism indicators, including HbA1c, HOMA-IR, TC, TG, and LDL-C, were compared between the hyperferritinemia and non-hyperferritinemia groups. HbA1c levels were significantly higher in the hyperferritinemia group [8.50 (7.00, 10.70)] than in the non-hyperferritinemia group [8.10 (6.75, 9.40)] ($p=0.001$) indicating poorer glycemic control in individuals with hyperferritinemia. Similarly, HOMA-IR, a marker of insulin resistance, was significantly elevated in the hyperferritinemia group [2.41 (1.51, 4.05)] compared to the non-hyperferritinemia group [2.10 (1.25, 3.71)] ($p=0.017$).

In terms of lipid metabolism, the hyperferritinemia group showed significantly higher TC levels [4.28 (3.67, 4.90)] compared to the non-hyperferritinemia group [4.06 (3.39, 4.76)] ($p=0.004$). TG levels were also significantly elevated in the hyperferritinemia group [1.75 (1.27, 2.61)] compared to the non-hyperferritinemia group [1.50 (1.09, 2.19)] ($p < 0.001$). LDL-C levels followed the same trend, with significantly higher values in the hyperferritinemia group [1.50 (1.09, 2.19)] compared to the non-hyperferritinemia group [2.72 (2.20, 3.31)] ($p=0.001$). (Figure 1)

Correlation analysis of the WWI with other clinical indices

There was a positive correlation between the WWI and SF levels in patients with T2DM ($R=0.263$, $p < 0.01$). The WWI was positively correlated with age, menopause status, hypertension status, BMI, WC, HC, SBP, SF, HbA1c, HOMA-IR, FCP, FINS, TG, LDL-C, SFA, VFA, and the prevalence of NAFLD ($p < 0.05$) but negatively correlated with height and HDL-C ($p < 0.01$). There were no significant correlations between the WWI and duration of diabetes, history of drinking or smoking, weight, DBP, FPG, or TC ($p > 0.05$) (Table 2).

Association between the WWI and SF levels

Multiple linear regression analysis was conducted, employing SF levels as the dependent variable and the WWI as the independent variable. For this analysis, the T1 group was designated as the reference group.

In Model 1, after adjusting for age, sex, duration of diabetes, BMI, SBP, and DBP, the WWI was associated with SF levels in the T2 and T3 groups [$\beta = 0.147$, 95% CI (18.324, 51.604), $p < 0.001$ and $\beta = 0.221$, 95% CI (61.651, 123.151), $p < 0.001$, respectively].

In Model 2, after further adjustments for HbA1c, HOMA-IR, FCP, and FINS, the WWI remained independently correlated with SF levels in the T2 and T3 groups [$\beta = 0.132$, 95% CI (14.912, 47.990), $p < 0.001$ and $\beta = 0.199$, 95% CI (52.329, 113.652), $p < 0.001$, respectively].

After adjusting for TG, HDL-C, LDL-C, SFA, and VFA in Model 3, the WWI maintained a significant, positive, independent correlation with SF levels in both T2 and T3 groups [$\beta = 0.126$, 95% CI (13.642, 46.543), $p < 0.001$ and $\beta = 0.194$, 95% CI (49.914, 112.12), $p < 0.001$, respectively].

As the tertiles of WWI increased, their association with SF became stronger (p for trend < 0.01) (Table 3).

Association between the WWI and hyperferritinemia

Binary logistic regression analysis was conducted to evaluate the association between hyperferritinemia and the WWI. The hyperferritinemia considered as the dependent variable.

In male patients with T2DM, after adjusting for age, duration of diabetes, BMI, and hypertension, the WWI remained a significant independent risk factor for hyperferritinemia [OR = 2.209, 95% CI (1.538, 3.172), $p < 0.001$]. In Model 2, after further adjustments for HbA1c, FINS, FCP, and HOMA-IR, the WWI continued to be an independent risk factor for hyperferritinemia [OR = 2.137, 95% CI (1.478, 3.090), $p < 0.001$]. HbA1c levels also demonstrated a significant positive correlation with hyperferritinemia [OR = 1.139, 95% CI (1.041, 1.245), $p < 0.01$]. Finally, after further adjustments for TG, HDL-C, LDL-C, SFA, and VFA, the WWI remained a significant independent risk factor for hyperferritinemia [OR = 2.091, 95% CI (1.432, 3.054), $p < 0.001$]. HbA1c and TG levels were positively correlated with hyperferritinemia [OR = 1.134, 95% CI (1.035, 1.242), $p < 0.01$; OR = 1.103, 95% CI (1.002, 1.214), $p < 0.05$, respectively]. Age was negatively correlated with hyperferritinemia [OR = 0.960, 95% CI (0.942, 0.979), $p < 0.001$].

In female patients with T2DM, after adjusting for age, diabetes duration, BMI, and hypertension, the WWI also remained an independent risk factor for hyperferritinemia [OR = 3.485, 95% CI (2.210, 5.497), $p < 0.001$]. In Model 2, after further adjustments for HbA1c, FINS, FCP, and HOMA-IR, the WWI continued to be a significant risk factor for hyperferritinemia [OR = 3.425, 95% CI (2.158, 5.438), $p < 0.001$]. HbA1c levels showed a positive correlation with hyperferritinemia [OR = 1.202, 95% CI (1.054, 1.371), $p < 0.01$].

After further adjustment for TG, HDL-C, LDL-C, SFA, and VFA, the WWI remained a significant independent risk factor for hyperferritinemia [OR = 3.248, 95% CI (2.027, 5.204), $p < 0.001$]. HbA1c levels were still correlated with hyperferritinemia [OR = 1.211, 95% CI (1.055, 1.390), $p < 0.01$]. (Table 4).

DISCUSSION

Given the important role of obesity in the occurrence and development of T2DM and the increased understanding of trace elements, several studies have focused on the association between new obesity indicators and metabolic disorders. The WWI was first introduced in 2018 as a novel indicator for assessing central obesity with the advantage of its simple calculation.²¹ Research has shown that the WWI is associated with unhealthy body composition and adverse metabolic outcomes and often serves as a comprehensive predictor of the onset and mortality of metabolic diseases.²² The correlation between the WWI and SF levels in patients with T2DM has not been previously reported currently. The present study revealed a significant positive correlation between the WWI and SF levels in patients with T2DM.

Prior investigations have demonstrated a significant positive association between BMI and SF levels, with a concomitant increase in the prevalence of hyperferritinemia as BMI increases.^{23,24} Nevertheless, the ability of BMI to precisely delineate adipose tissue distribution is limited. BMI cannot identify the presence of abdominal and visceral obesity, which are critical factors for understanding the association between adipose tissue function and iron metabolism. SF levels exhibit positive correlations with adipose tissue insulin resistance, visceral adiposity index, and lipid accumulation.²⁵ Elevated SF concentrations have been found to be positively correlated with the amount of visceral fat tissue.²⁶ Pathophysiological changes in adipose tissue and the activation of inflammatory cell populations, including neutrophils, monocytes, lymphocytes, and tissue-resident macrophages, lead to elevated levels of inflammatory biomarkers in the plasma and an increased number of circulating inflammatory cells. Within adipose tissue, the release of various inflammatory mediators, such as TNF- α and IL-6, further stimulates the production of C-reactive protein. Concurrently, a decrease in the secretion of the anti-inflammatory adipokine adiponectin contributes to the establishment of a proinflammatory environment.²⁷ Additionally, inflammatory and adipose factors may lead to elevated hepcidin levels, thereby enhancing intestinal iron absorption and hepatic release and resulting in increased SF levels.²⁸

Therefore, SF may serve as a pivotal biomarker for the early detection of adipose tissue dysfunction in obese individuals. Our research revealed a gradual increase in SF levels with increasing WWI. After adjusting for potential confounders, the WWI remained an independent risk factor for hyperferritinemia. Our findings are congruent with the outcomes reported by Han et al, indicating that the WWI is positively correlated with SF levels in American adults.²⁹ WWI shows the potential to assess the impact of adipose tissue on SF levels. But further research is needed to compare WWI with BMI and other measures of fat accumulation to confirm its advantage in accuracy.

In patients with T2DM, the association between the WWI and SF primarily focuses on aberrations in glucose and lipid metabolism. In terms of glucose metabolism, an increase in the tertile levels of WWI, HbA1c, FPG, HOMA-IR, FCP, and FINS exhibited an upward trend. Obesity is a well-known risk factor for insulin resistance in individuals with diabetes. Dysfunction within adipose tissue triggers an escalation of proinflammatory cytokines and chemokines, promotes immune cell infiltration, and fosters the accumulation of senescent cells. These alterations culminate in insulin resistance, perpetuation of chronic sterile inflammation, and dysregulation of lipid distribution.³⁰ The contribution of central obesity to diabetes has become a growing concern. As a reliable indicator of central obesity, the WWI has been confirmed by some studies to be an independent risk factor for T2DM. A prospective cohort study conducted in rural Chinese populations revealed a noteworthy correlation between the incidence of T2DM and the WWI. Compared with that in the lowest WWI quartile group, the risk of T2DM in men and women in the highest WWI quartile group increased by 1.604 and 1.899 times, respectively.⁵ A cross-sectional study using NHANES data revealed a notable positive correlation between the WWI and the risk of T2DM in American adults. For each additional increase in the WWI, the risk of developing T2DM increased by 1.14 times (OR = 2.14, 95% CI [1.98, 2.31], $p < 0.001$).³¹

Many previous studies have demonstrated that increased iron is closely associated with adverse health outcomes, particularly with metabolic diseases. In this study, participants were divided into two groups based on serum ferritin levels. Compared to the non-hyperferritinemia group, patients in the hyperferritinemia group exhibited higher levels of HbA1c, HOMA-IR, TC, TG, and LDL-c. A prospective cohort study carried out by Chen et al. among the Chinese population demonstrated that serum ferritin levels were associated with an elevated risk of type 2 diabetes. This association was independent of traditional risk factors, the hazard ratios for incident diabetes corresponding to one standard deviation increase in serum ferritin levels were 1.17.³² Another cross-sectional study based on a cohort of 1,928

Middle Eastern individuals showed a significant association between serum ferritin levels and dyslipidemia, impaired glucose tolerance, and metabolic syndrome.³³ Iron load may play a role in the development of T2DM by affecting insulin secretion, insulin resistance, and other interconnected pathways.^{13,34-36} Iron overload can increase the production of reactive oxygen species (ROS), lead to oxidative stress damage, and induce oxidative damage in islet β cells, resulting in a decrease in insulin synthesis and secretion.³⁷ Additionally, the coupling of glucose metabolism and insulin release also depends on the involvement of iron. Iron, an important coenzyme in the tricarboxylic acid cycle, participates in the glycosylation reduction pathway and affects glucose metabolism and insulin secretion.³⁸ In contrast, SF has a direct lipolytic effect on adipocytes, which leads to an excess of circulating free fatty acids and insulin resistance.³⁹

Furthermore, patients with a higher WWI exhibited a higher prevalence of metabolic disorders, including hypertension, abnormal blood lipid levels, and NAFLD. Metabolic syndrome (MetS) is a multifaceted metabolic disorder characterized by the concurrent presence of abdominal obesity, insulin resistance, hyperglycemia, dyslipidemia, and hypertension.⁴⁰ Epidemiological studies have revealed a correlation between dysregulated iron homeostasis and the presence of MetS. Systemic iron overload, although relatively mild, is frequently observed in patients with MetS.⁴¹ Clinically, it presents with the hallmark features of liver iron deposition and increased levels of non-transferrin-bound iron and SF.⁴² In the absence of traditional causes of iron overload, such as genetic hemochromatosis, chronic hemolysis, or long-term blood transfusions, patients may present with systemic iron accumulation and multiple components of MetS, a condition known as dysmetabolic iron overload syndrome (DIOS).⁴³ DIOS highlights the interaction between iron metabolism and MetS, indicating that iron metabolism disorders may be a potential factor in the pathogenesis of MetS, with a mutual influence between them.⁴⁴

This study revealed a more pronounced correlation between the WWI and hyperferritinemia in female patients with T2DM, indicating the presence of sex disparities, which may be related to the fact that the WWI of female patients included in this study was significantly higher than that of male patients, and most of the participants in this study were postmenopausal women. Postmenopausal women experience alterations in hormone levels, lipid metabolism, and visceral obesity. Compared to premenopausal women and men, postmenopausal women exhibited higher levels of ROS. Increased oxidative stress may serve as a predisposing factor for the development of iron metabolism disorders in individuals who are more susceptible to such conditions.⁴⁵

Finally, some limitations should be addressed in future studies. First, this was a cross-sectional study, and the observed correlation between SF levels and the WWI in individuals with T2DM does not imply causation. Second, the participants of the study were patients admitted with T2DM and a variety of complex treatment drugs, which may have introduced medication-related effects. Third, this study did not include a control group of healthy individuals, making it difficult to ascertain the correlation between SF levels and the WWI in the general population. Our study did not include inflammatory factors such as white blood cells, CRP, IL-6, etc. In future studies, we should gain a deeper understanding of the mechanism by which inflammation plays a role in disease development. Last, the dietary patterns of the study participants were not investigated, which means that the influence of diet-related factors, such as prolonged consumption of red meat, cannot be ruled out.

Consequently, a positive correlation was observed between the WWI and SF levels among patients with T2DM. These findings offer a new perspective on managing T2DM patients. For patients with higher WWI, it is essential to pay attention to their iron metabolism status and promptly implement interventions to prevent the onset of hyperferritinemia. Patients who are already experiencing hyperferritinemia, in addition to diabetes-specific treatments, should focus on weight management and fat distribution to improve iron metabolism status.

Conclusion

This study indicated a positive correlation between the WWI and SF levels among individuals with T2DM. The WWI is an independent risk factor for hyperferritinemia, especially in female patients with T2DM. In the management of type 2 diabetes, it is of utmost importance for patients to pay close attention to weight management, particularly the distribution of body fat. Simultaneously, iron metabolism should by no means be overlooked.

CONFLICT OF INTEREST AND FUNDING DISCLOSURE

The authors have no relevant financial or nonfinancial interests to disclose.

This study was funded by the National Natural Science Foundation of China (No. 81960155 and No. 82360161), the Science Foundation of Gansu Provincial Maternity and Child-care Hospital (GMCCH2024-3-2), and the Lanzhou Science and Technology Bureau (2022-3-46).

REFERENCES

1. Sun H, Saeedi P, Karuranga S, et al. IDF Diabetes Atlas: Global, regional and country-level diabetes prevalence estimates for 2021 and projections for 2045. *Diabetes Res Clin Pract.* 2022;183:109119. doi: 10.1016/j.diabres.2023.110945.
2. Ong KL, Stafford LK, Mclaughlin SA, Boyko EJ, Vollset SE, Smith AE, et al. Global, regional, and national burden of diabetes from 1990 to 2021, with projections of prevalence to 2050: a systematic analysis for the Global Burden of Disease Study 2021. *The Lancet.* 2023;402(10397):203–34.
3. Wu H, Ballantyne CM. Skeletal muscle inflammation and insulin resistance in obesity. *J Clin Invest.* 2017;127(1):43-54. doi: 10.1172/JCI88880. PubMed PMID: 28045398.
4. Kim KJ, Son S, Kim KJ, Kim SG, Kim NH. Weight-adjusted waist as an integrated index for fat, muscle and bone health in adults. *J Cachexia Sarcopenia Muscle.* 2023;14(5):2196-2203. doi: 10.1002/jcsm.13302.
5. Yu S, Wang B, Guo X, Li G, Yang H, Sun Y. Weight-Adjusted-Waist Index Predicts Newly Diagnosed Diabetes in Chinese Rural Adults. *J Clin Med.* 2023;12(4). doi: 10.3390/jcm12041620.
6. Zhang D, Shi W, Ding Z, Park J, Wu S, Zhang J. Association between weight-adjusted-waist index and heart failure: Results from National Health and Nutrition Examination Survey 1999-2018. *Front Cardiovasc Med.* 2022;9:1069146. doi: 10.3389/fcvm.2022.1069146.
7. Cai S, Zhu T, Ding Y, et al. The relationship between the weight-adjusted-waist index and left ventricular hypertrophy in Chinese hypertension adults. *Hypertens Res.* 2023;46(1):253-260. doi: 10.1038/s41440-022-01075-z.
8. Ding Y, Xu Z, Zhou X, Luo Y, Xie R, Li Y. Association between weight-adjusted-waist index and the risk of hyperuricemia in adults: a population-based investigation. *Front Endocrinol (Lausanne).* 2023;14:1236401. doi: 10.3389/fendo.2023.1236401.
9. Ye J, Hu Y, Chen X, et al. Association between the weight-adjusted waist index and stroke: a cross-sectional study. *BMC Public Health.* 2023;23(1):1689. doi: 10.1186/s12889-023-16621-8.
10. Fang H, Xie F, Li K, Li M, Wu Y. Association between weight-adjusted-waist index and risk of cardiovascular diseases in United States adults: a cross-sectional study. *BMC Cardiovasc Disord.* 2023;23(1):435. doi: 10.1186/s12872-023-03452-z.
11. Xie F, Xiao Y, Li X, Wu Y. Association between the weight-adjusted-waist index and abdominal aortic calcification in United States adults: Results from the national health and nutrition examination survey 2013-2014. *Front Cardiovasc Med.* 2022;9:948194. doi: 10.3389/fcvm.2022.948194.
12. Wang X, Zhou Y, Min J, Wang F. Zooming in and out of ferroptosis in human disease. *Front Med.* 2023;17(2):173-206. doi: 10.1007/s11684-023-0992-z.
13. Harrison AV, Lorenzo FR, McClain DA. Iron and the Pathophysiology of Diabetes. *Annu Rev Physiol.* 2023;85:339-362. doi: 10.1146/annurev-physiol-022522-102832.
14. Zhan Y, Tang Z, Yu J. Serum ferritin, diabetes, diabetes control, and insulin resistance. *Acta Diabetol.* 2014;51(6):991-998. doi: 10.1007/s00592-014-0656-1.

15. Cho MR, Park JK, Choi WJ, Cho AR, Lee YJ. Serum ferritin level is positively associated with insulin resistance and metabolic syndrome in postmenopausal women: A nationwide population-based study. *Maturitas*. 2017;103:3-7. doi: 10.1016/j.maturitas.2017.06.004.
16. Podmore C, Meidtner K, Schulze MB, et al. Association of Multiple Biomarkers of Iron Metabolism and Type 2 Diabetes: The EPIC-InterAct Study. *Diabetes Care*. 2016;39(4):572-581. doi: 10.2337/dc15-0257.
17. Tan L, Zhou Q, Liu J, Liu Z, Shi R. Association of iron status with non-alcoholic fatty liver disease and liver fibrosis in US adults: a cross-sectional study from NHANES 2017-2018. *Food Funct*. 2023;14(12):5653-5662. doi: 10.1039/d2fo04082d.
18. Kernan KF, Carcillo JA. Hyperferritinemia and inflammation. *Int Immunol*. 2017;29(9):401-409. doi: 10.1093/intimm/dxx031.
19. Sandnes M, Ulvik RJ, Vorland M, Reikvam H. Hyperferritinemia-A Clinical Overview. *J Clin Med*. 2021;10(9).
20. Beaton MD, Adams PC. Treatment of hyperferritinemia. *Ann Hepatol*. 2012;11(3):294-300. doi: 10.3390/jcm10092008.
21. Park Y, Kim NH, Kwon TY, Kim SG. A novel adiposity index as an integrated predictor of cardiometabolic disease morbidity and mortality. *Sci Rep*. 2018;8(1):16753. doi: 10.1038/s41598-018-35073-4.
22. Kim NH, Park Y, Kim NH, Kim SG. Weight-adjusted waist index reflects fat and muscle mass in the opposite direction in older adults. *Age Ageing*. 2021;50(3):780-786. doi: 10.1093/ageing/afaa208.
23. Castiella A, Zapata E, Urreta I, et al. Body mass index and alcohol consumption are directly related with liver steatosis. Results from a prospective study of patients referred for hyperferritinemia. *Ann Hepatol*. 19(6):697. doi: 10.1016/j.aohep.2020.07.012
24. Alam F, Memon AS, Fatima SS. Increased Body Mass Index may lead to Hyperferritinemia Irrespective of Body Iron Stores. *Pak J Med Sci*. 2015;31(6):1521-1526. doi: 10.12669/pjms.316.7724.
25. Ding X, Bian N, Wang J, et al. Serum Ferritin Levels Are Associated with Adipose Tissue Dysfunction-Related Indices in Obese Adults. *Biol Trace Elem Res*. 2023;201(2):636-643. doi: 10.1007/s12011-022-03198-3.
26. Zimiao C, Dongdong L, Shuoping C, et al. Correlations Between Iron Status and Body Composition in Patients With Type 2 Diabetes Mellitus. *Front Nutr*. 2022;9:911860. doi: 10.3389/fnut.2022.911860.
27. Moore Heslin A, O'Donnell A, Buffini M, et al. Risk of Iron Overload in Obesity and Implications in Metabolic Health. *Nutrients*. 2021;13(5). doi: 10.3390/nu13051539.
28. González-Domínguez Á, Visiedo-García FM, Domínguez-Riscart J, González-Domínguez R, Mateos RM, Lechuga-Sancho AM. Iron Metabolism in Obesity and Metabolic Syndrome. *Int J Mol Sci*. 2020;21(15). doi: 10.3390/ijms21155529.
29. Han H, Ni P, Zhang S, et al. The association of body mass index and weight waist adjustment index with serum ferritin in a national study of US adults. *Eur J Med Res*. 2023;28(1):374. doi: 10.1186/s40001-023-01343-9

30. Stout MB, Justice JN, Nicklas BJ, Kirkland JL. Physiological Aging: Links Among Adipose Tissue Dysfunction, Diabetes, and Frailty. *Physiology* (Bethesda). 2017;32(1):9-19. doi: 10.1152/physiol.00012.2016.
31. Zheng D, Zhao S, Luo D, et al. Association between the weight-adjusted waist index and the odds of type 2 diabetes mellitus in United States adults: a cross-sectional study. *Front Endocrinol (Lausanne)*. 2023;14:1325454. doi: 10.3389/fendo.2023.1325454.
32. Chen L, Li Y, Zhang F, Zhang S, Zhou X, Ji L. Elevated serum ferritin concentration is associated with incident type 2 diabetes mellitus in a Chinese population: A prospective cohort study. *Diabetes Res Clin Pract*. 2018;139:155-162. doi: 10.1016/j.diabres.2018.03.001.
33. Al Akl NS, Khalifa O, Errafii K, Arredouani A. Association of dyslipidemia, diabetes and metabolic syndrome with serum ferritin levels: a middle eastern population-based cross-sectional study. *Sci Rep*. 2021;11(1):24080. doi: 10.1038/s41598-021-03534-y.
34. Fillebeen C, Lam NH, Chow S, Botta A, Sweeney G, Pantopoulos K. Regulatory Connections between Iron and Glucose Metabolism. *Int J Mol Sci*. 2020;21(20). doi: 10.3390/ijms21207773.
35. Krisai P, Leib S, Aeschbacher S, et al. Relationships of iron metabolism with insulin resistance and glucose levels in young and healthy adults. *Eur J Intern Med*. 2016;32:31-37. doi: 10.1016/j.ejim.2016.03.017.
36. Liu J, Li Q, Yang Y, Ma L. Iron metabolism and type 2 diabetes mellitus: A meta-analysis and systematic review. *J Diabetes Investig*. 2020;11(4):946-955. doi: 10.1111/jdi.13216.
37. Miao R, Fang X, Zhang Y, Wei J, Zhang Y, Tian J. Iron metabolism and ferroptosis in type 2 diabetes mellitus and complications: mechanisms and therapeutic opportunities. *Cell Death Dis*. 2023;14(3):186. doi: 10.1038/s41419-023-05708-0.
38. Marku A, Galli A, Marciani P, Dule N, Perego C, Castagna M. Iron Metabolism in Pancreatic Beta-Cell Function and Dysfunction. *Cells*. 2021;10(11). doi: 10.3390/cells10112841.
39. Rumberger JM, Peters T, Jr., Burrington C, Green A. Transferrin and iron contribute to the lipolytic effect of serum in isolated adipocytes. *Diabetes*. 2004;53(10):2535-2541. doi: 10.2337/diabetes.53.10.2535.
40. Fahed G, Aoun L, Bou Zerdan M, et al. Metabolic Syndrome: Updates on Pathophysiology and Management in 2021. *Int J Mol Sci*. 2022;23(2). doi: 10.3390/ijms23020786.
41. Pitchika A, Kühn JP, Schipf S, et al. Hepatic steatosis and hepatic iron overload modify the association of iron markers with glucose metabolism disorders and metabolic syndrome. *Liver Int*. 2021;41(8):1841-1852. doi: 10.1111/liv.14868.
42. Ameka M, Hasty AH. Paying the Iron Price: Liver Iron Homeostasis and Metabolic Disease. *Compr Physiol*. 2022;12(3):3641-3663. doi: 10.1002/cphy.c210039.
43. Rametta R, Dongiovanni P, Pelusi S, et al. Hcpidin resistance in dysmetabolic iron overload. *Liver Int*. 2016;36(10):1540-1548. doi: 10.1111/liv.13124.
44. Hilton C, Sabaratnam R, Drakesmith H, Karpe F. Iron, glucose and fat metabolism and obesity: an intertwined relationship. *Int J Obes (Lond)*. 2023;47(7):554-563. doi: 10.1038/s41366-023-01299-0.

45. Leanza G, Conte C, Cannata F, et al. Oxidative Stress in Postmenopausal Women with or without Obesity. *Cells*. 2023;12(8). doi: 10.3390/cells12081137.

Not Proof Read

Table 1. Baseline characteristics according to WWI tertile grouping

Index	T1 (n = 309)	T2 (n = 269)	T3 (n = 369)	p	p for trend
Age (years)	57.17±11.08	58.14±9.90	61.71±10.33 ^{***}	<0.01	<0.01
Sex (male/female)	222/83	202/67	213/156	<0.01	0.025
Duration (years)	10.00 (4.00,15.00)	10.00 (5.00,14.50)	10.00 (5.00,15.00)	0.868	0.672
Menopause (%)	81.70%	88.10%	91.70%	0.077	0.025
Smoking (%)	24.30%	32.30%	21.70%	0.080	0.359
Drinking (%)	17.40%	20.40%	12.20%	0.016	0.054
SBP (mmHg)	139.00 (123.00,153.00)	140.00 (127.00,155.00)	142.00 (127.00,160.00) [*]	0.026	<0.01
DBP (mmHg)	83.00 (74.00,90.00)	85.00 (78.00,95.00) [*]	85.00 (74.00,95.00)	0.043	0.121
Hypertension (%)	43.60%	49.10%	56.40%	<0.01	<0.01
Weight (kg)	71.47±11.25	72.22±9.59	74.64±12.59 ^{***}	<0.01	<0.01
Hight (cm)	1.70 (1.64,1.75)	1.69 (1.63,1.75)	1.66 (1.60,1.72) ^{***}	<0.01	<0.01
BMI (kg/m ²)	24.94±3.20	25.24±2.73	27.16±3.64 ^{***}	<0.01	<0.01
WC (cm)	87.21±7.35	93.96±6.36 ^{**}	99.97±8.25 ^{***}	<0.01	<0.01
HC (cm)	91.66±6.40	94.48±5.98 ^{**}	97.48±7.48 ^{***}	<0.01	<0.01
SF (ng/mL)					
Male	147.00(88.73,256.25)	228.79(138.85,335.75) ^{**}	228.79(169.85,360.00) ^{**}	<0.001	<0.01
Female	84.10(51.00,121.00)	120.00(64.90,201.00) [*]	172.50(116.50,228.79) ^{***}	<0.001	<0.01
Hyperferritinemia					
Male	18.5% ^{**}	27.2% ^{**}	37.3% ^{**##}	<0.001	<0.01
Female	16.9%	27.2%	57.7% ^{**}	<0.001	<0.01
HbA1c (%)	7.90 (6.60,9.30)	8.40 (7.00,9.80)	8.40 (7.00,9.95) [*]	0.034	0.018
FPG (mmol/L)	8.23 (6.36,10.28)	8.34(6.73,11.43)	8.45(6.65,10.55)	0.240	0.171
HOMA-IR	1.86 (1.20,3.29)	2.33 (1.41,3.83) [*]	2.34 (1.44,4.04) ^{**}	<0.01	<0.01
FCP (ng/mL)	1.20 (0.84,1.55)	1.29 (0.97,1.63) [*]	1.34 (0.93,1.72) ^{**}	<0.01	<0.01
FINS (mU/L)	5.82 (3.73,8.70)	6.60(4.21,8.66)	6.88 (4.27,10.16) [*]	0.027	<0.01
TC (mmol/L)	4.13±1.06	4.28±1.03	4.18±1.01	0.220	0.557
TG (mmol/L)	1.43 (1.02,2.23)	1.69 (1.18,2.41) [*]	1.63 (1.20,2.27) [*]	0.010	0.012
HDL-C (mmol/L)	1.01 (0.89,1.17)	0.98 (0.85,1.12)	0.94 (0.82,1.13) ^{**}	<0.001	<0.01
LDL-C (mmol/L)	2.69 (2.14,3.19)	2.81 (2.35,3.39)	2.91 (2.32,3.48) ^{**}	<0.01	<0.01
SFA (cm ²)	171.00 (134.00,210.50)	179.00 (150.00,218.50)	184.00 (141.00,229.00) [*]	0.018	<0.01
VFA (cm ²)	111.00 (81.50,149.50)	119.00 (90.50,147.50)	124.00 (94.00,158.50) ^{**}	0.012	<0.01
NAFLD (%)	33.10%	46.80%	46.10%	<0.01	<0.01

BMI: body mass index; WC: waist circumference; HC: hip circumference; SF: serum ferritin; SBP: systolic blood pressure; DBP: diastolic blood pressure; HbA1c: glycated hemoglobin; FPG: fasting glucose; HOMA-IR: insulin resistance index; FINS: fasting insulin; FCP: fasting C-peptide; TC: total cholesterol; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; SFA: subcutaneous fat area; VFA: visceral fat area; NAFLD: nonalcoholic fatty liver disease. ^{**}p < 0.01 signifies a comparison with group T1, while

^{*}p < 0.05 indicates a comparison with group T1

^{##}p < 0.01 signifies a comparison with group T2

[#]p < 0.05 indicates a comparison with group T2

Table 2. Correlation analysis of the WWI and other clinical indices

INDEX	R	<i>p</i>
Age (years)	0.204	<0.001
Sex (male/female)	-.0172	<0.001
Duration (years)	0.026	0.430
Menopause (%)	0.140	0.015
Smoking (%)	-0.037	0.259
Drinking (%)	-0.059	0.068
Hypertension (%)	0.093	0.004
Weight (kg)	0.055	0.093
Hight (cm)	-0.235	<0.001
BMI (kg/m ²)	0.268	<0.001
WC (cm)	0.614	<0.001
HC (cm)	0.335	<0.001
SBP (mmHg)	0.075	0.022
DBP (mmHg)	0.024	0.457
SF (ng/mL)	0.263	<0.001
HbA1c (%)	0.073	0.024
FPG (mmol/L)	0.054	0.099
HOMA-IR	0.114	<0.001
FCP (ng/ml)	0.091	0.005
FINS (mU/L)	0.082	0.012
TC (mmol/L)	0.021	0.515
TG (mmol/L)	0.087	0.007
HDL-C (mmol/L)	-0.106	0.001
LDL-C (mmol/L)	0.114	<0.001
SFA (cm ²)	0.078	0.016
VFA (cm ²)	0.083	0.011
NAFLD (%)	0.099	0.002

BMI: body mass index; WC: waist circumference; HC: hip circumference; SBP: systolic blood pressure; DBP: diastolic blood pressure; SF: serum ferritin; HbA1c: glyated hemoglobin; FPG: fasting glucose; HOMA-IR: insulin resistance index; FCP: fasting C-peptide; FINS: fasting insulin; TC: total cholesterol; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; SFA: subcutaneous fat area; VFA: visceral fat area; NAFLD: nonalcoholic fatty liver disease.

Table 3. Multiple linear regression analysis of the relationships between the WWI and SF levels

WWI group	β	t	<i>p</i>	95% CI	<i>p</i> for trend
Model 1					
T1	0 ^a				
T2	0.147	4.124	<0.001	(18.324, 51.604)	
T3	0.221	5.897	<0.001	(61.651, 123.151)	<0.001
Model 2					
T1	0 ^a				
T2	0.132	3.732	<0.001	(14.912, 47.990)	
T3	0.199	5.312	<0.001	(52.329, 113.652)	<0.001
Model 3					
T1	0 ^a				
T2	0.126	3.590	<0.001	(13.642, 46.543)	
T3	0.194	5.112	<0.001	(49.914, 112.12)	<0.001

a: T1 as the reference group

In linear regression analysis, β represents standardized regression coefficients, t refers to test statistics, and CI indicates the confidence interval.

Model 1: Adjusted for age, sex, duration of diabetes, BMI, SBP, and DBP; Model 2: Adjusted for Model 1 + HbA1c, HOMA-IR, FCP, Fins; Model 3: Model 2 + TG, HDL-C, LDL-C, VFA, SFA.

Table 4. Binary logistic regression analysis of the association between hyperferritinemia and the WWI

	Male			Female		
	OR	95% CI	<i>p</i>	OR	95% CI	<i>p</i>
Model 1						
WWI	2.209	(1,538, 3.172)	<0.001	3.485	(2.210, 5.497)	<0.001
Age	0.955	(0,937, 0.974)	<0.001	0.999	(0.971, 1.028)	0.953
Duration	0.992	(0,962, 1.022)	0.588	0.966	(0.928, 1.005)	0.087
BMI	1.037	(0,977, 1.100)	0.229	0.953	(0.886, 1.024)	0.191
Hypertension	0.758	(0,510, 1.126)	0.170	0.946	(0.560, 1.600)	0.837
Model 2						
WWI	2.137	(1,478, 3.090)	<0.001	3.425	(2.158, 5.438)	<0.001
Age	0.960	(0,942, 0.979)	<0.001	0.998	(0.969, 1.028)	0.907
Duration	0.995	(0,964, 1.027)	0.753	0.977	(0.936, 1.020)	0.297
BMI	1.028	(0,964, 1.096)	0.402	0.950	(0.877, 1.029)	0.209
Hypertension	0.743	(0,496, 1.114)	0.150	0.966	(0.566, 1.650)	0.899
HbA1c	1.139	(1,041, 1.245)	0.004	1.202	(1.054, 1.371)	0.006
HOMA-IR	0.920	(0,793, 1.066)	0.267	0.876	(0.716, 1.071)	0.195
Fins	1.046	(0,974, 1.123)	0.216	1.023	(0.932, 1.123)	0.629
FCP	1.186	(0,860, 1.635)	0.299	1.189	(0.767, 1.842)	0.439
Model 3						
WWI	2.091	(1,432, 3.054)	<0.001	3.248	(2.027, 5.204)	<0.001
Age	0.965	(0,945, 0.984)	<0.001	0.995	(0.964, 1.026)	0.731
Duration	0.997	(0,965, 1.030)	0.855	0.978	(0.937, 1.022)	0.324
BMI	1.056	(0,963, 1.157)	0.245	0.979	(0.876, 1.095)	0.709
Hypertension	0.771	(0,512, 1.161)	0.214	0.962	(0.559, 1.658)	0.89
HbA1c	1.134	(1,035, 1.242)	0.007	1.211	(1.055, 1.390)	0.006
HOMA-IR	0.906	(0,780, 1.052)	0.195	0.880	(0.715, 1.084)	0.23
Fins	1.050	(0,977, 1.129)	0.182	1.016	(0.923, 1.119)	0.745
FCP	1.121	(0,795, 1.579)	0.515	1.342	(0.852, 2.113)	0.204
TG	1.103	(1,002, 1.214)	0.046	0.816	(0.650, 1.025)	0.081
HDL	0.392	(0,144, 1.071)	0.068	0.369	(0.123, 1.109)	0.076
LDL	1.000	(0,985, 1.015)	0.989	1.341	(0.947, 1.898)	0.098
SFA	0.996	(0,991, 1.002)	0.203	0.996	(0.991, 1.001)	0.099
VFA	1.000	(0,995, 1.005)	0.958	1.002	(0.995, 1.009)	0.511

Model 1: adjusted for age, duration, BMI, hypertension (0 = no, 1 = yes); Model 2: Model 1 + HbA1c, Fins, FCP, HOMA-IR; Model 3: Model 2 + TG, HDL-C, LDL-C, SFA, VFA.

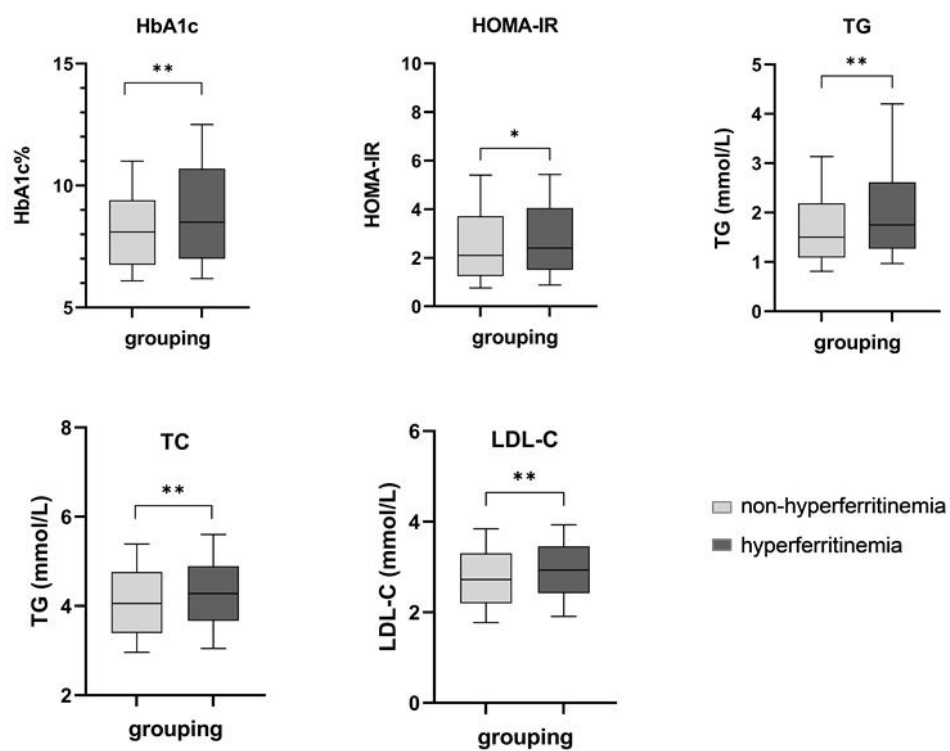


Figure 1. Comparison of glucose and lipid metabolism indicators between the hyperferritinemia and non-hyperferritinemia groups. HbA1c: glycated hemoglobin; HOMA-IR: insulin resistance index; TC: total cholesterol; TG: triglycerides; LDL-C: low-density lipoprotein cholesterol. ** represents $p < 0.01$, * represents $p < 0.05$