BMC Endocrine Disorders

RESEARCH

Open Access

Association between waist-to-hip ratio and testosterone in Chinese men with young-onset type 2 diabetes: a cross-sectional study

Yanan Luo[1], Hongya Shao[2], Qiuping Zhang[2], Fupeng Liu[2,3], Mei Zhang[2,3], Yanhong Zhang[2,3], Yaru Wang[4], Hui Pan[5], Bo Ban[2,3*] and Yanying Li[2,3*]

Abstract

Background Low testosterone levels is associated with higher cardiovascular risk in men with diabetes. However, there are few studies on testosterone levels and the factors affecting them in patients with young-onset diabetes (YOD). The objective of this study was to investigate the correlation between waist-to-hip ratio (WHR) and testosterone levels in men diagnosed with YOD.

Research design and methods This cross-sectional study involved 547 male patients with type 2 diabetes mellitus (T2DM) from the Endocrinology Department of the Affiliated Hospital of Jining Medical University. The participants were divided into two groups: a young-onset diabetes (YOD) group and a late-onset diabetes (LOD) group. Anthropometric measurements, including height, weight, waist circumference, and hip circumference, were recorded. Additionally, fasting blood samples were collected to assess various parameters, such as sex hormone levels and lipid profiles. The association between WHR and testosterone levels was analyzed by univariate linear regression and multivariable linear regression analysis.

Results Five hundred forty-seven patients with type 2 diabetes and aged 50.3 ± 12.1 years were enrolled in the study. One hundred ninety-three patients were assigned to the YOD group based on a diagnosis age of 40 years or younger, while the remaining 354 patients were assigned to the LOD group. The testosterone was significantly lower in the YOD group compare to the LOD group ($P = 0.049$), and the WHR had an independent effect on testosterone in men with the YOD group (β=-4.67, $P = 0.0251$), but there was no evidence of such an association in the LOD group (β =-1.13, $P = 0.4608$).

Conclusions According to our results, male patients with YOD exhibited lower testosterone levels compared to those with LOD. Furthermore, their testosterone levels were significantly negatively correlated with WHR. These findings indicate that it is more necessary to screen for testosterone in obese patients with YOD and that improving obesity, especially abdominal obesity, may help to interrupt the vicious cycle of low testosterone-obesity-insulin resistance-hyperglycemia-low testosterone.

*Correspondence:
Bo Ban
banbo2011@163.com
Yanying Li
liyanying510@126.com

Full list of author information is available at the end of the article

Keywords Testosterone, Waist-to-hip ratio, Type 2 diabetes, Young-onset diabetes, Late-onset diabetes

Introduction

In 2021, approximately 537 million adults worldwide were living with diabetes, accounting for 10.5% of the global population [1]. Type 2 diabetes constitutes the predominant form of diabetes, affecting 90–95% of individuals. It has emerged as one of the most pressing health challenges of the 21st century, with considerable societal implications worldwide [2]. Type 2 diabetes is traditionally recognized as a disease that occurs in middle-aged and elderly individuals [3]. Recent research has found that the prevalence and incidence of young-onset diabetes (YOD) are increasing in many parts of the world, with the most rapid rise occurring in Asia, where one in five individuals with diabetes is diagnosed below the age of 40 [4]. Research shows that in the same age group, individuals with early - onset type 2 diabetes have higher risks of cardiovascular and kidney diseases and mortality than those with later - onset type 2 diabetes [5, 6]. Low testosterone is associated with higher cardiovascular risk in men with diabetes [7]. Therefore, it is important to explore serum testosterone levels and the factors affecting them in patients with YOD.

In systematic reviews and meta-analyses, it has been suggested that total testosterone levels are consistently lower in diabetic men than in non-diabetic controls in all individual studies, with a mean pooled difference of 2.66 nmol/liter [8]. In addition, a cross-sectional study from China found that testosterone levels were significantly lower in young-onset diabetic patients compared to those with late-onset diabetic patients (11.93 ± 0.57 vs. 14.04 ± 0.56 nmol/L, $P = 0.011$) [9]. Analysis of longitudinal data from the Flory Adelaide Study on Male Aging Study showed an increased incidence of diabetes in men with serum testosterone concentrations below 16 nmol/L (461 ng/dL) [10]. Ding's analysis showed that men with serum testosterone concentrations greater than 15.5 nmol/L (447 ng/dL) had a 42% lower risk of developing type 2 diabetes compared to men with serum testosterone concentrations of 15.5 nmol/L or less [8]. In a recent randomized controlled trial, 1007 men with serum testosterone levels of 14.0 nmol/L or lower, who did not have pathological hypogonadism but exhibited impaired glucose tolerance or were newly diagnosed with type 2 diabetes, were randomly allocated to either a placebo or a testosterone treatment group. The study suggests that a two-year testosterone treatment reduced the proportion of participants with type 2 diabetes beyond the effects of a lifestyle program. Moreover, the testosterone group demonstrated significantly greater reductions in total and abdominal fat mass, as well as increases in lean body mass [11]. Kristy's mediation analysis found that the effects of testosterone treatment was found to be mediated by changes in fat mass, abdominal fat, skeletal muscle mass, grip strength, SHBG, and E2, but predominantly by changes in fat mass [12].

Low serum testosterone concentrations are common in men with obesity [13]. It was reported that diet-induced weight loss is also associated with a modest reversal of reduced serum testosterone in men with obesity with no recognized pathological hypothalamo-pituitary-testicular (HPT) disorders [14]. In randomized controlled trials, testosterone treatment has consistently led to modest reductions in fat mass and increases in lean mass and body composition changes that are expected to be metabolically favorable [15].

Previous studies have identified distinct incidence, prevalence, morbidity, mortality and pathogenesis of YOD compared to those of late-onset diabetes (LOD), but there are few studies exploring the testosterone levels and the related factors. At present, this study defined YOD as being diagnosed with T2DM at 40 years or younger. This research aimed to investigate the relationship between the waist-to-hip ratio (WHR) and testosterone in men with YOD.

Methods

Participants

This cross-sectional study was performed by reviewing the medical records of male patients with type 2 diabetes from the Department of Endocrinology, Affiliated Hospital of Jining Medical University between November 2019 and April 2021. In total, 547 patients were enrolled and divided into either the YOD group ($n = 193$) or the LOD group ($n = 354$) according to the age at which they were diagnosed with T2DM. The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of Affiliated Hospital of Jining Medical University.

Inclusion and exclusion criteria

The subjects were selected based on the diagnostic criteria for type 2 diabetes established by the World Health Organization (WHO). We classified patients as having YOD if they were diagnosed with T2DM at 40 years or younger, and as having LOD if they were diagnosed after the age of 40. The exclusion criteria were as follows: diabetic ketoacidosis, diabetic hyperosmolar hyperglycemic state, diabetic lactic acidosis, diabetic hypoglycemia, acute myocardial infarction, acute trauma, severe infections, and diseases that may affect total testosterone levels, such as severe hepatic dysfunction, renal dysfunction, pituitary-hypothalamic disorders, and malignant

tumors of the reproductive system. Additionally, patients using medications that interfere with testosterone were excluded. Overall, 629 patients were assessed during the study period, 547 of whom were eligible for inclusion in our study, as detailed in the flow chart (Fig. 1).

Lifestyle

Alcohol consumption was defined as consuming at least 30 g of alcohol per week for at least one year. Individuals who had consumed at least 100 cigarettes during their lifetime were classified as cigarette smokers.

Anthropometric measurements

Height and weight were measured using a standard procedure with the participants wearing no shoes or coats. Height was measured using a stadiometer (Nantong Best Industrial Co., Ltd., Jiangsu, China), which is accurate to 0.1 cm. An electronic scale (Wuxi Weigher Factory Co., Ltd., Jiangsu, China) was used to measure weight to the

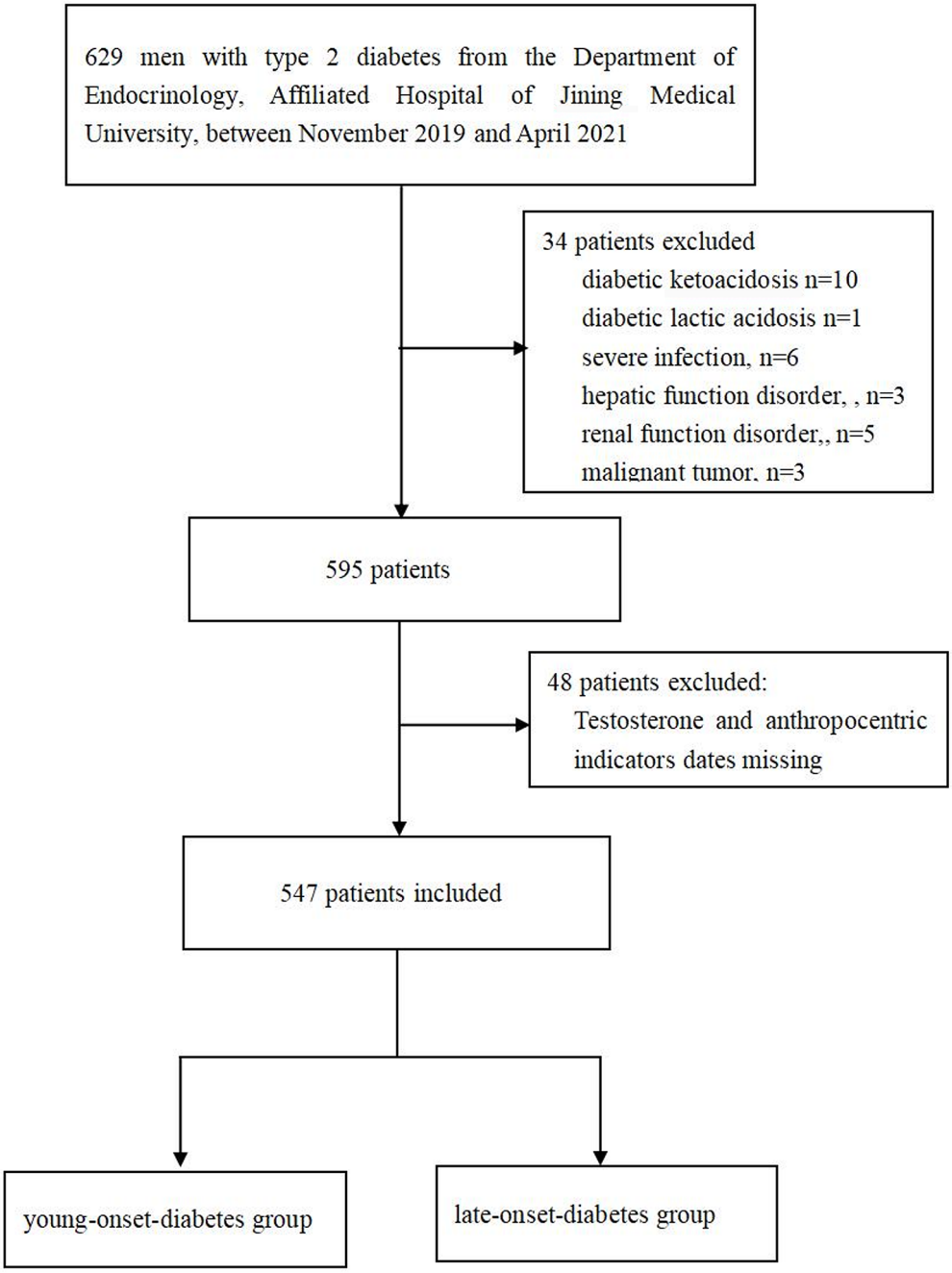

Fig. 1 Flow chart of study participants

nearest 0.1 kg. Body mass index (BMI) = weight(kg)/height(m)^2. According to the BMI criteria of the Chinese population, BMI ≥ 28 were defined as obese [16]. Abdominal circumference(AC), waist circumference(WC) and hip circumference(HC) were measured using a non-stretchable tape measure with allowable error ranges of 0.1 cm. Waist-to-hip ratio(WHR) = WC(cm)/HC (cm). The waist-to-hip ratio ≥ 0.90 was defined as abdominal obesity in men [17]. Blood pressure measurements were performed after a 10-min rest with the patitents in a seated position, and the blood pressure was measured three times on the right arm with an electronic sphygmomanometer (Omron HBP-1300, Dalian, China). The average of the three measurements was used in the analyses.

Laboratory measurements

Blood samples were collected in the morning (7–9 am) after an overnight fast. Levels of gonadotropins, including testosterone(T), estradiol(E2), progesterone(P), prolactin, follicle-stimulating hormone (FSH), luteinizing hormone (LH), free T3 (FT3), free T4 (FT4), thyroid-stimulating hormone (TSH), and C-peptide, were measured using a luminescence immunoassay system (Cobas c 602, Roche; Shanghai, China). Testosterone exhibited intra- and inter-assay coefficients of variation (CVs) of 2.7% and 25%, respectively. Hypogonadism was defined as total testosterone less than 3 ng/ml. Assessments of liver function (including levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and gamma-glutamyl transferase (GGT)), kidney function (including creatinine (Cr), blood urea nitrogen (BUN), and uric acid (UA)), lipid profiles (including total cholesterol (TC)), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG)), and fasting plasma glucose (FPG) were performed with a biochemical autoanalyzer (Cobas c702, Roche; Shanghai, China). Hemoglobin A1c (HbA1c) was measured with high-performance liquid chromatography (Akeley HA8180, Japan; Shanghai, China).

Statistical analysis

All statistical analyses were performed with R statistical software (https://www.r-project.org) and Empower Stats (http://www.empowerstats. com, X&Y solutions, Inc. Boston MA). Continuous variables with a normal or approximately normal distribution are presented as mean ± standard deviation, while those with non-normal distribution are presented as median and interquartile range. Categorical variables are described by the number and percentage. The characteristics of the study population were compared using independent samples t-test, Kruskal-Wallis test or $\chi 2$ test, as appropriate. A univariate analysis model was used to examine whether the WHR and other anthropometric and biochemical variables were associated with testosterone. Then, we explored the relationship between the WHR and testosterone via linear regression. Finally, we used a multivariate linear regression model to assess the independent association between the WHR and testosterone. *P* values (two-tailed) below 0.05 were considered statistically significant.

Results

Clinical and laboratory characteristics of the subjects

Five hundred forty-seven patients with type 2 diabetes and aged 50.3 ± 12.1 years were enrolled in the study. The demographic, anthropometric, and clinical characteristics of the 547 patients stratified according to diagnosis age are shown in Table 1. One hundred ninety-three patients were assigned to the YOD group based on a diagnosis age of 40 years or younger, and the other 354 patients were assigned to the LOD group. The patients in the YOD group showed lower testosterone ($p = 0.049$), follicle-stimulating hormone (FSH) ($p < 0.001$), luteinizing hormone (LH) ($p < 0.001$) and high-density lipoprotein cholesterol (HDL-C) ($p = 0.0408$) values than those in the LOD group. Moreover, BMI ($p < 0.001$), diastolic blood pressure (DBP) ($p < 0.001$), c-peptide (C-P) ($p = 0.001$), triglycerides (TG) ($p < 0.001$), total cholesterol (TC) ($p < 0.001$), uric acid (UA) ($p < 0.001$) and estradiol (E2) ($p = 0.011$) were higher in the YOD group than in the LOD group. However, there was no difference in the duration of diabetes, waist circumference, abdominal circumference, hip circumference, WHR, lipoprotein cholesterol (LDL-C) or hemoglobin A1c (HbA1c) between the two groups.

Associations of anthropometric and biochemical variables with testosterone

Univariate linear regression analysis was performed to determine the relationships between each variable and testosterone. As shown in Table 2, BMI, waist circumference, abdominal circumference, hip circumference, WHR, TG, HbA1C and C-P were significantly negatively associated with testosterone. It was also found that age, duration of diabetes and HDL were significantly positively associated with testosterone.

Associations between the WHR and testosterone in different groups

We performed univariate subgroup analysis by linear regression with testosterone as the dependent variable and WHR as the independent variable to explore the relationship between WHR and testosterone in different groups. As shown in Table 3, a significant negative association was found between the WHR and testosterone in the YOD group (β=-8.47, 95% CI -11.85–-5.08; $p < 0.001$) and in the late-onset diabetes group. The multivariate linear regression model demonstrated an independent

Table 1 Baseline characteristics of the participants

Variables	YOD (Diagnosed age < = 40years)	LOD (Diagnosed age > 40years)	*P*-value
N	193	354	
Age (years)	42.2 ± 9.3	58.9 ± 8.9	< 0.001
Duration (years)	6.0 (1.0,12.0)	8.0 (3.0,11.0)	0.489
SBP (mmHg)	134.3 ± 17.4	135.6 ± 16.5	0.421
DBP (mmHg)	83.8 ± 12.0	80.4 ± 11.2	< 0.001
BMI (kg/m^2)	27.0 ± 3.6	26.0 ± 2.9	< 0.001
BMI subgroup			0.002
BMI ≥ 28.0	72 (37.3%)	87 (24.6%)	
BMI<28.0	121 (62.7%)	267 (75.4%)	
WC (cm)	96.4 ± 9.1	95.3 ± 7.9	0.145
AC (cm)	98.3 ± 8.9	97.3 ± 8.7	0.199
HC (cm)	101.0 ± 6.6	100.9 ± 6.3	0.893
WHR	0.95 ± 0.1	0.94 ± 0.1	0.065
WHR subgroup			0.760
WHR ≥ 0.9	159 (83.2%)	278 (82.5%)	
WHR<0.9	32 (16.8%)	59 (17.5%)	
ALT(U/L)	23.6 (15.9,38.6)	17.6 (13.1,24.3)	< 0.001
Cr(umol/L)	63.4 (57.0,73.5)	64.7 (56.8,75.1)	0.375
TG (mmol/L)	2.2 (1.3,3.3)	1.3 (0.9,1.8)	< 0.001
TC (mmol/L)	4.6 (3.9,5.4)	4.2 (3.6,5.0)	< 0.001
HDL-C (mmol/L)	1.1 (0.9,1.2)	1.1 (1.0,1.3)	0.0408
LDL-C (mmol/L)	2.8 ± 0.9	2.7 ± 0.9	0.105
UA (umol/L)	332.0 (279.0,400.8)	310.0 (254.3,358.5)	< 0.001
HbA1c (%)	8.9 (7.7,10.3)	8.5 (7.3,10.0)	0.075
C-P (ng/ml)	2.4 (1.9,3.2)	2.0 (1.6,2.6)	0.002
T (ng/ml)	3.8 ± 1.5	4.0 ± 1.5	0.049
T subgroup			0.038
T<3.0(ng/ml)	64 (33.2%)	88 (24.9%)	
T ≥ 3.0(ng/ml)	129 (66.8%)	266 (75.1%)	
E2 (pg/ml)	40.0 (34.61,50.8)	39.4 (32.7,47.0)	0.011
LH (mIU/ml)	4.7 (3.2,6.6)	5.3 (3.7,7.8)	< 0.001
FSH (mIU/ml)	5.8 (3.9,8.2)	8.4 (5.6,12.6)	< 0.001
FT3 (pmol/L)	4.7 (4.3,5.1)	4.5 (4.0,5.0)	0.574
FT4 (pmol/L)	17.0 ± 2.9	17.1 ± 4.7	0.860
TSH (mIU/L)	1.7 (1.2,2.8)	1.8 (1.1,2.7)	0.119
Smoke			0.002
No	94 (48.7%)	125 (35.4%)	
Yes	99 (51.3%)	228 (64.6%)	
Drinking			0.013
No	112 (58.0%)	165 (46.9%)	
Yes	81 (42.0%)	187 (53.1%)	

YOD: young-onset diabetes; LOD: late-onset diabetes; SBP: systolic blood pressure; DBP: diastolic blood pressure; BMI: body mass index; WC: waist circumference; AC: abdomen circumference; HC: hip circumference; WHR: waist to hip ratio; ALT: aminotransferase; TG: triglyceride; TC: total cholesterol; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; UA: uric acid; HbA1c: hemoglobin A1c; E2: estradiol; LH: luteinizing hormone; FSH: follicle-stimulating hormone; FT3: free T3, FT4: free T4, TSH: thyroid-stimulating hormone; C-P: c-peptide; T: testosterone;

association between the WHR and testosterone after adjusting for age, duration of diabetes, BMI, TG, TC, UA, HbA1C, C-P, smoking and alcohol consumption; however, there was no such association in the late-onset diabetes group (β =-1.13, 95% CI: --4.13, 1.87, $p = 0.461$), as shown in Table 3.

Discussion

In this retrospective cross-sectional study, we analyzed the association between the WHR and testosterone in YOD patients. The results showed that the testosterone of the patients with YOD was significantly lower than that of the late-onset diabetes group which is consistent with the outcome of Hu et al. [9]. Further regression

Table 2 Association between testosterone and different variables

	β (95% CI)	*P*-value
Age (years)	0.01 (0.00, 0.02)	0.0255
Duration (years)	0.02 (0.01, 0.04)	0.0112
SBP (mmHg)	-0.00 (-0.01, 0.00)	0.4804
DBP (mmHg)	-0.01 (-0.02, 0.00)	0.0784
BMI (kg/m2)	-0.14 (-0.17, -0.10)	< 0.0001
WC (cm)	-0.06 (-0.07, -0.04)	< 0.0001
AC (cm)	-0.05 (-0.06, -0.04)	< 0.0001
HC (cm)	-0.05 (-0.07, -0.03)	< 0.0001
WHR	-5.83 (-7.92, -3.75)	< 0.0001
TG (mmol/L)	-0.09 (-0.13, -0.04)	0.0002
TC (mmol/L)	0.05 (-0.05, 0.15)	0.3380
HDL (mmol/L)	1.28 (0.82, 1.73)	< 0.0001
LDL (mmol/L)	0.14 (-0.01, 0.28)	0.0603
UA (umol/L)	-0.00 (-0.00, 0.00)	0.0991
HbA1c (%)	-0.07 (-0.13, -0.01)	0.0272
C-P (ng/ml)	-0.31 (-0.42, -0.20)	< 0.0001

SBP: systolic blood pressure; DBP: diastolic blood pressure; BMI: body mass index; WC: waist circumference; AC: abdomen circumference; HC: hip circumference; WHR: waist to hip ratio; TG: triglyceride; TC: total cholesterol; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; UA: uric acid; HbA1c: hemoglobin A1c; C-P: c-peptide; $P < 0.05$ is considered to be statistically significant

analysis showed that the WHR was independently associated with testosterone.

Adiposity is an established risk factor for type 2 diabetes [2]. WHR serves as a reliable indicator of central obesity, and it excels in predicting the development of type 2 diabetes compared to other traditional obesity-associated metrics [18]. According to the criteria for abdominal obesity defined by a waist-to-hip ratio equal to or greater than 0.9 [17]. In this study, the incidence of abdominal obesity was 82.6% in the total population, while the incidence rates in the YOD and LOD groups were 83.3% and 82.2%, respectively. Although the difference was not statistically significant, the trend of increased adiposity in patients with YOD was obvious and consistent with reports in the literature [19]. Prevailing evidence suggests a similar hallmark etiology for YOD and LOD: β-cell dysfunction, insulin resistance and other obesity-driven mechanisms. However, it was reported that the prevalence of obesity among young adults with type 2 diabetes is much greater than that in older adults with type 2 diabetes [20]. In this study, we found that BMI was higher in YOD patients than in LOD patients. The incidence of obesity was 37.3% in the YOD group and 24.6% in the LOD group. These findings implicate obesity-related mechanisms as key players in the development of the young-onset phenotype; however, the precise pathways remain elusive.

Reduced serum testosterone is common in men with type 2 diabetes [21]. A previous study established that between 25% and 50% of men with type 2 diabetes have reduced testosterone levels [22]. It is well documented that serum testosterone was inversely related to age in men; however, lowered testosterone was not restricted to the aged, with 20% of young men with diabetes also having low circulating testosterone, and low testosterone predicted mortality, independent of multiple confounders [23]. In our study, testosterone was lower in the YOD group than in the LOD group, and the same trend was observed for follicle-stimulating hormone and luteinizing hormone. According to the criteria for low testosterone [24], our data showed that the prevalence of low testosterone in the total population was 27.8%, while the incidence of low testosterone was 33.2% in the YOD group and 24.9% in the LOD group.

Observational studies consistently show that obesity is a major determinant of low testosterone [25]. Our study indicated that testosterone was negatively related to the WHR by univariate analysis, whereas a further subgroup analysis revealed that testosterone was negatively related to the WHR in the YOD group, while the effect was weakened in the LOD group. Furthermore, multivariate linear regression analysis demonstrated that the WHR had an independent effect on testosterone in patients with YOD but not in those with LOD.

Based on our findings, although the specific mechanism for the relationship between the WHR and testosterone remains unclear, we considered that it may differ based on the degree of obesity, especially abdominal obesity. This perspective is supported by an observational study. The inverse relationships between testosterone

Table 3 Multiple regression analysis of WHR and testosterone in different groups

Group	Crude Model		Adjusted Mode I		Adjusted Mode II		Adjusted Mode III	
	β (95% CI)	*P* value	β (95% CI)	*P* value	β (95% CI)	*P* value	β (95% CI)	*P* value
YOD	-8.47 (-11.85, -5.08)	< 0.0001	-7.55 (-10.90, -4.20)	< 0.0001	-4.43 (-8.44, -0.42)	0.0320	-4.55 (-8.56, -0.53)	0.0278
LOD	-4.24 (-6.88, -1.59)	0.0018	-5.06 (-7.78, -2.34)	0.0003	-0.74 (-3.76, 2.28)	0.6319	-1.31 (-4.42, 1.79)	0.4085

Model I Adjustment variables: smoke; drinking; age; duration

Model II Adjustment variables: BMI; TG; TC; UA; HbA1c; C-P

Model III Adjustment variables: smoke; drinking; age; duration, BMI; TG; TC; UA; HbA1c; C-P

YOD: young-onset diabetes; LOD: late-onset diabetes; BMI: body mass index; TG: triglyceride; TC: total cholesterol; HDL-C: high-density lipoprotein cholesterol; UA: uric acid; HbA1c: hemoglobin A1c; C-P: c-peptide; $P < 0.05$ is considered to be statistically significant

and measures of insulin resistance lost significance after adjustment for visceral fat and/or truncal fat, suggesting that visceral fat and/or truncal fat play an important role in testosterone levels. It was suggested that insulin resistance is the likely common mediator of lowered testosterone levels in men with type 2 diabetes. The current evidence is consistent with the findings of a bidirectional relationship between obesity and testosterone, which creates a self-perpetuating cycle promoting insulin resistance. Thus, increased visceral fat leads to increased secretion of proinflammatory cytokines, estradiol, insulin, and leptin, all of which may inhibit the activity of the hypothalamopituitary gonadal axis at multiple levels. Low testosterone further increases fat accumulation, insulin resistance (IR), and deterioration of glycemic control, creating a vicious cycle.

Some limitations in our study must be noted. First, we did not conduct sexual function-related questionnaires, such as questions about erectile dysfunction and decreased libido. We intend to analyze sexual function in men with diabetes at different ages at diagnosis in a prospective study. Second, due to the cross-sectional design, the present findings showed a negative association between the WHR and testosterone only in men with YOD. Prospective studies are also required to further observe the results after normalization of blood glucose using medications.

In summary, men in the YOD group presented a lower testosterone level than those in the LOD group, and their testosterone was negatively associated with the WHR, suggesting that it is more necessary to screen for testosterone in obese patients with YOD and that improving obesity, especially abdominal obesity, may help to interrupt the vicious cycle of low testosterone-obesity-insulin resistance-hyperglycemia-low testosterone. However, our findings should be verified by larger studies including different ethnic populations.

Acknowledgements
We would like to thank the biochemical laboratory of the Affiliated Hospital of Jining Medical University for measuring all biochemical indicators. No potential conflicts of interest exist.

Author contributions
Yanan Luo, Yanying Li and Bo Ban contributed to the study conception and design and the data acquisition, analysis, and interpretation and drafted and critically revised the manuscript; Yaru Wang and Fupeng Liu contributed to the study conception and design and the data analysis and interpretation and critically revised the manuscript; Mei Zhang, Qiuping Zhang, Yanhong Zhang, and Hui Pan contributed to the study design and data interpretation and critically revised the manuscript. All authors provided final approval and agree to be accountable for all aspects of the work.

Funding
This research received no external funding.

Data availability
The datasets analysed during the current study available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This study was approved by the Ethics Committee of the Affiliated Hospital of Jining Medical University(2022-11-C012). Written informed consent was obtained from each participant before the study, which was approved by the Affiliated Hospital of Jining Medical University(2020-BS-008). Clinical trial number: not applicable.

Consent for publication
Written informed consent for publication was obtained.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of Clinical Medicine, Jining Medical University, Jining, Shandong 272067, China
[2]Department of Endocrinology, Affiliated Hospital of Jining Medical University, 89 Guhuai Road, Jining, Shandong 272029, China
[3]Chinese Research Center for Behavior Medicine in Growth and Development, 89 Guhuai Road, Jining, Shandong 272029, China
[4]Zibo Central Hospital, 55 Liuquan Road, Zhangdian District, Zibo, Shandong 272029, China
[5]Key Laboratory of Endocrinology of National Health and Family Planning Commission, Department of Endocrinology, Peking Union Medical College Hospital, Chinese Academy of Medical Science and Peking Union Medical College, Beijing 100730, China

Received: 10 September 2024 / Accepted: 7 January 2025
Published online: 06 March 2025

References

1. Magliano DJ, Boyko EJ, I.D.F. D.A.t.e.s. committee, IDF Diabetes Atlas, in Idf diabetes atlas. 2021, International Diabetes Federation © International Diabetes Federation, 2021.: Brussels.
2. Zheng Y, Ley SH, Hu FB. Global aetiology and epidemiology of type 2 diabetes mellitus and its complications. Nat Rev Endocrinol. 2018;14(2):88–98.
3. Misra S, et al. Current insights and emerging trends in early-onset type 2 diabetes. Lancet Diabetes Endocrinol. 2023;11(10):768–82.
4. Luk AOY, Kong APS, Basu A. Young-onset diabetes, nutritional therapy and novel insulin delivery systems: a report from the 21(St) Hong Kong Diabetes and Cardiovascular Risk factors - East meets West Symposium. Diabet Med. 2020;37(8):1234–43.
5. Huo X, et al. Risk of non-fatal cardiovascular diseases in early-onset versus late-onset type 2 diabetes in China: a cross-sectional study. Lancet Diabetes Endocrinol. 2016;4(2):115–24.
6. Morton JI, et al. The association between age of onset of type 2 diabetes and the long-term risk of end-stage kidney disease: a National Registry Study. Diabetes Care. 2020;43(8):1788–95.
7. Kouidrat Y, et al. High prevalence of erectile dysfunction in diabetes: a systematic review and meta-analysis of 145 studies. Diabet Med. 2017;34(9):1185–92.
8. Ding EL, et al. Sex differences of endogenous sex hormones and risk of type 2 diabetes: a systematic review and meta-analysis. JAMA. 2006;295(11):1288–99.
9. Hu Y et al. Low total testosterone levels in men with newly diagnosed early-onset type 2 diabetes: a cross-sectional study in China. J Diabetes Res. 2023;2023:2082940.
10. Atlantis E, et al. Predictive value of serum testosterone for type 2 diabetes risk assessment in men. BMC Endocr Disord. 2016;16(1):26.
11. Wittert G, et al. Testosterone treatment to prevent or revert type 2 diabetes in men enrolled in a lifestyle programme (T4DM): a randomised, double-blind, placebo-controlled, 2-year, phase 3b trial. Lancet Diabetes Endocrinol. 2021;9(1):32–45.
12. Robledo KP, et al. Mediation analysis of the testosterone treatment effect to prevent type 2 diabetes in the testosterone for prevention of type 2 diabetes mellitus trial. Eur J Endocrinol. 2023;188(7):613–20.

13. Grossmann M, Wittert GA. Testosterone in prevention and treatment of type 2 diabetes in men: focus on recent randomized controlled trials. Ann N Y Acad Sci. 2024;1538(1):45–55.
14. Grossmann M. Low testosterone in men with type 2 diabetes: significance and treatment. J Clin Endocrinol Metab. 2011;96(8):2341–53.
15. Corona G, et al. Testosterone supplementation and body composition: results from a meta-analysis of observational studies. J Endocrinol Invest. 2016;39(9):967–81.
16. China D. o.M.A.N.H.C.o.t.P.s.R.o., Chinese guidelines for the clinical management of obesity(2024 Edition). Med J Peking Union Med. pp. 1–60. https://doi.org/10.12290/xhyxzz.2024-0918
17. Nishida C, Ko GT, Kumanyika S. Body fat distribution and noncommunicable diseases in populations: overview of the 2008 WHO Expert Consultation on Waist Circumference and Waist-Hip ratio. Eur J Clin Nutr. 2010;64(1):2–5.
18. Sadeghi E, et al. Novel anthropometric indices for predicting type 2 diabetes mellitus. BMC Public Health. 2024;24(1):1033.
19. Magliano DJ, et al. Young-onset type 2 diabetes mellitus - implications for morbidity and mortality. Nat Rev Endocrinol. 2020;16(6):321–31.
20. Wilmot E, Idris I. Early onset type 2 diabetes: risk factors, clinical impact and management. Ther Adv Chronic Dis. 2014;5(6):234–44.
21. Gianatti EJ, Grossmann M. Testosterone deficiency in men with type 2 diabetes: pathophysiology and treatment. Diabet Med. 2020;37(2):174–86.
22. Morton A. Frequent occurrence of hypogonadotropic hypogonadism in type 2 diabetes. J Clin Endocrinol Metab. 2005;90(3):1903. author reply 1903.
23. Tint AN, et al. Association of sex hormone-binding globulin and free testosterone with mortality in men with type 2 diabetes mellitus. Eur J Endocrinol. 2016;174(1):59–68.
24. Colleluori G, et al. Testosterone therapy effects on bone mass and turnover in hypogonadal men with type 2 diabetes. J Clin Endocrinol Metab. 2021;106(8):e3058–68.
25. Caliber M, Saad F. Testosterone therapy for prevention and reversal of type 2 diabetes in men with low testosterone. Curr Opin Pharmacol. 2021;58:83–9.

Publisher's note