## RESEARCH



# Developing a risk model for early diagnosis of metabolic syndrome in Chinese adults aged 40 years and above based on BMI/HDL-C: a cross-sectional study



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## Abstract

**Background** This study aimed to compare the diagnostic accuracy of four indicators, including waist-to-height ratio (WHTR), vascular adiposity index (VAI), TG/HDL-C, and BMI/HDL-C for metabolic syndrome (MS) in Chinese adults aged 40 years and above. Additionally, the study aimed to develop an efficient diagnostic model displayed by a nomogram based on individual's BMI and circulating HDL-C level.

**Methods** A cross-sectional study was conducted on 699 participants aged 40 years and above. Quartiles of BMI/ HDL-C, TG/HDL-C, VAI, and WHTR were used as independent variables, and metabolic syndrome was used as the dependent variable. Logistic regression was conducted to explore the impact of each parameter on the risk of MS. The areas under the receiver operating characteristics were compared to determine the accuracy of the indicators in diagnosing MS in the participants. Logistic regression was run to construct the nomograms, and the performance of the nomogram was assessed by a calibration curve.

**Results** MS subjects had higher levels of BMI, BFM, PBF, VFA, AMC, WC, SCR, TG, and insulin, but lower LDH and HDL-C levels than the subjects without MS. The BMI/HDL-C ratio was positively correlated with the prevalence of MS and its components. The final diagnostic model included five variables: gender, BFM, WC, TG, and BMI/HDL-C. The model showed good calibration and discrimination power with an AUC of 0.780. The cut-off value for the nomogram was 0.623 for diagnosing MS.

**Conclusions** BMI/HDL-C ratio was an independent risk factor for MS in Chinese adults. BMI/HDL-C was significantly correlated with MS and its components. BMI/HDL-C was the most powerful diagnostic indicator compared to other

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indicators, including TG/HDL-C, VAI and WHTR for diagnosing MS. The nomogram drawn based on the diagnostic model provided a practical tool for diagnosing MS in Chinese adults.

Keywords BMI/HDL-C ratio, Metabolic syndrome, Nomogram, Diagnostic model

## Background

Metabolic syndrome (MS) is composed of a series of common metabolic disorders, including obesity, hyperglycemia, hypertension, and disturbance of lipid metabolism [1]. Currently, it is estimated that a quarter of the global population has MS, which affects over a billion people [2-4]. In 2017, the reported prevalence of MS in China was approximately 31.3% [5]. MS causes varying degrees of damage to the cardiovascular, digestive, and endocrine systems of the body, leading to a higher mortality rate among patients compared to those without MS [6]. Because of the severe impact of MS on health, early diagnosis and prediction of MS have become especially crucial. The traditional diagnosis of MS mainly relies on clinical physical examination and biochemical testing, which makes it difficult to screen sensitively and accurately diagnose in the community residents [7]. Meanwhile, the clinical community has not widely adopted emerging early diagnostic methods such as metabolomics and mass spectrometry [8]. Therefore, early screening, detection, and health management to prevent the progression of MS is becoming an essential public health concern.

Many blood biochemical and anthropometry indicators have been used to predict MS for early diagnosis and prevention [9]. Besides, the emerging discipline developed in the last century, metabolomics, was also used for diagnosing MS [10, 11]. Abnormal glucose and lipid metabolism are the typical characteristics of MS [12]. Moreover, insulin resistance was closely related to the pathology of MS. Homeostasis model assessment, including insulin resistance (HOMA-IR) [13], triglyceride and glucose index (TyG) [14], was commonly used to predict MS [15]. The World Health Organization (WHO) also proposed that MS needs to focus on insulin resistance and hyperglycemia [16]. Additionally, the TG/HDL-C index was also considered a classic diagnostic of MS, which fully considered the lipid metabolites including triglycerides and high-density lipoprotein cholesterol (HDL-C) [17].

Anthropometry indicators were widely used to diagnose MS and assess the prevalence of risk factors for their convenient access, and body mass index (BMI) and waist circumference (WC) are extensively used for the diagnosis of MS. However, the inaccuracy of measurement reduced the prediction efficiency as individuals with higher muscle mass were also classified as overweight or obese according to the cutoff value of BMI and WC. Studies had confirmed a positive association existed between neck circumference and the risk of MS, and neck circumference was considered as a surrogate parameter for BMI and WC in diagnosing MS [18, 19]. Besides, increasing studies try to explore the indicators that combine multiple factors to enhance diagnostic stability, such as Waist-to-Height Ratio (WHTR) and Visceral Adiposity Index (VAI) [20], etc. WHTR was considered to have better diagnostic power on cardiovascular metabolismrelated risk factors than waist circumference and BMI [21]. The VAI index, an effective indicator reflecting visceral fat accumulation, was reported to be associated with various components of MS [22]. Although BMI is a useful indicator for evaluating health status due to its simplicity, it cannot directly reflect the degree of metabolic disorder [23].

Due to the complex components of metabolic syndrome, it is necessary to construct a reliable diagnostic model using routine clinical indicators. To explore an indicator with better diagnostic performance for MS, our study proposed a new diagnostic indicator that combines body composition with blood biochemistry (BMI/ HDL-C). Moreover, different from previous studies that only constructed indicators, our study also evaluated the diagnostic power with conventional diagnostic indicators including TG/HDL-C, WHTR, and VAI.

## **Materials and methods**

#### Participants

The adults aged 40 years and above who accepted health examination were collected at Suzhou Science and Technology City Hospital and Dong Zhu Health Service Center in 2020. A total of 699 participants, including 215 males and 484 females, were recruited. Three participants were included in the baseline data analysis but were excluded from the subsequent studies for lacking blood biochemical indicators. The study protocol was approved by the Committee on Medical Ethics of Capital Medical University (No. 2012SY23), and the study procedures followed the ethical standards of the Helsinki Declaration of 1975. Informed consent was signed by all participants.

The MS was defined according to the Clinical guidelines for prevention and treatment of type 2 diabetes mellitus in China (2022 version) [24]. The MS defined by Chinese Diabetes Society (CDS) guidelines consists of three or more of the following: (1) Abdominal Obesity: Abdominal obesity was defined as WC  $\geq$  90 cm for males, WC  $\geq$  85 cm for females. (2) Hyperglycemia: FPG  $\geq$  6.1 mmol/L or OGTT  $\geq$  7.8 mmol/L or those who have been diagnosed with diabetes and treated. (3) Hypertension: Blood pressure  $\geq$  130/85 mmHg (1 mmHg=0.133 kPa) or those who have been diagnosed with hypertension and treated. (4) Hypertriglyceridemia: Triglycerides level of  $\geq$ 1.70 mmol/L was used as the cut-off level. (5) Hypohigh-density lipoproteinemia (HHDL): HDL-Cholesterol level of <1.04 mmol/L was used as the cut-off value.

## **Data collection**

All data were collected by trained medical staff according to standard operating procedures. A questionnaire survey (see Additional file 1) was conducted at the beginning of the examination to collect basic demographic information, disease history (past medical history, medication history, and medication compliance), family disease history, and personal lifestyle. Physical examination and biochemical testing were conducted. All the information was input into a database, and strict quality control was carried out.

#### Physical examination and blood sample collection

Anthropometric measurement was conducted according to the guidelines of the WHO [25]. The height was measured by a standard height gauge (Seca). The weightmachine was required to calibrate the scale daily (Seca). The waist circumference was measured using a plastic tape measure, taken at the midpoint between the lower margin of the last palpable rib and the top of the iliac crest. The body composition, including body fat mass (BFM), body fat percentage (BFP), and visceral fat area (VFA), was measured by the Body Composition Analyzer (INBODY S10). The upper arm of the subject was raised at an angle of about 45 degrees, the palm of the hand was upward and clenched the fist and bent the elbow forcefully, then the measurer stood on the side and measured the muscle circumference of the upper arm by circling the tape around the thickest part of the biceps of the upper arm to obtain the value of arm muscle circumference (AMC). A sphygmomanometer was used to measure blood pressure twice in a sitting position.

Subjects were required to fast for at least 12 h. Venous blood samples (5 ml) were collected from 6:30-10:00 a.m. After centrifugation (480 g, 20 min), the plasma was separated. Plasma triglyceride (TG), creatinine (SCR) and lactate dehydrogenase (LDH) were measured by ILAB600 clinical chemistry analyzer (Instrumentation Laboratory, Lexington, WI, USA). Plasma HDL cholesterol (HDL-C) was measured using a commercially available assay from the Instrumentation Laboratory (Lexington, WI, United States). Low-density lipoprotein cholesterol (LDL-C) was calculated based on the Friedewald formula (FF) [26]. The patient took 75 g of glucose orally after fasting blood collection and drew venous blood two hours after meals to measure insulin levels. Plasma insulin concentrations were measured according to the manufacturer's instructions (ELISA; Mercodia Ltd).

Body mass index (BMI) was classified as normal 18.5–23.9 kg/m<sup>2</sup>, overweight 24–27.9 kg/m<sup>2</sup> and obese  $\geq$  28 kg/m<sup>2</sup> [27]. Educational level was defined as illiteracy or primary school level and secondary school or above. Smoking status was categorized as non-smoker and smoker (including ex-smoker and current smoker).

Composite indicators are calculated according to the formula. TG/HDL-C: triglyceride to high-density lipoprotein cholesterol ratio. WHTR: waist-to-height ratio. VAI: Visceral Adiposity Index, was calculated as:

$$VAI \ (males) = \frac{WC}{39.68 + (1.88 \times BMI)} \times \frac{TG}{1.03} \times \frac{1.31}{HDL - c}$$
$$VAI \ (females) = \frac{WC}{36.58 + (1.89 \times BMI)} \times \frac{TG}{0.81} \times \frac{1.52}{HDL - c}$$

WC was measured in centimeters, BMI in kg/m<sup>2</sup>, TG and HDL-C in mmol/L.

## Statistical analysis

SPSS 26.0 and R 4.2.2 were used for statistical analysis. Measurement data were expressed as M (SD). If the data conformed to the normal distribution, the t-test was used to compare the differences between groups; otherwise, the rank sum test was applied to compare the differences between groups. The categorical data were expressed in numbers and proportions, and the  $\chi^2$  test was used to compare differences between groups. The Cramer's V coefficient was used to evaluate the correlation strength when there was statistical significance in the  $\chi^2$  test, and 0.1 < Cramer's V < 0.3 indicates a weak intensity correlation;  $0.3 \le$  Cramer's V<0.5 indicates a moderate intensity correlation; Cramer's V≥0.5 indicates a high intensity correlation. Cramer's V was represented by the pound sign (#). Quartiles of BMI/HDL-C, TG/HDL-C, VAI, and WHTR were used as independent variables, and metabolic syndrome was used as the dependent variable. Logistic regression was conducted to explore the impact of each parameter on the risk of MS and to construct diagnostic models for MS. After adjusting for confounding factors, five variables were included in the final model, including gender, BFM, WC, TG, and BMI/ HDL-C, and the OR value and 95% CI of MS risk were analyzed. The calibration curve and the receiver operating characteristic curve (ROC) of the prediction model were plotted to evaluate its diagnostic value. P < 0.05 was considered statistically significant.

#### Results

#### Demographic characteristics of the participants

The demographic characteristics and plasma parameters of the participants were shown in Tables 1 and 2. The percentage of females was higher in the MS group than that in the non-MS group (P<0.05). The subjects with MS had

Table 1	Demographic characteristics and body composition	on
paramet	ers of the participants	

Indices	Non-MS	MS	P-value
	(n=370)	(n = 329)	
Age (y), M (SD)	64.7 (8.4)	64.2 (9.0)	0.877
Gender, n (%)			0.014
Male	129 (34.9)	86 (26.1)	
Female	241 (65.1)	243 (73.9)	
Educational level, n (%)			0.024
Illiteracy <sup>a</sup> or primary school	261 (70.5)	256 (78.3)	
Secondary school or above <sup>b</sup>	109 (29.5)	71 (21.7)	
Smoking, n (%)			
Never smoking	300 (81.1)	278 (84.5)	0.271
Ex-smoker and current smoker	70 (18.9)	51 (15.5)	
BMI (kg/m <sup>2</sup> ), M (SD)	25.0 (2.7)	27.0 (3.3)	< 0.001
Normal, n (%)	119 (32.2)	54 (16.4)	< 0.001
Overweight, n (%)	194 (52.4)	152 (46.2)	
Obese, n (%)	57 (15.4)	123 (37.4)	
BFM (kg), M (SD)	18.5 (5.5)	23.0 (6.3)	< 0.001
PBF (%), M (SD)	29.6 (7.5)	33.9 (6.7)	0.035
VFA (cm <sup>2</sup> ), M (SD)	85.5 (32.5)	110.9 (37.3)	< 0.001
AMC (cm), M (SD)	26.7 (3.1)	27.5 (2.4)	< 0.001
WC (cm), M (SD)	83.0 (7.2)	89.3 (9.0)	< 0.001
WHTR (M (SD))	0.5 (0.1)	0.6 (0.1)	< 0.001
VAI (M (SD))	1.5 (0.9)	3.3 (2.8)	< 0.001

Data were expressed as M (SD) or n (%). Parameters including BMI, BFM, PBF, VFA, AMC, and WC, were compared using t-tests or rank sum tests. Gender, educational level, smoking status, and BMI were compared using Chi-square tests. P < 0.05 was considered significant. Ex-smoker: an individual who has given up cigarettes before our investigation. BMI, Body Mass Index; BFM, Body Fat Mass; PBF, Percent Body Fat; VFA, Visceral Fat Area; AMC, Arm Muscle Circumference; WC, Waist Circumference; WHTR, waist-to-height ratio; VAI, visceral adiposity index; non-MS: non-metabolic syndrome; MS: metabolic syndrome. a: non-formal education; b: including junior high school, high school, technical secondary school, junior college, undergraduate, graduate, and above

 Table 2
 Comparison of plasma parameters between MS and non-MS subjects

Indices	Non-MS	MS	P-value
M (SD)	( <i>n</i> = 370)	(n = 329)	
SCR (mmol/L)	70.8 (19.1)	71.3 (21.2)	< 0.001
LDH (mmol/L)	194.3 (50.2)	186.3 (36.1)	0.043
HDL-C(mmol/L)	1.4 (0.3)	1.2 (0.3)	< 0.001
TG (mmol/L)	1.2 (0.6)	2.2 (1.4)	< 0.001
LDL-C (mmol/L)	2.8 (0.8)	2.9 (0.8)	0.416
Insulin (mIU/L)	47.8 (40.1)	53.0 (38.3)	0.022
BMI/HDL-C	18.2 (4.3)	23.4 (5.7)	< 0.001
TG/HDL-C	0.9 (0.6)	2.0 (1.6)	< 0.001

Data were expressed as M (SD). Serum parameters including SCR, LDH, TG, HDL-C, and Insulin were compared using t-tests or rank sum tests. SCR, serum creatinine; LDH, lactate dehydrogenases; HDL-C, high-density lipoprotein cholesterol; TG, triglyceride; LDL-C, low-density lipoprotein cholesterol; Insulin, 2-hour postprandial serum insulin; non-MS: non-metabolic syndrome; MS: metabolic syndrome

higher levels of BMI, BFM, PBF, VFA, AMC, WC, SCR, TG, and insulin, but lower LDH and HDL-C levels than the subjects without MS (P<0.05). The subjects with MS had a lower education level than the subjects without MS

(P<0.05). The BMI/HDL-C, TG/HDL-C, WHTR, and VAI levels in the MS group are higher than those in the non-MS group (P<0.001).

## Correlation between MS and quartiles of BMI/HDL-C, TG/ HDL-C, WHTR, and VAI

The prevalence of MS and its components were compared according to the quartiles of BMI/HDL-C, TG/ HDL-C, WHTR, and VAI, and the Cramer's V index was used to evaluate the correlation (Table 3; Fig. 1).

After grouping the participants according to the quartile of BMI/HDL-C ratio, the results revealed significant differences in the prevalence of MS, abdominal obesity, hyperglycemia, hypertension, hypertriglyceridemia, and HHDL (P<0.001). The prevalence of MS, abdominal obesity, hyperglycemia, hypertension, hypertriglyceridemia, and HHDL increases in proportion to the BMI/HDL-C ratio. The quartile of BMI/HDL-C is weakly correlated with the prevalence of abdominal obesity, hyperglycemia, and hypertension, and moderately correlated with the prevalence of MS and hypertriglyceridemia, and strongly correlated with the prevalence of HHDL (P<0.05, Cramer's V <sub>MS</sub> = 0.414, Cramer's V <sub>abdominal obesity</sub> = 0.270, Cramer's V <sub>hyperglycemia</sub> = 0.134, Cramer's V <sub>hypertension</sub> = 0.160, Cramer's V <sub>hypertriglyceridemia</sub> = 0.301, Cramer's V <sub>HHDL</sub> = 0.630) (Table 3; Fig. 1).

The prevalence of MS, hypertriglyceridemia, and HHDL varies based on TG/HDL-C ratio quartiles (P<0.001). The quartile of TG/HDL-C is moderately correlated with the prevalence of MS and HHDL, but strongly linked with the prevalence of hypertriglyceridemia, and the relationship is statistically significant (P<0.05, Cramer's V <sub>MS</sub> = 0.358, Cramer's V <sub>hypertriglyceridemia</sub> = 0.789, Cramer's V <sub>HHDL</sub> = 0.452) (Table 3; Fig. 1).

The prevalence of MS, abdominal obesity, and hyperglycemia varies according to the quartiles of WHTR, and a positive correlation between WHTR and the prevalence of MS, abdominal obesity, hyperglycemia, hypertension, hypertriglyceridemia, and HHDL was observed (P<0.05). The WHTR quartile is weakly correlated with the prevalence of hyperglycemia, moderately correlated with the prevalence of MS, and strongly correlated with the prevalence of abdominal obesity (P<0.05, Cramer's V <sub>MS</sub> = 0.328, Cramer's V <sub>abdominal obesity</sub> = 0.811, Cramer's V <sub>hyperelycemia</sub> = 0.108) (Table 3; Fig. 1).

Based on the quartile of VAI, there are variations in the prevalence of MS, hypertriglyceridemia, and HHDL (P<0.001). The prevalence of MS and its components increases correspondingly with the increase of VAI. The quartile of VAI is moderately connected with the prevalence of MS and HHDL but strongly correlated with the prevalence of hypertriglyceridemia, and the relationship between parameters was statistically significant (P<0.05,

	Q1	Q2	Q3	Q4	X2	P-value	Cramer's V	٥1	Q2	G3	Q4	X2	P-value	Cramer's V
	BMI/HC	DL-C						TG/HDL	ų					
MS	36 (20.7)	62 (35.6) <sup>a</sup>	101 (58.0) <sup>a, b</sup>	130 (74.7) <sup>a, b,c</sup>	119.5	< 0.001	0.414	41 (23.6)	67 (38.7) <sup>a</sup>	97 (55.4) <sup>a, b</sup>	124 (71.3) <sup>a, b,c</sup>	89.2	< 0.001	0.358
Abdominal obesity	58 (33.3)	66 (37.9)	95 (54.6) <sup>a, b</sup>	117 (67.2) <sup>a, b</sup>	50.9	< 0.001	0.270	85 (48.9)	90 (52.0)	85 (49.1)	75 (43.1)	2.9	0.406	0.065
Hyperglycemia	107 (61.5)	119 (68.4)	133 (76.4) <sup>a</sup>	132 (75.9) <sup>a</sup>	12.5	< 0.001	0.134	121 (69.5)	125 (72.3)	125 (71.4)	120 (69.0)	0.6	0.896	0.029
Hypertension	79 (45.4)	89 (51.1)	104 (59.8) <sup>a</sup>	115 (66.1) <sup>a, b</sup>	17.7	< 0.001	0.160	82 (47.1)	103 (59.5)	99 (56.6)	103 (59.2)	7.1	0.068	0.101
Hypertriglyceridemia	39 (22.4)	71 (40.8) <sup>a</sup>	85 (48.9) <sup>a</sup>	111 (63.8) <sup>a, b,c</sup>	63.0	< 0.001	0.301	0 (0:0)	26 (15.0) <sup>a</sup>	107 (61.1) <sup>a, b</sup>	173 (99.4) <sup>a, b,c</sup>	433.5	< 0.001	0.789
ННDГ	0 (0.0) WHTR	2 (1.1)	31 (17.8) <sup>a, b</sup>	109 (62.6) <sup>a, b,c</sup>	276.2	< 0.001	0.630	2 (1.1) <b>VAI</b>	17 (9.8) <sup>a</sup>	37 (21.1) <sup>a, b</sup>	86 (49.4) <sup>a, b,c</sup>	141.9	< 0.001	0.452
MS	53 (30.3)	55 (31.6)	103 (58.9) <sup>a, b</sup>	118 (67.4) <sup>a, b</sup>	75.4	< 0.001	0.328	41 (23.6)	56 (32.2)	111 (63.8) <sup>a, b</sup>	121 (69.5) <sup>a, b</sup>	108.8	< 0.001	0.395
Abdominal obesity	1 (0.6)	30 (17.2) <sup>a</sup>	131 (74.9) <sup>a, b</sup>	174 (99.4) <sup>a, b,c</sup>	459.6	< 0.001	0.811	76 (43.7)	80 (46.0)	98 (56.3)	82 (47.1)	6.4	0.091	0.096
Hyperglycemia	129 (73.7)	109 (62.6)	121 (69.1)	132 (75.4)	8.169	0.043	0.108	123 (70.7)	119 (68.4)	132 (75.9)	117 (67.2)	3.7	0.307	0.073
Hypertension	95 (54.3)	85 (48.9)	102 (58.3)	105 (60.0)	5.2	0.159	0.086	87 (50.0)	94 (54.0)	108 (62.1)	98 (56.3)	5.4	0.146	0.088
Hypertriglyceridemia	75 (42.9)	76 (43.7)	81 (46.3)	74 (42.3)	0.7	0.883	0.031	2 (1.1)	38 (21.8) <sup>a</sup>	95 (54.6) <sup>a, b</sup>	1 <i>7</i> 1 (98.3) <sup>a, b,c</sup>	380.4	< 0.001	0.739
HHDL	36 (20.6)	35 (20.1)	31 (17.7)	40 (22.9)	1. 4.	0.703	0.045	3 (1.7)	14 (8.0) <sup>a</sup>	44 (25.3) <sup>a, b</sup>	81 (46.6) <sup>a, b,c</sup>	129.6	< 0.001	0.431
Data were expressed as coefficient was used to ex- correlation. (a: $P < 0.05$ vs. waist-to-height ratio. VAI	(%). The c aluate the c Q1 of the s visceral ad	correlation stu ame indicatou liposity index	t was used to c rength. $0.1 < Crar, b: P < 0.05 vs. (. Quartile of BM$	:ompare the diff imer's V < 0.3 ind 22 of the same ii 1/HDL-C: Q1: < 1:	ferences be licates a wei ndicator, c: / 7.168; Q2: 17	tween the qui ak intensity co 2< 0.05 vs. Q3 (168 ~ 20.560;4	artiles of various i rrelation; 0.3 ≤Cra of the same indica Q3: 20.560 ~ 24.22	ndicators (l mer's V <0! itor.). BMI, B 9; Q4: 224	3MI/HDL-C, 1 5 indicates a ody Mass Inc 229; Quartile	G/HDL-C, WH noderate inter lex; HDL-C, hig of TG/HDL-C:	TR, and VAI) and nsity correlatior jh-density lipop Q1: < 0.781; Q2: (	d MS and its t; Cramer's V protein cholo 0.781 ~ 1.265	> components	. The Cramer's V s a high intensity jlyceride; WHTR: .934; Q4: ≥ 1.934;

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**Fig. 1** Prevalence of MS and its components according to the quartiles of BMI/HDL-C, TG/HDL-C, WHTR, and VAI. (**A**) Comparison of the prevalence of MS in the quartile of four key diagnostic indicators. (**B**) Comparison of the prevalence of abdominal obesity in the quartile of four key diagnostic indicators. (**C**) Comparison of the prevalence of hyperglycemia in the quartile of four key diagnostic indicators. (**D**) Comparison of the prevalence of hyperglycemia in the quartile of four key diagnostic indicators. (**D**) Comparison of the prevalence of hypertension in the quartile of four key diagnostic indicators. (**E**) Comparison of the prevalence of hypertriglyceridemia in the quartile of four key diagnostic indicators. (**F**) Comparison of the prevalence of HHDL in the quartile of four key diagnostic indicators. BMI, Body Mass Index; HDL-C, high-density lipoprotein cholesterol; TG, triglyceride; WHTR: waist-to-height ratio. VAI: visceral adiposity index. (\*P < 0.05, \*\*P < 0.01, \*\*\*P < 0.001 a: P < 0.05 vs. Q1 of the same indicator, b: P < 0.05 vs. Q2 of the same indicator, c: P < 0.05 vs. Q3 of the same indicator. #: 0.1 < Cramer's V < 0.3, indicates a weak intensity correlation; ##: 0.3 < Cramer's V < 0.5, indicates a moderate intensity correlation; ###: Cramer's V > 0.5, indicates a high intensity correlation)

Cramer's V  $_{MS}$  = 0.395, Cramer's V  $_{hypertriglyceridemia}$  = 0.739, Cramer's V  $_{HHDL}$  = 0.431) (Table 3; Fig. 1).

#### **Comparison of diagnostic models**

Logistic regression models were established based on the quartiles of BMI/HDL-C, TG/HDL-C, VAI, and WHTR. Then, the odds ratio (OR) values under different adjustment conditions were compared. After adjusting for confounding factors, the subjects with Q3  $(P_{BMI/HDL-C} = 0.001, \text{ OR}_{BMI/HDL-C} = 3.077, 95\%$  $CI_{BMI/HDL-C}$ : 1.575–6.010;  $P_{WHTR} = 0.006$ ,  $OR_{WHTR} =$ 4.171, 95%CI<sub>WHTR</sub>: 1.505–11.556) level of BMI/HDL-C and WHTR showed a higher risk of MS than the subjects in the Q1 group, and Q4 ( $P_{BMI/HDL-C}$  < 0.001,  $OR_{BMI/HDL-C} = 4.629, 95\% CI_{BMI/HDL-C} : 1.982-10.811)$ level of BMI/HDL-C continuously showed a higher risk of MS than the subjects in the Q1 group, while the subjects with Q2 to Q4 (Q2,  $P_{TG/HDL-C} < 0.001$ ,  $OR_{TG/HDL-C}$ = 3.063, 95%  $CI_{TG/HDL-C}$ : 1.662–5.643,  $P_{VAI}$  = 0.010,  $OR_{VAI}$ = 2.189, 95%  $CI_{VAI}$ : 1.202–3.987; Q3,  $P_{TG/HDL-C} < 0.001$ ,  $OR_{TG/HDL-C} = 11.884, 95\% CI_{TG/HDL-C}$ : 5.968–23.665,

 $\begin{array}{l} P_{VAI} < 0.001, \ \mathrm{OR}_{VAI} = 15.914, \ 95\% \ \mathrm{CI}_{VAI} \div 8.097 - 31.280; \\ \mathrm{Q4}, \ P_{TG/HDL-C} < 0.001, \ \mathrm{OR}_{TG/HDL-C} = 75.468, \ 95\% \\ \mathrm{CI}_{TG/HDL-C} \div 28.850 - 197.413, \\ P_{VAI} < 0.001, \ \mathrm{OR}_{VAI} = 80.318, \\ 95\% \ \mathrm{CI}_{VAI} \div 29.840 - 216.182) \ \text{level of TG/HDL-C} \ \text{and VAI} \\ \text{consistently have a higher risk of MS than the subjects in the Q1 group (Fig. 2. A-D, Table S1).} \end{array}$ 

The ROC curves of models under different adjustment conditions were depicted (Fig. 2. E - G) and the diagnostic value of the models was compared. In model 1, no covariates were corrected (Fig. 2. E). In model 2, we further adjusted for gender, education level, and smoking (Fig. 2. F). In model 3, BMI, BFM, PBF, VFA, AMC, WC, SCR, LDH, TG, HDL-C, and insulin were adjusted (Fig. 2. G). We also found that the AUC of the ROC curves of model 3 (AUC=0.780, 95% CI: 0.746–0.814) constructed based on BMI/HDL-C was higher than the AUC of the ROC curves of model 1 and 2 (AUC Model 1 = 0.731, 95% CI: 0.693–0.768; AUC Model 2 = 0.752, 95% CI: 0.716–0.788) (Table 4).

The sensitivity, specificity, AUC, and the cutoff values of the prediction models were shown in Table 4. The



Fig. 2 Forest plot and ROC curves of the models constructed based on BMI/HDL-C, TG/HDL-C, VAI, and WHTR. Correlation between quartile of BMI/HDL-C (A), TG/HDL-C (B), VAI (C), and WHTR (D) and MS with adjustment of different confounding factors. Different background of forest plot represents different confounding factor correction: Blue - without adjustment of confounding factors; red - adjustment of confounding factors including gender, education level, and smoking; yellow - adjustment of confounding factors including BMI, BFM, PBF, VFA, AMC, WC, SCR, LDH, TG, HDL-C, and insulin. Comparison of ROC curves (E) without adjustment of confounding factors; (F) with adjustment of confounding factors including gender, education level, and smoking; (G) with adjustment of confounding BMI, BFM, PBF, VFA, AMC, WC, SCR, LDH, TG, HDL-C, and insulin. BMI, Body Mass Index; HDL-C, high-density lipoprotein cholesterol; TG, triglyceride; WHTR: waist-to-height ratio. VAI: visceral adiposity index

sensitivity and specificity of the model are both higher than 60%. The model 3 based on BMI/HDL-C and Model 3 based on VAI have the highest AUC values compared to the AUC values of other models, which are 0.780 (95% CI: 0.746, 0.814) and 0.780 (95% CI: 0.745, 0.814).

## The optimal model

Considering both simplicity (easy to calculate) and accuracy (the AUC value of the ROC curve), and its correlation with MS, model 3 based on BMI/HDL-C

Models	Sensitivity	Specificity	AUC	(95%CI)	Cut off value
BMI/HDL-C <sup>a</sup>					
Model 1	0.700	0.681	0.731	(0.693,0.768)	0.468
Model 2	0.688	0.714	0.752	(0.716,0.788)	0.462
Model 3	0.810	0.640	0.780	(0.746,0.814)	0.623
TG/HDL-C <sup>b</sup>					
Model 1	0.670	0.651	0.699	(0.661,0.738)	0.471
Model 2	0.783	0.575	0.718	(0.680,0.756)	0.425
Model 3	0.725	0.728	0.779	(0.745,0.813)	0.717
VAI <sup>c</sup>					
Model 1	0.703	0.684	0.712	(0.673,0.750)	0.480
Model 2	0.703	0.684	0.716	(0.678,0.755)	0.458
Model 3	0.878	0.580	0.780	(0.745,0.813)	0.500
WHTR <sup>d</sup>					
Model 1	0.670	0.649	0.672	(0.632,0.712)	0.452
Model 2	0.606	0.714	0.684	(0.644,0.724)	0.549
Model 3	0.875	0.550	0.772	(0.737,0.807)	0.516

**Table 4** The sensitivity, specificity, AUC and cutoff value of different models

a: prediction model based on quartile of BMI/HDL-C. b: prediction model based on the quartile of TG/HDL-C. c: prediction model based on the quartile of VAI. d: prediction model based on the quartile of WHTR. Model 1: no confounding factors were adjusted. Model 2: adjusting for confounding factors including gender, education level, and smoking status. Model 3: further adjustment of confounding factors including BMI, BFM, PBF, VFA, AMC, WC, SCR, LDH, TG, HDL-C, and insulin. BMI, Body Mass Index; HDL-C, high-density lipoprotein cholesterol; TG, triglyceride; WHTR: waist-to-height ratio. VAI: visceral adiposity index



Fig. 3 Nomogram and calibration curve of the optimal model. (A) Nomogram of the optimal prediction model of metabolic syndrome. (B) Calibration curve of the optimal prediction model of metabolic syndrome. BFM, Body Fat Mass; WC, Waist Circumference; TG, triglyceride; Q1, Q3, Q4, quartile of BMI/ HDL-C

was selected as the optimal model to predict metabolic syndrome.

Beginning with the risk factors of MS (Table 1), results of logistic regression analysis showed that gender, BFM, WC, TG, and BMI/HDL-C remained important predictors after adjusting for confounding factors. Then a nomogram (Fig. 3A) was built to predict the risk of metabolic syndrome. To use the nomogram, clinicians could position on the variable axis based on the patients' situation, and extend a line upwards to determine the points of each variable based on the topmost axis. Then add the points obtained from each variable, extend a line downwards from the total point axis to the risk axis to determine the risk of metabolic syndrome at the lower line of the nomogram. The cut-off value of BMI/HDL-C for the nomogram was 0.623 for diagnosing metabolic syndrome.

We plot the calibration curve (Fig. 3B) and ROC curve (Fig. 2G) to evaluate the diagnostic efficiency of the models. Sensitivity, specificity, the AUC, and cut-off value of

different models were used to analyze the ROC curve, and we found that the AUC value of the model is 0.780 (0.746, 0.814), indicating a powerful prediction ability.

#### Model equation

The equation for the developed model is provided below:

$$\begin{split} Logit &= -16.994 - 1.780 \times Gender \, (male) \\ &- 0.103 \times BFM + 0.195 \times WC \, (cm) + 1.763 \\ &\times TG(mmol/L) + 1.124 \times Q3 + 1.532 \times Q4 \end{split}$$

## Discussion

In this cross-sectional study, we investigated the correlation between TG/HDL-C, WHTR, VAI, and BMI/ HDL-C ratios and the prevalence of MS. Our data indicated that higher level of TG/HDL-C, WHTR, VAI, and BMI/HDL-C ratios were positively associated with MS, abdominal obesity, hyperglycemia, hypertension, hypertriglyceridemia, and HHDL in adults aged 40 to 85. BMI, BFM, PBF, VFA, AMC, WC, SCR, TG, insulin, LDH, and HDL-C varied between subjects with MS and subjects without MS. The prevalence of MS, abdominal obesity, hyperglycemia, hypertension, hypertriglyceridemia, and HHDL was compared according to the quartiles of BMI/ HDL-C, TG/HDL-C, VAI, and WHTR. We found that, in comparison with classical indicators including TG/ HDL-C, VAI, and WHTR, BMI/HDL-C showed a significantly stronger correlation with MS. Based on the BMI/ HDL-C quartile, we proposed a new model to predict MS, which includes five factors: gender, BFM, WC, TG, and BMI/HDL-C ratio. The nomogram visually displayed the model.

Previous prospective and cross-sectional studies have reported that patients with metabolic syndrome (MS) exhibited an increased level of BMI, BFM, PBF, VFA, AMC, WC, SCR, TG, and insulin as comparing with non-MS subjects (P < 0.05) [28, 29]. These findings align with our results. Body fat mass commonly reflects the content of visceral fat and is a widely accepted parameter for evaluating visceral obesity. A study found that the increase in BFM leads to an increased risk of MS in adults [30]. Subjects who had accumulated visceral fat were at high risk for MS, which had a cumulative effect on the development of obesity [31]. Serum creatinine level in Chinese adults showed a positive correlation with the risk of MS. Even for individuals with SCR levels within the normal range, a high SCR level implies a high risk of MS [32]. A previous study found that the levels of total LDH, LDH1,2,4, and 5 in obese and diabetes patients were lower than in normal control subjects [33]. In our study, we also found that the level of total LDH in the MS patients was lower than in the non-MS subjects (mean LDH 186.3 [SD 36.1] vs. 194.3 [50.2]; P=0.043).

However, other studies reported inconsistent results. Vizir OO et al. found that the subjects with severe diabetes were characterized by a significant change in LDH activity [34]. This was demonstrated by an increase in the activity of total LDH, LDH4, and LDH5, but a decrease in LDH1 and LDH2 activity when compared to the healthy subjects [34]. These data indicated the LDH subtypedependent relation between LDH activity and metabolic diseases. Gender differences in the prevalence of MS were demonstrated by a previous study, which reported that females showed a lower risk for MS than the males [22]. Consistent with previous results, our research also found that the proportion of MS in males (40.0%) was lower than in females (50.2%). The gender difference in the prevalence of MS may be attributed to the discrepant distribution of fat in females and males, and the study has reported that females have more body fat than males. In addition, unhealthy lifestyles such as smoking and drinking in the male population are more common than in females, which are also risk factors for the development of MS [35].

For elderly people in China, there is a stable association between obesity and lipid-related indicators and MS, which were considered effective indicators for diagnosing chronic diseases [36]. As one of the most extensively used indices for evaluating body shape, abnormal BMI was strongly associated with the risk of obesity and metabolic syndrome [37]. Additionally, numerous studies have confirmed the correlation between BMI and MS. A cohort study analyzed the correlation between BMI and the risk of MS, and recommended a BMI level of 27 as the ideal cut-off value for identifying metabolic syndrome [38]. Another longitudinal study divided the participants into fast trajectory (slope=1.447) and slow trajectory (slope=1.433) according to the growth rate of BMI with age, which confirmed that the population with a fast growth rate of BMI had a higher incidence of MS than the population with a slow growth rate (OR=3.40)[39]. The prevalence of MS increased with the rise of BMI and age [40]. As blood pressure, blood glucose, and abdominal obesity increased with age, people at the age of 60 years old and above have more metabolic complications [35]. As a reverse marker of MS and cardiovascular disease, the subclasses of HDL-C, especially the level of HDL3, were reported to have a negative relationship with MS, which could provide more information on diagnosing MS than HDL-C [41].

Studies consistently report that MS patients have a higher TG/HDL-C ratio, WHTR, and VAI than non-MS subjects [42, 43]. In our study, we found a significant association between BMI/HDL-C and MS, abdominal obesity, hyperglycemia, hypertension, hypertriglyceridemia, and HHDL (P<0.05, Cramer's V>0.1). The correlations of TG/HDL-C, WHTR, and VAI with MS were also

significant (P < 0.05, Cramer's V>0.1), but the correlations of these parameters with hyperglycemia and hypertension were undetected (P > 0.05), implying a better diagnostic efficacy of BMI/HDL-C than the other three indicators. It is reported that the TG/HDL-C ratio is a reliable predictor of MS, particularly because it showed a significant correlation with MS in the elderly Chinese population [44]. Furthermore, studies have indicated that the TG/HDL-C ratio is an efficient biomarker for abnormal lipid metabolism and cardiovascular disease [45]. In our study, we found that the diagnostic potency of TG/HDL-C in diagnosing central obesity, hyperglycemia, and hypertension was poor. Consistently, studies have also reported that WC was not related to the TG/ HDL-C ratio. Therefore, we speculate that WC was not an effective predictor of central obesity in adults [46]. As our results showed, there was no significant correlation between TG/HDL-C and abdominal obesity (P=0.406). Due to comprehensively consideration of waist circumference and height, the WHTR value was suggested as the best predictor of central obesity. However, in our study, the diagnostic effect of WHTR on blood lipids and blood pressure abnormalities was not significant (P>0.05). The same drawback appeared in the VAI value, which failed to reflect the blood pressure status, and the calculation of VAI was complex [47]. Comparing the models constructed with BMI/HDL-C, TG/HDL-C, VAI, and WHTR, our data demonstrated that model based on BMI/HDL-C was the most comprehensive and reliable model with the greatest potential for diagnosing MS in Chinese adults aged 40-85.

The advantage of this study is that we identified a new key indicator and developed a new model to predict MS in patients clinically. BMI/HDL-C was easy to obtain and had better accuracy and comprehensiveness than other indicators (TG/HDL-C, VAI, WHTR), which provided a new tool for early prediction of MS. Figure 3 showed a nomogram of the developed MS diagnostic model, which provided help for the clinician to assess the risk of MS according to clinical indicators.

There are some limitations in this study. Firstly, the cross-sectional study design makes us fail to draw a causal conclusion. Secondly, the sample size of this study is small and age-related analysis of metabolic syndrome is lacking, thus the conclusions derived from this study need to be further verified in large-scale population studies. Thirdly, the study was conducted in Chinese; therefore, extrapolation of our results to other populations should be done with caution.

## Conclusions

This study established a new model based on BMI/ HDL-C to predict MS in Chinese adults aged 40–85, which compensated for the lack of existing research on the prediction of MS based on BMI/HDL-C quartile. The model established in our study might provide a sensitive and effective tool for diagnosing MS.

#### **Supplementary Information**

The online version contains supplementary material available at https://doi.org/10.1186/s12902-024-01752-9.

Supplementary Material 1 Supplementary Material 2

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Not applicable.

#### Author contributions

Yu Liu: Conceptualization, Methodology, Software, Writing - Original Draft. Xixiang Wang: Methodology, Formal analysis, Visualization. Jie Mu: Investigation. Yiyao Gu: Investigation. Shaobo Zhou: Writing – Review & Editing. Xiaojun Ma: Investigation. Jingjing Xu: Investigation. Lu Liu: Investigation. Xiuwen Ren: Investigation. Zhi Duan: Investigation. Lin Liu: Investigation. Xing Vang: Conceptualization, Supervision, Writing - Review & Editing, Funding acquisition.

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#### Data availability

Availability of data and materialsThe datasets generated during and analyzed during the current study are available from the corresponding author upon reasonable request.

#### Declarations

#### **Ethics** approval

The study was approved by the Committee on Medical Ethics of Capital Medical University. The study protocol was approved by the Committee on Medical Ethics of Capital Medical University (No. 2012SY23), and the study procedures followed the ethical standards of the Helsinki Declaration of 1975.

#### **Consent to participate**

Written informed consent was obtained from individual or guardian participants.

## Consent for publication

Not applicable.

#### **Competing interests**

The authors have no relevant financial or non-financial interests to disclose.

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#### References

- Lemieux I, Despres JP. Metabolic syndrome: past, Present and Future. Nutrients. 2020;12(11).
- Saklayen MG. The global epidemic of the metabolic syndrome. Curr Hypertens Rep. 2018;20(2):12.
- Pan WH, Yeh WT, Weng LC. Epidemiology of metabolic syndrome in Asia. Asia Pac J Clin Nutr. 2008;17(Suppl 1):37–42.
- Bener A, Zirie M, Musallam M, et al. Prevalence of metabolic syndrome according to adult treatment panel III and international diabetes federation criteria: a population-based study. Metab Syndr Relat Disord. 2009;7(3):221–9.
   Yao F, Bo Y, Zhao L et al. Prevalence and influencing factors of metabolic
- Yao F, Bo Y, Zhao L et al. Prevalence and initiaencing factors of metabolic syndrome among adults in China from 2015 to 2017. Nutrients. 2021;13(12).
- Lu J, Wang L, Li M, et al. Metabolic syndrome among adults in China: the 2010 China noncommunicable disease surveillance. J Clin Endocrinol Metab. 2017;102(2):507–15.
- Li R, Li W, Lun Z, et al. Prevalence of metabolic syndrome in Mainland China: a meta-analysis of published studies. BMC Public Health. 2016;16:296.
- Chen Y, Xu W, Zhang W, et al. Plasma metabolic fingerprints for large-scale screening and personalized risk stratification of metabolic syndrome. Cell Rep Med. 2023;4(7):101109.
- Cho Y, Lee SY. Useful biomarkers of metabolic syndrome. Int J Environ Res Public Health. 2022;19(22).
- 10. Pujos-Guillot E, Brandolini M, Petera M, et al. Systems metabolomics for prediction of metabolic syndrome. J Proteome Res. 2017;16(6):2262–72.
- Christakoudi S, Tsilidis KK, Muller DC, et al. A body shape index (ABSI) achieves better mortality risk stratification than alternative indices of abdominal obesity: results from a large European cohort. Sci Rep. 2020;10(1):14541.
- 12. Silva VM, Vinagre CG, Dallan LA, et al. Plasma lipids, lipoprotein metabolism and HDL lipid transfers are equally altered in metabolic syndrome and in type 2 diabetes. Lipids. 2014;49(7):677–84.
- Son DH, Lee HS, Lee YJ, et al. Comparison of triglyceride-glucose index and HOMA-IR for predicting prevalence and incidence of metabolic syndrome. Nutr Metab Cardiovasc Dis. 2022;32(3):596–604.
- 14. Raimi TH, Dele-Ojo BF, Dada SA, et al. Triglyceride-glucose index and related parameters predicted metabolic syndrome in nigerians. Metab Syndr Relat Disord. 2021;19(2):76–82.
- Kang SW, Kim SK, Kim YS, et al. Risk prediction of the metabolic syndrome using TyG index and SNPs: a 10-year longitudinal prospective cohort study. Mol Cell Biochem. 2023;478(1):39–45.
- Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. Diabet Med. 1998;15(7):539–53.
- Ho Cl, Chen JY, Chen SY, et al. Relationship between TG/HDL-C ratio and metabolic syndrome risk factors with chronic kidney disease in healthy adult population. Clin Nutr. 2015;34(5):874–80.
- Zanuncio VV, Sediyama C, Dias MM, et al. Neck circumference and the burden of metabolic syndrome disease: a population-based sample. J Public Health (Oxf). 2022;44(4):753–60.
- Laohabut I, Udol K, Phisalprapa P, et al. Neck circumference as a predictor of metabolic syndrome: a cross-sectional study. Prim Care Diabetes. 2020;14(3):265–73.
- Al-Shami I, Alkhalidy H, Alnaser K, et al. Assessing metabolic syndrome prediction quality using seven anthropometric indices among Jordanian adults: a cross-sectional study. Sci Rep. 2022;12(1):21043.
- Ashwell M, Gunn P, Gibson S. Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: systematic review and meta-analysis. Obes Rev. 2012;13(3):275–86.
- 22. Amato MC, Giordano C, Galia M, et al. Visceral adiposity index: a reliable indicator of visceral fat function associated with cardiometabolic risk. Diabetes Care. 2010;33(4):920–2.
- Radetti G, Fanolla A, Grugni G, et al. Indexes of adiposity and body composition in the prediction of metabolic syndrome in obese children and adolescents: which is the best? Nutr Metab Cardiovasc Dis. 2019;29(11):1189–96.
- 24. [National guidelines for the prevention and control of diabetes in primary care. (2022)]. Zhonghua Nei Ke Za Zhi, 2022,61(3):249–262.
- World Health Organization. The WHO STEPwise approach to noncommunicable disease risk factor surveillance. Geneva 2017.[Z].

- Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem. 1972;18(6):499–502.
- 27. Chen C, Lu FC. The guidelines for prevention and control of overweight and obesity in Chinese adults. Biomed Environ Sci. 2004;17(Suppl):1–36.
- Cho SA, Joo HJ, Cho JY, et al. Visceral fat area and serum adiponectin level predict the development of metabolic syndrome in a community-based asymptomatic population. PLoS ONE. 2017;12(1):e169289.
- 29. Ying X, Jiang Y, Qin G, et al. Association of body mass index, waist circumference, and metabolic syndrome with serum cystatin C in a Chinese population. Med (Baltim). 2017;96(10):e6289.
- Oh YH, Choi S, Lee G et al. Changes in body composition are associated with metabolic changes and the risk of metabolic syndrome. J Clin Med. 2021;10(4).
- Kim SH, Kang HW, Jeong JB, et al. Association of obesity, visceral adiposity, and Sarcopenia with an increased risk of metabolic syndrome: a retrospective study. PLoS ONE. 2021;16(8):e256083.
- Wang J, Li X, Han X, et al. Serum creatinine levels and risk of metabolic syndrome in a middle-aged and older Chinese population. Clin Chim Acta. 2015;440:177–82.
- Johari TY, Ghoneim MA, Moselhy SS. Thyroid profile and LDH isoenzymes as prognostic biomarkers for diabetic and/or obese subjects. Afr Health Sci. 2018;18(3):697–706.
- Vizir OO. [Activity of blood serum lactate dehydrogenase in diabetes mellitus]. Probl Endokrinol (Mosk). 1977;23(3):15–7.
- Slagter SN, van Waateringe RP, van Beek AP, et al. Sex, BMI and age differences in metabolic syndrome: the dutch lifelines cohort study. Endocr Connect. 2017;6(4):278–88.
- Gui J, Li Y, Liu H, et al. Obesity- and lipid-related indices as a predictor of obesity metabolic syndrome in a national cohort study. Front Public Health. 2023;11:1073824.
- Bramante CT, Palzer EF, Rudser KD, et al. BMI metrics and their association with adiposity, cardiometabolic risk factors, and biomarkers in children and adolescents. Int J Obes (Lond). 2022;46(2):359–65.
- Kobo O, Leiba R, Avizohar O, et al. Normal body mass index (BMI) can rule out metabolic syndrome: an Israeli cohort study. Med (Baltim). 2019;98(9):e14712.
- Ying M, Hu X, Li Q, et al. Long-term trajectories of BMI and cumulative incident metabolic syndrome: a cohort study. Front Endocrinol (Lausanne). 2022;13:915394.
- 40. Razzouk L, Muntner P. Ethnic, gender, and age-related differences in patients with the metabolic syndrome. Curr Hypertens Rep. 2009;11(2):127–32.
- Yang HS, Hur M, Kim H, et al. HDL subclass analysis in predicting metabolic syndrome in koreans with high HDL cholesterol levels. Ann Lab Med. 2020;40(4):297–305.
- 42. Jialal I, Adams-Huet B, Remaley AT. A comparison of the ratios of C-reactive protein and triglycerides to high-density lipoprotein-cholesterol as biomarkers of metabolic syndrome in African americans and non-hispanic whites. J Diabetes Complications. 2022;36(7):108231.
- Ma A, Fang K, Dong J, et al. Prevalence and related factors of metabolic syndrome in Beijing, China (Year 2017). Obes Facts. 2020;13(6):538–47.
- Nie G, Hou S, Zhang M, et al. High TG/HDL ratio suggests a higher risk of metabolic syndrome among an elderly Chinese population: a cross-sectional study. BMJ Open. 2021;11(3):e41519.
- Kosmas CE, Rodriguez PS, Bousvarou MD et al. The triglyceride/high-density lipoprotein cholesterol (TG/HDL-C) ratio as a risk marker for metabolic syndrome and cardiovascular disease. Diagnostics (Basel). 2023;13(5).
- Ren X, Chen ZA, Zheng S, et al. Association between triglyceride to HDL-C ratio (TG/HDL-C) and insulin resistance in Chinese patients with newly diagnosed type 2 diabetes mellitus. PLoS ONE. 2016;11(4):e154345.
- Lazzer S, D'Alleva M, Isola M et al. Cardiometabolic index (CMI) and visceral adiposity index (VAI) highlight a higher risk of metabolic syndrome in women with severe obesity. J Clin Med. 2023;12(9).

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