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Lihua Wang, Pingping Yu, Yao Chen, Mengxue Chen, Jing Deng & Lehua Yu

Association of Central Adiposity and Metabolic Markers With Osteopenia and Osteoporosis in Chinese Adults: A QCT-Based Cross-Sectional Study

Authors:
Lihua Wang[1], Pingping Yu[1] , Yao Chen[1] ,Mengxue Chen[1] , Jing Deng1[*+] , Lehua Yu1[*+]
Affiliations:
Health Medical Center, Second Affiliated Hospital of Chongqing Medical University, Chongqing, China
*Corresponding Authors:
Jing Deng−305930@hospital.cqmu.edu.cn
Lehua Yu−300895@cqmu.edu.cn
[+] These authors have contributed equally to this work.

Abstract

Aim: To evaluate the associations and comparative performance of novel anthropometric and metabolic indices with osteopenia and osteoporosis among middle-aged and older Chinese adults.

Methods: A cross-sectional study was conducted among 10,142 Chinese adults aged ≥45 years who underwent quantitative computed tomography (QCT) for lumbar spine BMD assessment. Participants were categorized as normal, osteopenia, and osteoporosis. Associations and predictive capabilities of anthropometric indices were analyzed using multivariable logistic regression and receiver operating characteristic (ROC) curve analyses.

Results: The prevalences of osteopenia and osteoporosis were 35.14% and 14.05%, respectively. After adjusting for confounders, weight-adjusted waist index (WWI), relative fat mass (RFM), a body shape index (ABSI), triglyceride – glucose (TyG) index, and glycated hemoglobin (HbA1c) were found to be independently associated with osteopenia and osteoporosis. Among all indices, WWI demonstrated the strongest predictive value for osteoporosis (area under the curve = 0.726), followed by RFM and ABSI. In contrast, BMI and the visceral adiposity index (VAI) showed no significant associations with low BMD.

Conclusion: Indices associated with central adiposity and metabolic dysfunction, especially WWI, may provide more precise prediction of osteoporosis risk. Incorporating such indices into early risk stratification for osteoporosis among older Chinese adults may have potential clinical utility.

Keywords Osteopenia; Osteoporosis; Anthropometric indices; Metabolic indices; Bone mineral density; Quantitative computed tomography

Introduction

Osteopenia and osteoporosis are serious global public health issues especially in middle-aged and elderly populations, characterized by decreased BMD, deterioration of bone tissue structure, and a heightened risk of fractures. Osteopenia is clinically defined as BMD values below normal thresholds but above the diagnostic criteria for osteoporosis, representing an intermediate state that substantially elevates fracture risk if left untreated [1,2]. Osteoporosis, a more severe condition, dramatically increases susceptibility to fractures, predominantly at skeletal sites, such as the spine, hips, and wrists, significantly impacting the quality of life and imposing considerable socioeconomic burdens worldwide [3,4].

The global prevalence of osteopenia and osteoporosis is rising sharply owing to rapidly aging populations. It is predicted that approximately one-third of women aged 50 and above and one-fifth of men will experience osteoporosis in their lifetime [5]. In China, the large aging population has made osteoporosis and its complications a serious public health crisis, emphasizing the need for more effective early screening strategies [6,7].

Although the pathogenesis of osteoporosis remains uncertain, bone loss typically progresses silently, causing many individuals to overlook the condition. This highlights the critical importance of early bone health assessment [8,9]. The most commonly used bone density screening method of bone densityis dual energy X-ray absorptiometry (DXA), which also serves as the basic examination for the World Health Organization's diagnostic criteria for osteoporosis [10]. However, its diagnostic accuracy and ability to assess fracture risk are subject to several limitations. When interpreting T-scores, it is necessary to exclude vertebral segments affected by endplate sclerosis, osteophyte formation, or previous compression fractures, as these conditions may compromise measurement accuracy [11]. Furthermore, DXA does not provide information on bone microarchitecture, which may result in missed diagnoses of early stage osteoporosis [12]. In contrast, quantitative computed tomography (QCT), which can independently evaluate cortical and trabecular bone densities, yields more accurate measurements and demonstrates higher sensitivity in detecting early bone loss. QCT is not influenced by body size or weight, which enhances its applicability to diverse populations [13,14].

Emerging evidence indicates that BMD is closely associated with several metabolic factors. A higher BMI is generally associated with increased bone density, which may be due to the mechanical loading effect of excess body weight that stimulates bone formation and supports bone maintenance. Additionally, a higher BMI commonly reflects a more favorable nutritional status, which is supposed to be beneficial for bone health [15]. In contrast, visceral adipose tissue, although contributing to increased body weight, it is believed to be negatively correlated with bone density, possibly because of its metabolic activity is involved in the development of insulin resistance, hyperglycemia, and dyslipidemia, all of whic have a negative impact on bone metabolism and disrupt bone density [16–19]. However, conventional indicators such as BMI have inherent limitations as they may fail to identify individuals

with normal overall weight but excess abdominal fat, leading to an underestimation of fracture risk in such cases [20].

In recent years, several novel anthropometric indices such as the Weight-Adjusted Waist Index (WWI), A Body Shape Index (ABSI), and Relative Fat Mass (RFM) have emerged as more precise surrogates of central obesity and fat distribution[21–23]. The WWI is calculated as the ratio of waist circumference to body weight, providing a measure of central obesity while accounting for overall body size. The ABSI reflects visceral fat distribution and is considered a better predictor of metabolic risk compared to traditional body mass index (BMI). The RFM is a simple method that estimates body fat percentage based on height and weight. Concurrently, metabolic markers including the Triglyceride-Glucose (TyG) index, a composite measure of insulin resistance derived from fasting triglyceride and glucose levels, have been identified as strong predictors of insulin resistance and vascular aging, both of which may play critical roles in bone metabolism[24–26].

Although previous studies have explored the relationship between certain metabolic indicators and BMD, few have systematically evaluated the comprehensive predictive efficacy of emerging anthropometric and metabolic markers in large, apparently healthy populations undergoing high-resolution volumetric BMD measurements using QCT [27,28].

Therefore, this study was designed to explore the association between novel anthropometric and metabolic indicators and the prevalence of osteopenia and osteoporosis in middle-aged and elderly people in China. The most effective indicators for the early screening of bone mineral loss may be identified by evaluating and comparing the predictive performance of these emerging metabolic markers.

2. Methods

2.1. Study Design and Participants

This cross-sectional study included 12,656 adults who underwent comprehensive health examinations, including QCT for BMD evaluation, at the Health Medical Center of the Second Affiliated Hospital of Chongqing Medical University from July 1, 2020, to January 31, 2024.

Participants who were 45 years old or older and had complete data on BMD, metabolic parameters, anthropometric measures, and lifestyle factors were included in the study. The exclusion criteria were as follows: (a) age < 45 years (n=2232); (b) severe liver or kidney disease, tumors, or diagnosed metabolic disorders (n=65); and (c) missing or incomplete data on BMD (n=217). Ultimately, 10,142 eligible participants (5196 females, 4946 males) were included in the final study analysis (Fig. 1).

Ethical approval was obtained from the Medical Ethics Committee of the Second Affiliated Hospital of Chongqing Medical University, and all procedures conformed to the Declaration of Helsinki.

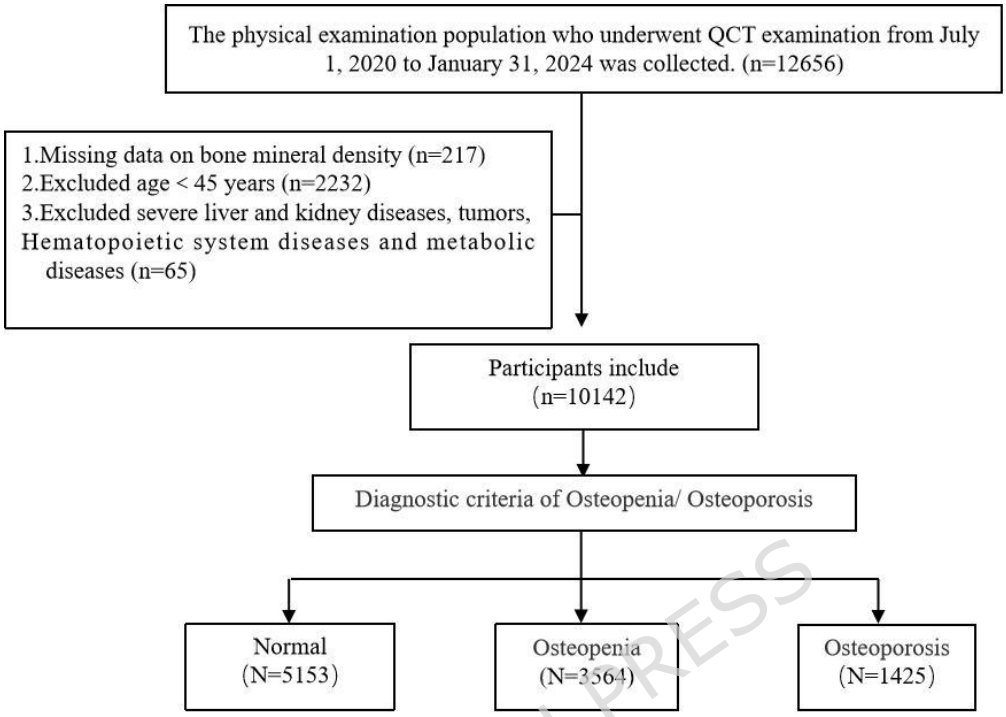

Fig. 1. Study flowchart

2.2 QCT-Based BMD Measurement and Grouping

BMD was assessed using a calibrated 64-slice CT scanner (Somatom go.Top; Siemens Healthineers, Germany) combined with QCT Pro software (version 6.1; Mindways Software, Inc., USA). Prior to scanning, the CT system and QCT software were calibrated using a standardized solid cylindrical calibration phantom (Mindways Model 3). Calibration was performed daily to ensure the accuracy and consistency of measurements. During each scan, a standardized QCT phantom was positioned beneath the lumbar spine region of each participant to guarantee measurement precision.

Using automated software algorithms, regions of interest (ROIs) in the trabecular area of the L1-L5 vertebrae were segmented. The corresponding BMD values (mg/cm ^{3}) were recorded for each vertebral level. Participants were then classified into three diagnostic categories based on average lumbar BMD values: normal (>120 mg/cm^3), osteopenia (80 – 120 mg/cm^3), and osteoporosis ($\leqslant$80 mg/cm^3). These thresholds are consistent with those recommended by the International Society for Clinical Densitometry (ISCD) and have been widely used in previous studies to classify bone density in both Western and Chinese populations. Prior Chinese studies have also used similar BMD thresholds for categorizing bone health status in middle-aged and older adults.[29,30]

2.3 Data Collection and Definitions

Data acquisition encompassed demographic profiles, clinical history, anthropometric measurements, biochemical parameters, and lifestyle habits. Detailed medical history information was collected, including diagnoses of hypertension and diabetes, along with smoking status (smoker or non-smoker), alcohol intake (classified as non-drinker, light drinker <140 g/week for males and <70 g/week for females, and heavy drinker ⩾140 g/week for males and ⩾70 g/week for females). Physical activity levels were assessed using a validated self-reported questionnaire (none, ⩽3 days/week, or ⩾3 days/week with each session lasting 30–60 minutes of moderate intensity).

Anthropometric indices, such as height and weight, were obtained using standardized Omron body scales, while waist and hip circumferences were measured by trained staff following standardized procedures. Blood pressure and heart rate were obtained using an automatic Omron device after a minimum rest period of 5 minutes.

Venous blood samples were collected between 7:00 and 10:00 AM after at least 8 hours of overnight fasting. All biochemical analyses were conducted using an automated analyzer (Hitachi, Tokyo, Japan). The measured parameters included liver function markers (γ-glutamyl transferase [GGT], albumin [ALB], 5′-nucleotidase [5-NT], alanine aminotransferase [ALT], aspartate aminotransferase [AST],direct bilirubin [DBiL], indirect bilirubin [IDBiL], total bilirubin [TBil]), lipid profiles (low-density lipoprotein cholesterol [LDL-C], total cholesterol [TC], triglycerides [TG], high-density lipoprotein cholesterol [HDL-C]), renal function indicators (uric acid [UA], blood urea nitrogen [BUN], serum creatinine [SCr]), glucose metabolism parameters (fasting plasma glucose [FPG], glycated hemoglobin [HbA1c]), and complete blood count indices (platelets [PLT], white blood cells [WBC], hemoglobin [HB], mean corpuscular hemoglobin [MCH], mean corpuscular hemoglobin concentration [MCHC])

Several derived metabolic indicators were calculated : TyG index as ln [TG (mg/dL)×FPG (mg/dL)/2]. Non-high-density lipoprotein cholesterol to HDL-cholesterol ratio (NHHR) as (TC - HDL-C) / HDL-C, and WWI as WC (cm) divided by the square root of body weight (kg). Additional anthropometric and metabolic indices included the following: a body shape index (ABSI) calculated as waist circumference (WC) divided by BMI^(2/3) × height^(1/2); relative fat mass (RFM) calculated as 64 − 20 × (height / waist) for men and 76 − 20 × (height / waist) for women; body adiposity index (BAI) defined as (hip circumference / height^1.5) − 18; waist-to-height ratio (WHTR) defined as WC / height; and waist-to-hip ratio (WHR) calculated as WC / hip circumference. The visceral adiposity index (VAI), a sex-specific indicator of visceral adipose dysfunction, was computed as (WC / (39.68 + 1.88 × BMI)) × (TG / 1.03) × (1.31 / HDL) for men and (WC / (36.58 + 1.89 × BMI)) × (TG / 0.81) × (1.52 / HDL) for women. Liver fat content (LFC) and visceral adipose area (VAA), representing visceral adipose tissue (VAT), were quantified using QCT imaging

following standardized abdominal fat assessment methods described in recent CT-based body composition studies [31,32].

2.4 Statistical Analyses

Statistical procedures were conducted using R (version 4.3.2) and EmpowerStats (X&Y Solutions, Boston, MA, USA). Continuous variables were summarized as mean ± standard deviation (SD) or median with interquartile range (IQR), while categorical data were described by frequencies and percentages.

Group comparisons for continuous data utilized weighted one-way ANOVA, and categorical variables were evaluated via weighted chi-square tests. To address potential multicollinearity, variables with variance inflation factor (VIF) $\geqslant 5$ were excluded from multivariable models. Multiple logistic regression analysis was developed to assess the relationship between the anthropometric/metabolic indices and osteopenia or osteoporosis, adjusting for potential confounders. Receiver operating characteristic (ROC) curve analyses were were used to evaluate the predictive abilities of selected metabolic and anthropometric indicators for identifying osteopenia and osteoporosis, with area under the curve (AUC) values compared to identify the most robust indicators. Statistical significance was defined as a two-sided P-value below 0.05.

3. Results

3.1 Baseline Characteristics of Study Participants

In total, 10,142 eligible participants were included in this study and categorized into three groups according to their BMD status: normal (n = 5,153), osteopenia (n = 3,564), and osteoporosis (n = 1,425). The baseline characteristics and biochemical parameters of the three groups are summarized in Table 1.

Table 1. Baseline characteristics of participants categorized by bone mineral density status

Variables	ALL (N=10142)	Normal (N=5153)	Osteopenia (N=3564)	Osteoporosis (N=1425)	p value
MASLD, n (%)					
NO	3345.00 (44.21)	1892.00 (46.74)	1101.00 (42.25)	352.00 (38.60)	<0.001
YES	4221.00 (55.79)	2156.00 (53.26)	1505.00 (57.75)	560.00 (61.40)	
Sex, n (%)					
Female	5196.00 (51.23)	2446.00 (47.47)	1766.00 (49.55)	984.00 (69.05)	<0.001
Male	4946.00 (48.77)	2707.00 (52.53)	1798.00 (50.45)	441.00 (30.95)	
Age (years)	57.50 (9.35)	53.15 (6.44)	59.07 (8.24)	69.26 (9.45)	<0.001
BMI (kg/m^2)	24.08 (3.03)	24.09 (2.99)	24.10 (3.01)	23.95 (3.19)	0.307
Waistline (cm)	82.03 (9.49)	81.35 (9.57)	82.71 (9.38)	82.81 (9.31)	<0.001
WHR	0.87 (0.07)	0.86 (0.07)	0.87 (0.07)	0.88 (0.07)	<0.001
WHTR	0.51 (0.05)	0.50 (0.05)	0.51 (0.05)	0.53 (0.06)	<0.001
BAI	28.22 (3.76)	27.72 (3.43)	28.22 (3.72)	30.08 (4.40)	<0.001
ABSI	5.91 (1.68)	5.82 (1.63)	5.99 (1.62)	6.04 (1.96)	<0.001
RFM	30.41 (6.84)	29.28 (6.11)	30.55 (6.82)	34.17 (7.94)	<0.001
VAI	2.21 (2.40)	2.18 (2.49)	2.24 (2.28)	2.30 (2.32)	0.279

SBP (mmHg)	127.77 (18.44)	124.34 (17.50)	128.97 (18.09)	137.13 (18.97)	<0.001
DBP (mmHg)	75.29 (11.29)	75.22 (11.44)	75.85 (11.06)	74.18 (11.22)	<0.001
HB (g/L)	143.27 (14.87)	144.40 (15.43)	143.79 (14.33)	137.96 (12.90)	<0.001
PLT	213.33 (57.53)	217.97 (59.27)	210.29 (55.01)	204.17 (55.74)	<0.001
MCH (pg)	30.21 (2.24)	30.12 (2.33)	30.27 (2.20)	30.38 (1.95)	<0.001
MCHC (g/L)	328.49 (10.06)	329.36 (10.60)	328.31 (9.52)	325.87 (8.85)	<0.001
WBC	5.90 (1.53)	5.88 (1.52)	5.92 (1.56)	5.91 (1.54)	0.582
NHR	0.57 (0.08)	0.57 (0.08)	0.57 (0.08)	0.57 (0.09)	0.026
LHR	1.47 (0.62)	1.47 (0.63)	1.49 (0.61)	1.40 (0.59)	<0.001
MHR	0.30 (0.15)	0.31 (0.15)	0.30 (0.15)	0.29 (0.14)	<0.001
GGT (IU/L)	34.38 (45.12)	34.26 (39.07)	35.69 (43.83)	31.51 (64.45)	0.063
ALT (IU/L)	23.95 (15.97)	24.57 (16.56)	24.14 (15.56)	21.22 (14.42)	<0.001
AST (IU/L)	24.07 (10.03)	23.68 (10.04)	24.36 (9.61)	24.77 (10.94)	<0.001
DBIL (umol/L)	3.44 (1.81)	3.38 (1.57)	3.50 (2.11)	3.48 (1.81)	0.013
IDBil (umol/L)	9.25 (4.13)	9.10 (4.25)	9.43 (4.18)	9.36 (3.58)	0.004
TBil (umol/L)	12.52 (5.47)	12.29 (5.42)	12.75 (5.72)	12.74 (4.93)	<0.001
5-NT (IU/L)	3.26 (2.25)	3.32 (2.19)	3.32 (2.39)	2.87 (2.06)	<0.001
GLB (g/L)	31.56 (4.09)	31.40 (4.14)	31.58 (4.02)	32.12 (4.03)	<0.001
UA (mg/dL)	347.98 (93.72)	351.45 (95.48)	349.46 (92.65)	331.65 (88.14)	<0.001
BUN (mg/dL)	5.73 (1.47)	5.65 (1.41)	5.76 (1.42)	5.95 (1.74)	<0.001
SCr (mg/dL)	69.23 (17.60)	69.61 (17.55)	68.65 (16.65)	69.28 (19.91)	0.039
HDLC (mg/dL)	1.39 (0.32)	1.37 (0.32)	1.38 (0.32)	1.44 (0.32)	<0.001
LDLC (mg/dL)	2.99 (0.78)	2.96 (0.75)	3.04 (0.79)	2.99 (0.85)	<0.001
FPG (mg/dL)	5.59 (1.51)	5.48 (1.44)	5.63 (1.51)	5.88 (1.71)	<0.001
HbA1c (mg/dL)	5.94 (0.98)	5.83 (0.94)	5.98 (0.97)	6.17 (1.07)	<0.001
TyG	8.78 (0.64)	8.75 (0.66)	8.81 (0.62)	8.82 (0.59)	<0.001
WWI	10.35 (0.68)	10.18 (0.63)	10.42 (0.65)	10.77 (0.76)	<0.001
NHHR	2.93 (0.94)	2.95 (0.95)	2.97 (0.94)	2.77 (0.92)	<0.001
Smoking, n (%)					
No	8353.00 (82.36)	4202.00 (81.54)	2933.00 (82.30)	1218.00 (85.47)	0.003
YES	1789.00 (17.64)	951.00 (18.46)	631.00 (17.70)	207.00 (14.53)	
Drinking, n (%)					
No	7525.00 (74.20)	3716.00 (72.11)	2665.00 (74.78)	1144.00 (80.28)	
Light drinking	2346.00 (23.13)	1298.00 (25.19)	793.00 (22.25)	255.00 (17.89)	<0.001
Heavy drinking	271.00 (2.67)	139.00 (2.70)	106.00 (2.97)	26.00 (1.82)	
Physical activity, n (%)					
Low	3427.00 (37.10)	1604.00 (34.22)	1219.00 (37.59)	604.00 (46.21)	
Moderate	3110.00 (33.67)	1689.00 (36.04)	1089.00 (33.58)	332.00 (25.40)	<0.001
High	2700.00 (29.23)	1394.00 (29.74)	935.00 (28.83)	371.00 (28.39)	
Hypertension, n (%)					
NO	6417.00 (64.72)	3577.00 (71.14)	2190.00 (62.68)	650.00 (46.66)	<0.001
YES	3498.00 (35.28)	1451.00 (28.86)	1304.00 (37.32)	743.00 (53.34)	
Diabetes, n (%)					
NO	8625.00 (87.14)	4471.00 (89.01)	3027.00 (86.73)	1127.00 (81.37)	<0.001

YES	1273.00 (12.86)	552.00 (10.99)	463.00 (13.27)	258.00 (18.63)	
VAA (cm^2)	183.74 (93.79)	178.59 (91.65)	184.95 (93.33)	203.16 (101.65)	<0.001
LFC (%)	7.70 (5.61)	7.63 (5.60)	7.69 (5.53)	8.00 (5.92)	0.238
BMD	120.98 (37.41)	150.44 (23.15)	101.88 (11.35)	62.24 (14.06)	<0.001

The data are presented as the mean (SD) or n (%). All estimates were obtained from complex survey designs, analysis of variance, or χ^2 tests where appropriate. MASLD, metabolic dysfunction–associated steatotic liver disease; BMI, body mass index; WHR, waist-to-hip ratio; WHTR, waist-to-height ratio; BAI, body adiposity index; ABSI, a body shape index; RFM, relative fat mass; VAI, visceral adiposity index; SBP, systolic pressure; DBP, diastolic pressure; HB, hemoglobin; PLT, platelet; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; WBC white blood cells; NHR, neutrophil to high-density lipoprotein cholesterol ratio; LHR, lymphocyte-to-high density lipoprotein cholesterol ratio; MHR, monocyte to high density lipoprotein-cholesterol ratio; GGT, γ-glutamyl transpeptidase; ALT, alanine aminotransferase; AST, aspartate Aminotransferase; DBiL, direct bilirubin; IDBil, indirect bilirubin; TBil, total bilirubin; 5-NT, 5' -nucleotidase; GLB, globulin; UA, uric acid; BUN, blood urea nitrogen; SCr, serum creatinine; HDLC, high-density lipoprotein cholesterol; LDLC, low-density lipoprotein cholesterol; FPG, fasting blood glucose; HbA1c, glycosylated hemoglobin; TyG, triglyceride-glucose; WWI, weight-adjusted waist index; NHHR, non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio; VAA, visceral adipose area; LFC, liver fat content; BMD, bone mineral density

Participants with osteoporosis were significantly older (mean age: 69.26 ± 9.45 years) compared with those in the osteopenia (59.07 ± 8.24 years) and normal (53.15 ± 6.44 years, $P < 0.001$) groups. The proportion of female participants was notably higher in the osteoporosis group (69.05%) than in the control group (47.47%; $P < 0.001$). BMI was similar across all groups ($P = 0.307$), whereas indicators reflecting central adiposity, including WC, WHR, WHTR, BAI, ABSI, RFM, and WWI, were significantly higher in the osteoporosis group than in the other groups (all $P < 0.001$). In contrast, the visceral adiposity index (VAI) did not differ significantly between the groups ($P = 0.279$).

Regarding metabolic parameters, participants with osteoporosis exhibited significantly higher levels of FPG, HbA1c, and TyG index than those in the other groups (all $P < 0.001$). Significant differences were also observed in lipid profiles, with higher HDL-C and altered NHHR levels among individuals with osteoporosis (all $P < 0.001$). Additionally, UA, liver enzymes (ALT and AST), total bilirubin (TBil), and BUN levels were significantly different between groups (all $P < 0.05$).

Lifestyle factors differed significantly among the groups, with a greater proportion of non-smokers, non-drinkers, and individuals reporting low physical activity levels in the osteoporosis group (all $P < 0.001$). The prevalence of hypertension and diabetes was notably higher in individuals with osteoporosis, indicating a more adverse cardiovascular and metabolic risk profile (both $P < 0.001$). Finally, as expected, the osteoporosis group presented markedly lower mean BMD levels compared to osteopenia and normal groups (62.24 ± 14.06 mg/cm^3 vs. 101.88 ± 11.35 mg/cm^3 and 150.44 ± 23.15 mg/cm^3, respectively, $P < 0.001$).

Overall, these results indicate that reduced BMD, particularly osteoporosis, is significantly associated with older age, female sex, increased central adiposity, adverse metabolic profiles, and a higher prevalence of comorbidities.

3.2 Subgroup Differences in the Prevalence of Osteopenia and Osteoporosis

Considerable variations were observed in the prevalence of osteopenia and osteoporosis according to sex, age, and BMI (Fig. 2). While osteopenia prevalence was similar between sexes and BMI groups, osteoporosis was significantly more prevalent among women (18.94%) than among men (8.92%, $P < 0.001$), emphasizing the greater susceptibility of females to advanced bone loss. Osteoporosis prevalence increased markedly with age, rising from 1.74% among individuals aged 45 – 54 years to 61.32% among those aged ⩾75 years ($P < 0.001$). Osteopenia was more prevalent than osteoporosis in the 45 – 54-, 55 – 64-, and 65 – 74-year age groups ($p < 0.05$), but this trend was reversed in the oldest age group, where osteoporosis prevalence exceeded that of osteopenia. In the BMI-specific analyses, the prevalence of osteoporosis was significantly higher among underweight participants (26.63%) than among normal-weight, overweight, and obese individuals (13.59%, 13.24%, and 14.73%, respectively; $P < 0.001$). Conversely, the prevalence of osteopenia was similar across all the BMI categories.

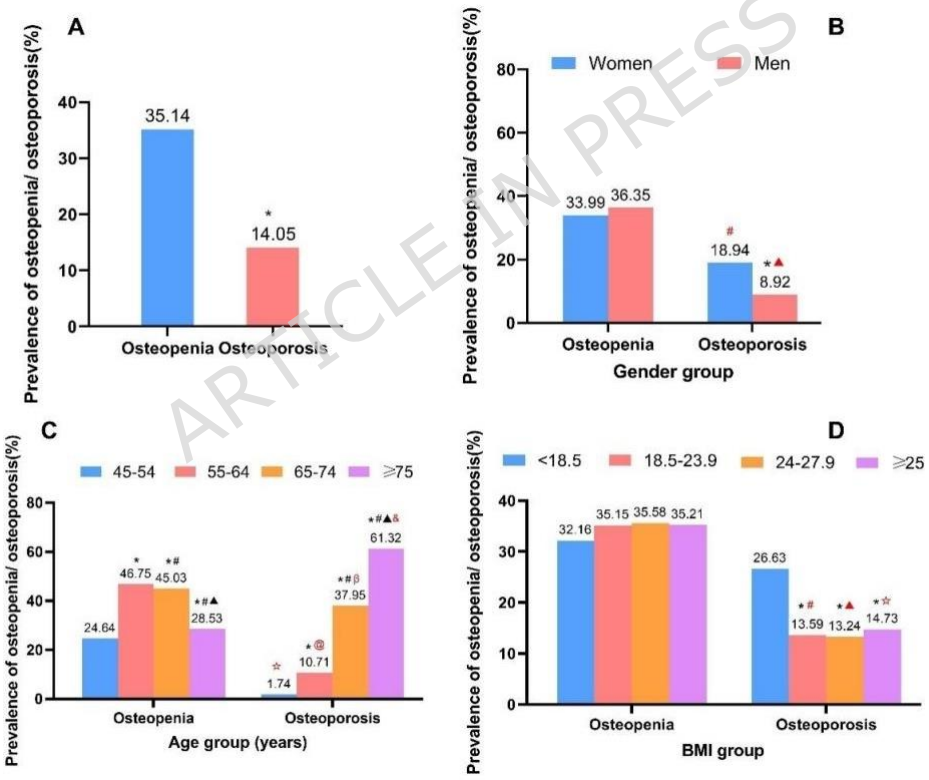

Fig. 2. Prevalence of osteopenia and osteoporosis across sex, age, and BMI subgroups. A. Overall prevalence of osteopenia and osteoporosis. B. Sex-specific prevalence of osteopenia and osteoporosis. C. Age-specific prevalence of osteopenia and osteoporosis. D. BMI-specific prevalence of osteopenia and osteoporosis.

3.3 Metabolic predictors Associated with Osteopenia

Univariate logistic regression analyses (Table 2) revealed that increased age, central adiposity indicators (including WHTR, WHR, BAI, ABSI, RFM, and WWI), elevated systolic and diastolic blood pressure, MCH, LDL cholesterol, HbA1c, and TyG index were significantly positively associated with osteopenia (all $P < 0.05$). In contrast, alcohol consumption (odds ratio [OR] = 0.87, 95% confidence interval [CI]: 0.79 – 0.96, P = 0.0058) and moderate and high levels of physical activity (moderate: OR = 0.85, 95% CI: 0.76 – 0.94, P = 0.002; high: OR = 0.88, 95% CI: 0.79 – 0.99, P = 0.028) demonstrated a significant inverse association with osteopenia.

After adjustment for sex, BMI, VAI, blood pressure parameters, hematological indices, lipid and glycemic profiles, lifestyle factors, and comorbidities (Table 2, Fig. 3), multivariable logistic regression identified older age (OR = 1.12, 95% CI: 1.10 – 1.13, P < 0.001), WHTR (OR = 1.17, 95% CI: 1.14 – 1.19, P < 0.001), WHR (OR = 1.06, 95% CI: 1.05 – 1.07, P < 0.001), BAI (OR = 1.09, 95% CI: 1.06 – 1.13, P < 0.001), ABSI (OR = 3.29, 95% CI: 2.71 – 3.98, P < 0.001), RFM (OR = 1.21, 95% CI: 1.18 – 1.25, P < 0.001), WWI (OR = 2.12, 95% CI: 1.88 – 2.39, P < 0.001), LDL cholesterol (OR = 1.30, 95% CI: 1.10 – 1.54, P = 0.002), HbA1c (OR = 1.20, 95% CI: 1.07 – 1.33, P = 0.001), and TyG index (OR = 1.87, 95% CI: 1.52 – 2.31, P < 0.001) as independent predictors of osteopenia. These findings collectively indicate that advancing age, increased central adiposity, insulin resistance, dyslipidemia, and impaired glycemic control are key metabolic predictors contributing to the development of osteopenia.

Table 2. Predictors for osteopenia

Variables	Univariable OR (95%CI)	*P* Value	Multivariable OR (95%CI)[a]	*P* Value
Sex, n (%)	0.92 (0.84–1)	0.056		
Age (years)	1.12 (1.11–1.13)	<0.001	1.12 (1.10–1.13)	<0.001
BMI (kg/m^2)	1 (0.99–1.02)	0.875		
WHTR	75.81 (32.15–178.73)	<0.001	1.17 (1.14–1.19)	<0.0001
WHR	13.48 (7.27–24.97)	<0.001	1.06 (1.05–1.07)	<0.0001
BAI	1.04 (1.03–1.05)	<0.001	1.09 (1.06–1.13)	<0.0001
ABSI	1.07 (1.04–1.1)	<0.001	3.29 (2.71–3.98)	<0.0001
RFM	1.03 (1.02–1.04)	<0.001	1.21 (1.18–1.25)	<0.0001
VAI	1.01 (0.99–1.03)	0.297		
SBP (mmHg)	1.01 (1.01–1.02)	<0.001	1.01 (1.01–1.02)	<0.0001
DBP (mmHg)	1 (1–1.01)	0.011	1.01 (1.00–1.01)	0.0291
HB (g/L)	1 (0.99–1)	0.069		
PLT	1 (1–1)	<0.001		
MCH (pg)	1.03 (1.01–1.05)	0.003	1.08 (1.04–1.11)	<0.0001
MCHC (g/L)	0.99 (0.99–0.99)	<0.001	0.99 (0.99–1.00)	0.1080
WBC	1.01 (0.99–1.04)	0.316		
NHR	0.49 (0.29–0.84)	0.01	0.47 (0.11–2.02)	0.3124
LHR	1.05 (0.98–1.12)	0.194		
MHR	0.91 (0.67–1.23)	0.537		
GGT (IU/L)	1 (1–1)	0.124		

ALT (IU/L)	1 (1–1)	0.238		
AST (IU/L)	1.01 (1–1.01)	0.002		
5-NT (IU/L)	1 (0.98–1.02)	0.943		
GLB (g/L)	1.01 (1–1.02)	0.056		
UA (mg/dL)	1 (1–1)	0.34		
BUN (mg/dL)	1.06 (1.03–1.09)	<0.001	1.05 (1.00–1.10)	0.0503
SCr (mg/dL)	1 (0.99–1)	0.012		
HDLC (mg/dL)	1.08 (0.94–1.23)	0.273		
LDLC (mg/dL)	1.13 (1.07–1.2)	<0.001	1.30 (1.10–1.54)	0.0018
TG (mg/dL)	1.01 (0.98–1.04)	0.731		
FPG (mg/dL)	1.07 (1.04–1.1)	<0.001		
HbA1c (mg/dL)	1.18 (1.1–1.26)	<0.001	1.20 (1.07–1.33)	0.0011
TyG	1.16 (1.08–1.25)	<0.001	1.87 (1.52–2.31)	<0.0001
ALB	1.57 (1.3–1.89)	<0.001	1.29 (0.94–1.76)	0.1125
WWI	1.81 (1.69–1.95)	<0.001	2.12 (1.88–2.39)	<0.0001
NHHR	1.03 (0.98–1.07)	0.29		
Smoking, n (%)	0.95 (0.85–1.06)	0.372		
Drinking, n (%)	0.87 (0.79–0.96)	0.0058	0.86 (0.71–1.05)	0.1510
Physical activity, n (%)				
Moderate	0.85 (0.76–0.94)	0.002	0.85 (0.70–1.04)	0.1172
High	0.88 (0.79–0.99)	0.028	0.95 (0.77–1.16)	0.6032
Hypertension, n (%)	1.47 (1.34–1.61)	<0.001	1.00 (0.80–1.25)	0.9913
Diabetes, n (%)	1.24 (1.09–1.41)	0.001	1.22 (0.95–1.57)	0.1233
VAA (cm^2)	1 (1–1)	0.007		
LFC (%)	1 (0.99–1.01)	0.708		

OR, odds ratio; CI confidence interval.

[a] Adjusted for: Sex, BMI, VAI, SBP, DBP, HB, PLT, WBC, LHR, MHR, GGT, ALT, AST, IDBil, 5-NT, GLB, UA, SCR, HDLC, NHHR, Smoking, VAA, and LFC

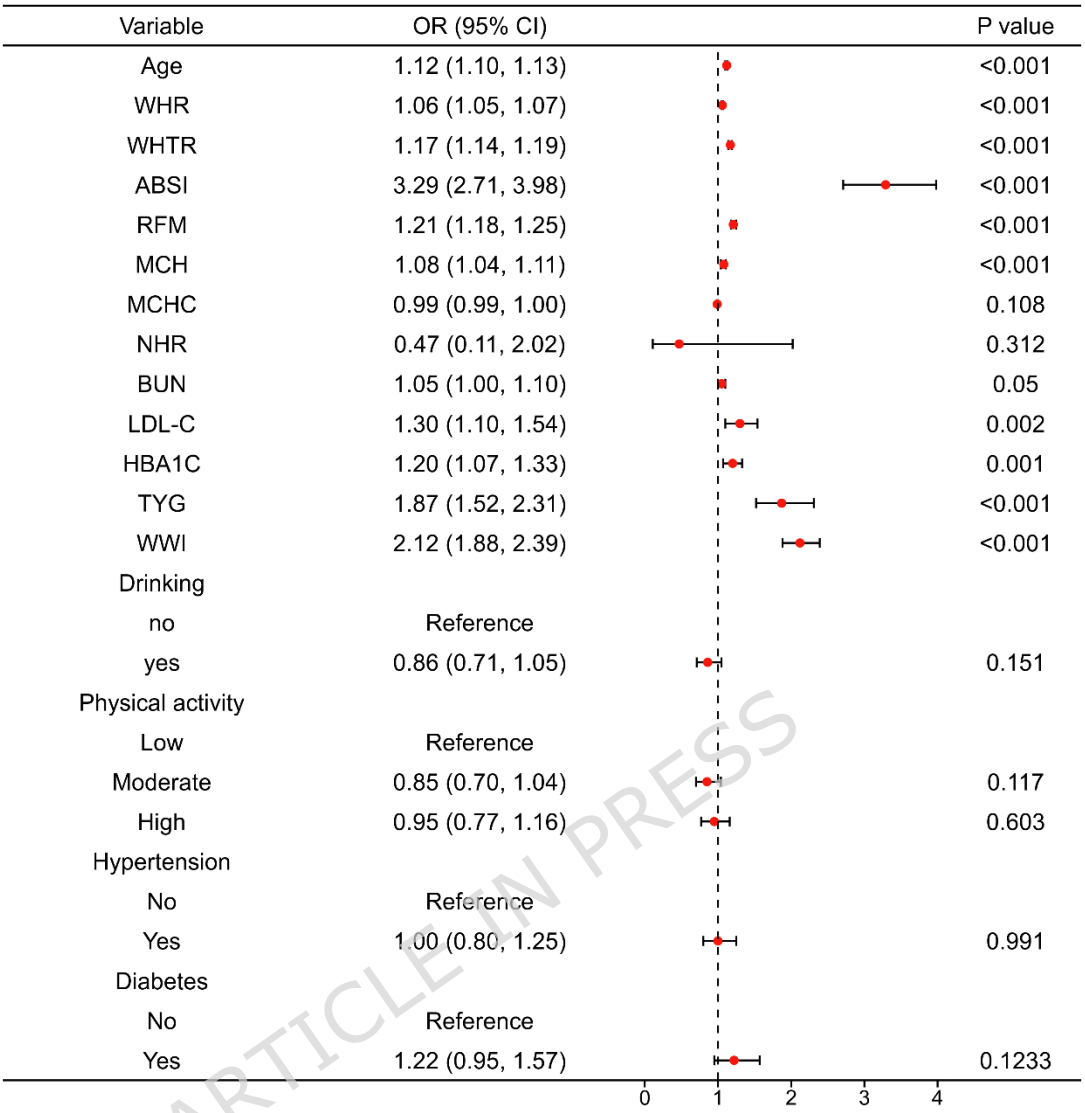

Fig. 3. Forest plot of factors associated with osteopenia.

3.4 Metabolic Predictors Associated with Osteoporosis

A range of variables were found to be significantly associated with osteoporosis in the univariate logistic regression analyses (Table 3). These included male sex, advanced age, elevated central adiposity indices (WHR, ABSI, RFM, and WWI), higher blood pressure, increased LDL cholesterol level, HbA1c level, TyG index, hypoalbuminemia, and the presence of diabetes mellitus (all $P < 0.05$).

Subsequent multivariate logistic regression analysis (Table 3, Fig. 4), adjusted for confounders such as sex, BMI, blood pressure, hematological indices, liver and renal function, lipid and glycemic markers, lifestyle factors, and comorbidities, identified several independent predictors. Specifically, osteoporosis was significantly associated with: WHR (OR = 1.10, 95% CI: 1.07–1.13, $P < 0.001$), ABSI (OR = 3.23, 95% CI: 2.24–4.65, $P < 0.001$), RFM (OR = 1.37, 95% CI: 1.29–1.45, $P < 0.001$), WWI (OR = 3.29, 95% CI: 2.62–4.14, $P < 0.001$), HbA1c (OR = 1.23, 95% CI: 1.01–1.49, $P = 0.042$), TyG index (OR = 1.73, 95% CI: 1.17–2.56, $P = 0.006$), hypoalbuminemia (OR = 3.30,

95% CI: 1.69–6.44, P < 0.001), diabetes mellitus (OR = 1.57, 95% CI: 1.07–2.30, P = 0.021), and reduced physical activity levels (Moderate: OR = 0.49, 95% CI: 0.35–0.69, P < 0.001; High: OR = 0.70, 95% CI: 0.51–0.98, P = 0.036). These results emphasize that central adiposity, metabolic disturbances (insulin resistance and hyperglycemia), poor nutritional status, and inadequate physical activity are significant independent contributors to the risk of osteoporosis.

Table 3. Predictors for osteoporosis

Variables	Univariable OR (95%CI)	*P* Value	Multivariable OR (95%CI)[a]	*P* Value
Male, n (%)	0.4 (0.36–0.46)	<0.001		
Age (years)	1.24 (1.22–1.25)	<0.001		
BMI (kg/m^2)	0.99 (0.97–1)	0.14		
WHR	25.49 (10.83–59.96)	<0.001	1.10 (1.07–1.13)	<0.0001
ABSI	1.09 (1.05–1.13)	<0.001	3.23 (2.24–4.65)	<0.0001
RFM	1.12 (1.11–1.13)	<0.001	1.37 (1.29–1.45)	<0.0001
VAI	1.02 (0.99–1.04)	0.167		
SBP (mmHg)	1.04 (1.03–1.04)	<0.001		
DBP (mmHg)	0.99 (0.99–1)	0.003		
HB (g/L)	0.97 (0.97–0.98)	<0.001		
PLT	1 (0.99–1)	<0.001		
MCH (pg)	1.06 (1.03–1.09)	<0.001		
MCHC (g/L)	0.97 (0.96–0.97)	<0.001		
WBC	1.01 (0.97–1.05)	0.565		
NHR	1.03 (0.5–2.15)	0.928		
MHR	0.4 (0.26–0.62)	<0.001	2.76 (0.74–10.26)	0.1305
GGT (IU/L)	1 (1–1)	0.04		

ALT (IU/L)	0.98 (0.98–0.99)	<0.001		
AST (IU/L)	1.01 (1–1.01)	0.001		
DBil (umol/L)	1.02 (1–1.03)	0.055		
5-NT (IU/L)	0.88 (0.84–0.91)	<0.001		
GLB (g/L)	1.04 (1.03–1.06)	<0.001		
UA (mg/dL)	1 (1–1)	<0.001		
BUN (mg/dL)	1.14 (1.09–1.18)	<0.001	1.08 (0.98–1.19)	0.1185
SCr (mg/dL)	1 (1–1)	0.542		
HDLC (mg/dL)	1.86 (1.56–2.23)	<0.001	0.68 (0.41–1.15)	0.1533
LDLC (mg/dL)	1.05 (0.97–1.14)	0.191		
HbA1c	1.37 (1.27–1.48)	<0.001	1.23 (1.01–1.49)	0.0417
TyG	1.19 (1.08–1.31)	0.001	1.73 (1.17–2.56)	0.0062
ALB	6.24 (4.73–8.23)	<0.001	3.30 (1.69–6.44)	0.0005
WWI	3.65 (3.29–4.05)	<0.001	3.29 (2.62–4.14)	<0.0001
NHHR	0.8 (0.75–0.86)	<0.001	1.08 (0.84–1.38)	0.5365
Smoking	0.75 (0.64–0.88)	0.001	1.29 (0.93–1.80)	0.1312
Drinking	0.64 (0.55–0.73)	<0.0001	0.87 (0.64–1.19)	0.3829
Physical activity, n (%)				
Moderate	0.52 (0.45–0.61)	<0.001	0.49 (0.35–0.69)	<0.0001
High	0.71 (0.61–0.82)	<0.001	0.70 (0.51–0.98)	0.0360
Hypertension, n (%)	2.82 (2.5–3.18)	<0.001	0.82 (0.57–1.18)	0.2900
Diabetes, n (%)	1.85 (1.58–2.18)	<0.001	1.57 (1.07–2.30)	0.0212

VAA (cm^2)	1 (1–1)	<0.001
LFC (%)	1.01 (1–1.02)	0.08

OR, odds ratio; CI confidence interval.

[a] Adjusted for: Sex, BMI, VAI, SBP, DBP, HB, PLT, MCH, MCHC, WBC, NHR, GGT, ALT, AST, IDBil, 5-NT, GLB, UA, SCR, LDLC, VAA, and LFC

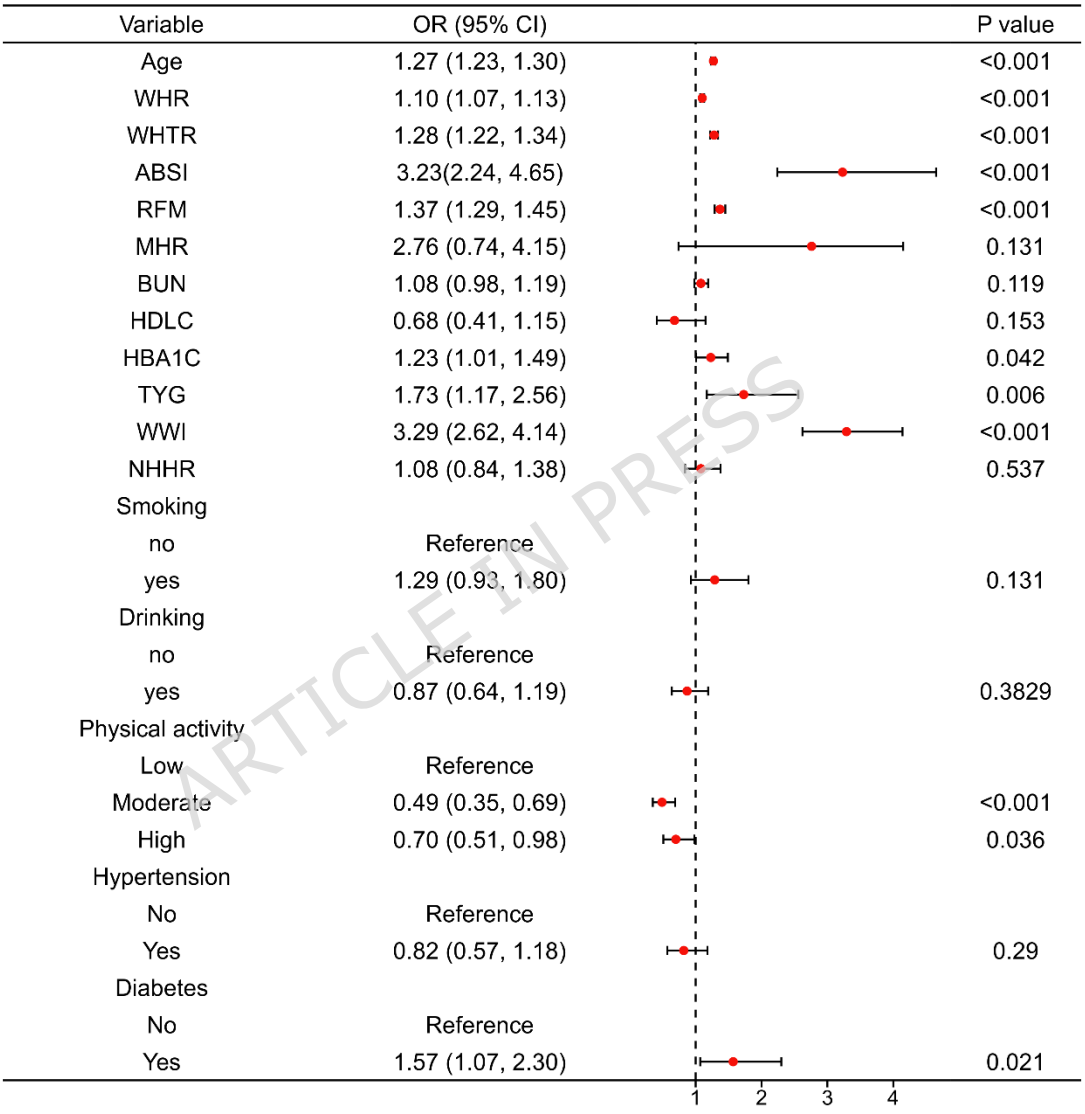

Fig. 4. Forest plot of factors associated with osteoporosis.

3.5 Sex-Specific Comparison of Metabolic Indicators for Osteopenia and Osteoporosis Prediction

The predictive abilities of various metabolic indicators for osteopenia and osteoporosis were assessed using ROC curve analyses, and the corresponding AUC values (95%CI) were calculated for both females and males (Fig. 5). For osteopenia prediction (Fig. 5A), WWI exhibited the highest discriminatory power in both sexes, with AUCs of 0.638 for females and 0.591 for males. Other indices, including RFM

(female AUC = 0.605, male AUC = 0.546), WHTR (female AUC = 0.603, male AUC = 0.545), and WHR (female AUC = 0.603, male AUC = 0.547), showed moderate predictive performance, with AUC values ranging from 0.558 to 0.593 for females and from 0.504 to 0.582 for males. TyG and BAI demonstrated relatively lower AUCs, with TyG showing the least discriminatory power (female AUC = 0.589, male AUC = 0.504). In contrast, the predictive performance for osteoporosis (Fig. 5B) was notably stronger. WWI showed the best discriminative ability, with AUCs of 0.785 for females and 0.679 for males, followed closely by RFM (female AUC = 0.723, male AUC = 0.577) and ABSI (female AUC = 0.716, male AUC = 0.582). Other indices such as WHTR, WHR, BAI, and TyG demonstrated lower AUCs, with values ranging from 0.544 to 0.606 for females and from 0.518 to 0.571 for males. Overall, all the evaluated indices performed better in predicting osteoporosis than osteopenia, with WWI consistently exhibiting the strongest predictive utility under both conditions. RFM and ABSI also showed considerable value in osteoporosis risk prediction, whereas WHR and the TyG index were less effective in distinguishing between conditions.

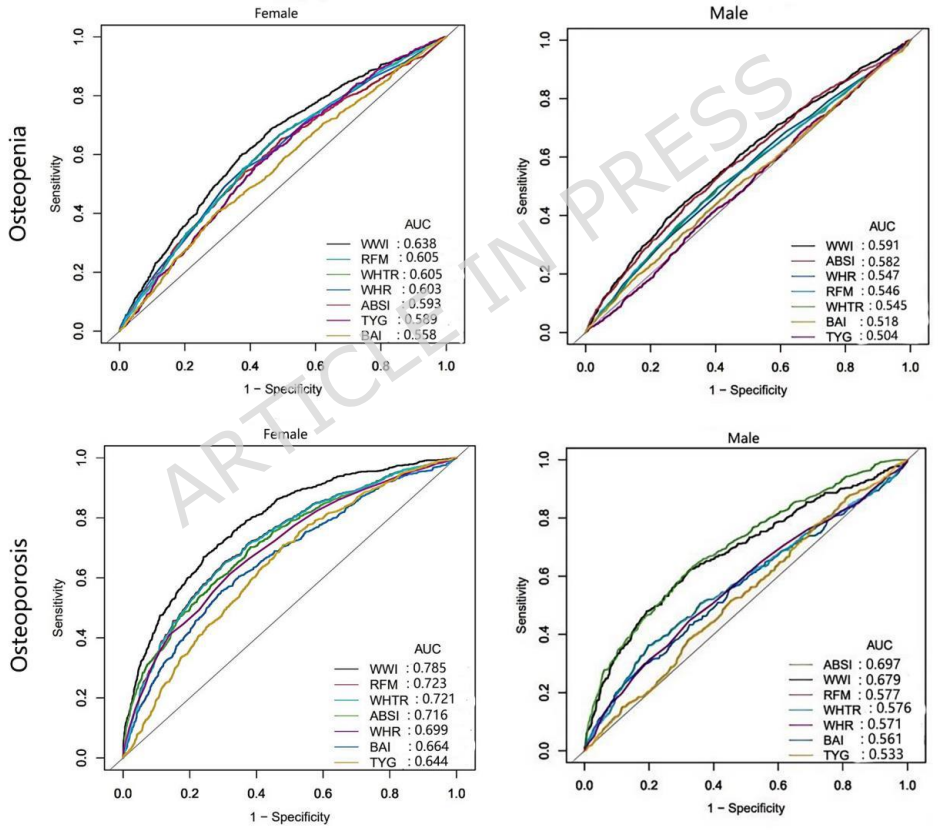

Fig. 5. Sex-Stratified ROC Curves for WWI, ABSI, WHTR, WHR, RFM, TyG, and BAI in Predicting Osteopenia and Osteoporosis Risk.

Discussion

Osteoporosis and osteopenia are not only considered skeletal disorders but also closely associated with fat distribution and systemic metabolic dysfunction. In this large cohort of more than 10,000 Chinese adults, a significant negative correlation was

observed between WWI and lumbar spine BMD, indicating that WWI may serve as a useful indicator of osteoporosis risk. Other novel obesity related indices, including ABSI, RFM, and BAI, were identified as negative predictors of BMD. Furthermore, the TyG index, an alternative marker of insulin resistance, had also been found to be closely associated with osteoporosis and bone loss. These findings support the hypothesis that metabolic dysfunction, particularly central obesity and insulin resistance, may interfere with bone maintenance, although the underlying mechanisms needed further investigation.

Although obesity is traditionally regarded as a risk factor for conditions such as cardiovascular disease, hypertension, and diabetes, accumulating evidence suggests it may exert a protective influence on BMD [33,34]. Several studies have indicated that higher fat mass and elevated BMI are often associated with increased bone mineral density (BMD), supporting the concept of the so-called "obesity paradox" in relation to osteoporosis [35]. Consistent with prior reports, our subgroup analysis demonstrated that individuals with a BMI below 18.5 kg/m² had the highest prevalence of osteoporosis, whereas those with a BMI ≥18.5 kg/m², including overweight participants, showed a significantly reduced risk. Insufficient body weight could negatively impact BMD, potentially due to a decline in muscle mass that accelerates bone deterioration. Conversely, other research has indicated that excessive adiposity, particularly in the abdominal region, may have adverse effects on BMD and fracture risk [36]. In our analysis, RFM showed a strong inverse relationship with lumbar spine BMD. One possible explanation for the obesity paradox is that BMI does not effectively distinguish between muscle and fat tissue [33]. Studies from rural China suggest that low BMI may coexist with central obesity and sarcopenia, creating a double burden of bone fragility [37].

Our findings align with prior international research indicating that central adiposity is more detrimental to bone health than generalized obesity. WWI, a marker of abdominal fat accumulation, was found to be negatively correlated with total BMD, displaying a clear saturation effect in American adolescents [38]. To our knowledge, this study is the first to investigate the association between WWI and QCT-derived BMD in a middle-aged and elderly Chinese population undergoing routine health examinations. Research from Korea and Japan confirmed that higher WHTR and ABSI may predict lower BMD and higher fracture risk [39,40]. These indices, which reflect abdominal adiposity independent of height, weight, and BMI, have also demonstrated strong cardiometabolic predictive value. Within our study, WWI exhibited the strongest association with BMD and the best discriminatory performanc for individuals with osteoporosis.

A significant methodological strength of this research is the implementation of QCT for volumetric BMD assessment. Unlike DXA, which provides areal BMD and is susceptible to soft tissue artifacts, spinal degeneration, and aortic calcification, QCT facilitates high-resolution evaluation of trabecular bone microstructure, making it particularly effective in identifying early-stage metabolic bone disorders [41,42]. This distinction is especially applicable to centrally obese individuals in whom DXA

readings may be distorted by excess abdominal fat. Using QCT, our study captured the nuanced skeletal impact of adiposity and metabolic abnormalities with greater precision, reinforcing the observed associations and enhancing their clinical interpretability. Given its ability to detect early deterioration before overt bone loss, QCT may function as a valuable tool for early diagnosis, particularly in metabolically high-risk populations that may not meet DXA-based thresholds.

From a mechanistic perspective, central adiposity may influence bone loss through multiple biological pathways. Excess visceral fat promotes a chronic inflammatory state characterized by elevated tumor necrosis factor-alpha and interleukin-6, which can enhances the receptor activator of nuclear kB ligand (RANKL)-mediated osteoclastogenesis while suppressing osteoblastogenesis. This inflammatory milieu shifts mesenchymal stem cells toward adipogenic rather than osteogenic differentiation, increasing marrow adiposity and reducing bone formation. Obesity-related alterations in adipokines and insulin resistance further disrupt bone turnover, ultimately driving an imbalance between bone formation and resorption. [43,44]. Leptin resistance, reduced adiponectin, and increased marrow adiposity further compromise bone turnover and quality [45–47]. These mechanisms provide a biological explanation for the strong negative associations observed between central adiposity indices WWI , ABSI and BMD in this study.

Despite the well-known mechanical loading effects of body mass on bone, BMI was not associated with BMD in our cohort. BMI does not differentiate between lean mass and fat mass, nor does it reflect fat distribution. In populations with prevalent central adiposity, a substantial number of individuals may have sarcopenic obesity, characterized by reduced muscle mass and excess visceral fat. This phenotype weakens the mechanical stimuli traditionally attributed to body weight while amplifying metabolic and inflammatory stress on bone, counteracting any protective effect of increased body mass.

Additionally, the relationship between bone metabolism and metabolic dysfunction appears to be of critical importance. As an established indicator of insulin resistance, the TyG index is significantly correlated with increased risk of metabolic disorders, including diabetes and cardiovascular disease [48]. In our study, elevated TyG levels independently correlated with reduced BMD, a finding that aligns with previous evidence from postmenopausal populations in Europe and South America [49,50]. Chronic insulin resistance may impair osteoblast survival via oxidative stress and disruption of insulin-like growth factor 1 pathways [51]. Elevated TyG values are frequently observed in individuals with diabetes, in whom hyperglycemia facilitates the accumulation of advanced glycation end-products within collagen, thereby reducing bone toughness and increasing fracture susceptibility [52–54]. HbA1c, a recognized indicator of long-term glycemic status, has been linked to decreased bone density and greater cortical porosity in both diabetic and non-diabetic individuals [55,56]. Metabolic dysfunction is increasingly linked to oxidative and biochemical disturbances that contribute to bone degradation. Postmenopausal women demonstrate elevated oxidative stress, with excess reactive oxygen species (ROS) impairing mitochondrial function in osteoblasts and enhancing osteoclast differentiation [57]. Recent

metabolomic studies show that individuals with low BMD exhibit alterations in lipid, amino-acid, and energy metabolism pathways [58,59], indicating impaired collagen synthesis, disrupted membrane signaling, and compromised energetic capacity. Upon extending this "metabolism–bone" model to apparently healthy Chinese adults, these findings highlight the silent burden of metabolic osteopenia in this population.

Sex stratified ROC analyses demonstrated that WWI, RFM, and ABSI exhibited better discriminative performance for osteoporosis in females compared with males. This aligns with established sex differences in skeletal biology. Estrogen deficiency accelerates trabecular bone loss and magnifies the skeletal impact of visceral adiposity and metabolic dysfunction in women [60]. Men experience slower trabecular decline and exhibit weaker metabolic–skeletal coupling [61].

A comparison of our data with those of other studies reveals both consistency and innovation. While previous research has confirmed the link between obesity and osteoporosis in urban and older populations [62], few studies have integrated detailed metabolic profiling with QCT-based volumetric BMD. Our study fills this gap by providing an integrated perspective of anthropometric, metabolic, and lifestyle variables.

Our findings have several clinical implications. First, they emphasized that simple, inexpensive markers, such as WWI, ABSI, and TyG, can be used in routine clinical and community screening to identify individuals at an elevated risk of osteoporosis, even when BMI appears normal. A key advantage of WWI is its simplicity and feasibility. Unlike imaging-based adiposity metrics or biochemical biomarkers, WWI requires only waist circumference and body weight, two measurements that are quick, inexpensive, and consistently available in primary care settings. This practicality enables early identification of individuals with adverse fat distribution in large populations and supports the potential use of WWI in community and clinical screening programs.Second, they suggest that early lifestyle interventions targeting abdominal fat reduction and metabolic control may offer dual benefits to cardiovascular and skeletal health. Resistance training during weight reduction has been reported to beneficially influence both BMD and and metabolic marker levels [63]. Pharmacological interventions targeting insulin sensitivity (e.g. metformin and glucagon-like peptide-1 receptor agonists) may also hold promise for bone protection, although randomized evidence remains limited [64].

From a public health perspective, the findings of this study support the development of integrated strategies that address both metabolic and bone health, particularly in aging populations where the rising prevalence of type 2 diabetes and obesity parallels the increasing burden of osteoporosis [65]. Future clinical guidelines should consider incorporating metabolic risk markers into existing fracture risk assessment tools to enhance their predictive accuracy.

In summary, this large-scale cross-sectional study successfully identified a negative association between central obesity, insulin resistance-related indices (particularly the WWI), and BMD. The use of a representative cohort ensured that the findings reflected the diversity of the majority of the population in China. Moreover, the large sample size enabled subgroup analyses based on gender and age, enhancing

the broader applicability of our results. However, several limitations should be considered when interpreting the conclusions. First, the cross-sectional design precludes establishing a causal relationship between WWI, insulin resistance markers, and BMD. In addition, the single-center and predominantly urban sample limits the generalizability of our findings to rural populations, who may differ in lifestyle, nutritional status, physical labor intensity, and metabolic characteristics. The study cohort was derived from individuals undergoing routine health examinations, which may introduce selection bias because such participants are generally more health-conscious and may not fully represent the broader community population. These factors should be considered when interpreting the external validity of the results. Furthermore, several important bone-related biomarkers, such as serum 25-hydroxyvitamin D, parathyroid hormone , sex hormones (estradiol, testosterone), and bone turnover markers such as c-terminal telopeptide and N-terminal propeptide of type I procollagen, were not measured in this study. These factors are critical in bone metabolism, especially in postmenopausal women and older men. However, the absence of these variables is not uncommon in large-scale epidemiological studies, where practical and logistical constraints often limit the inclusion of comprehensive bone metabolism data[66,67]. Although the exclusion of these factors may introduce residual confounding, the study still provides valuable insights into the relationship between central obesity, metabolic markers, and bone health. Finally,the absence of fracture data in our study means we could not assess whether participants with higher WWI or insulin resistance-related indices had an elevated fracture risk compared to the general population.

In conclusion, our findings show that osteoporosis and osteopenia are strongly influenced by metabolic health, especially central adiposity and insulin resistance. Indicators such as WWI, ABSI, TyG, and HbA1c are accessible, predictive, and clinically relevant tools that should be considered in research and practice. Their integration into early screening protocols and risk models may help identify hidden high-risk individuals and inform more targeted and metabolically informed prevention strategies.

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Abbreviations

BMD Bone Mineral Density
QCT Quantitative Computed Tomography
DXA Dual-energy X-ray Absorptiometry
ISCD International Society for Clinical Densitometry
SD Standard Deviation
IQR Interquartile Range
VIF Variance Inflation Factor
BMI Body Mass Index
WWI Weight-adjusted Waist Index
ABSI A Body Shape Index
RFM Relative Fat Mass
VAI Visceral Adiposity Index
TyG Triglyceride-glucose Index
HbA1c Hemoglobin A1c
ROC Receiver Operating Characteristic
AUC Area Under the Curve
OR Odds Ratio
CI Confidence Interval
MASLD Metabolic-associated Steatotic Liver Disease
SBP Systolic Blood Pressure
DBP Diastolic Blood Pressure
UA Uric Acid
BUN Blood Urea Nitrogen
SCr Serum Creatinine
FPG Fasting Plasma Glucose
HDLC High-density Lipoprotein Cholesterol
LDLC Low-density Lipoprotein Cholesterol
NHHR Non-HDL-C to HDL-C Ratio
MCH Mean Corpuscular Hemoglobin
MCHC Mean Corpuscular Hemoglobin Concentration
HGB Hemoglobin
PLT Platelet
WBC White Blood Cell Count
ALB Albumin
ALT Alanine Aminotransferase
AST Aspartate Aminotransferase
5-NT 5’-Nucleotidase
DBiL Direct Bilirubin
IDBiL Indirect Bilirubin
TBil Total Bilirubin
LFC Liver Fat Content
VAA Visceral Adipose Area
RANKL Receptor Activator of Nuclear kB Ligand

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None.

Author contributions

All the authors contributed significantly to this study. LHW designed and supervised the study. PPY and MXC performed data collection and statistical analysis. JD interpreted the results and drafted the manuscript. LHY and YC critically revised the manuscript for intellectual content. All the authors have read and approved the final version of this manuscript.

Ethics approval and consent to participate

This study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the Second Affiliated Hospital of Chongqing Medical University (Approval Number: 2022(129)). Written informed consent was obtained from all participants prior to enrollment.

Consent for publication

Not applicable.

Availability of data and materials

The datasets generated and analyzed during the current study are not publicly available due to institutional restrictions but are available from the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interests.

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