The Impact of Waist Circumference on Incident of Cardiovascular Death During 9 Years of Follow Up in General Population

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Abstract

Background: Risk assessment is essential for the primary prevention of cardiovascular death among general population. Although studies have shown that waist circumference (WC) is positively associated with an increased risk of cardiovascular death among the general population, few studies have investigated the prognostic value of WC during a long-term follow up and the risk threshold of WC remains controversial. We aimed to investigate whether higher level of WC measurements was able to predict mortality in general population.

Methods: In this prospective cohort study, a total of 1521 consecutive subjects free of clinical cardiovascular disease were included. The end point was cardiovascular death. The Kaplan-Meier method was used to evaluate the cumulative risk of outcome at different WC levels, and compared by log-rank tests. Univariate and multivariable-adjusted Cox regression models were used to investigate the association between WC and outcomes.

Results: During a median follow up of 9.2 years, there were 265 patients died. Kaplan-Meier survival estimates indicated that the patients with higher levels of WC ($WC > 94\text{cm}$) had a significantly increased risk of cardiovascular death (log-rank $p < 0.001$). After adjustment for potential confounders, multiple COX regression models showed that higher level of WC was an independent predictor in developing cardiovascular death (HR 3.02; 95% CI: 1.88–3.83; $p < 0.001$). We saw a significant increase of (area under the curve) AUC in ROC (receiver operating characteristic) curve after addition of WC to a clinical model, for long-term cardiovascular death the increase of AUC 0.766 vs 0.642 (95% CI: 0.787–0.846 $p < 0.001$). The addition of WC to established risk factors significantly improved risk prediction of cardiovascular death (net reclassification index, and integrated discrimination improvement, all $p < 0.05$).

Conclusion: Higher level of WC is significantly associated with long-term cardiovascular death. WC may provide incremental prognostic value beyond traditional risks factors.

Background

Recent data from the United States showed that nearly a third of the world's population is now classified as overweight or obese [1]. Obesity has long been recognized associated with cardiovascular disease [2]. In epidemiological studies, body mass index (BMI) is often used to define overweight and obesity. However, BMI has a low sensitivity and there are large individual differences between body fat percentages, partly due to age, gender, and race [3]. Recent studies have indicated that the visceral adipose tissue (VAT) was the major threat to the cardiovascular risk [4]. Waist circumference (WC) has been reported associated with abnormal distribution of reactive fat. It has been reported that compared with BMI, WC can better reflect body fat distribution, more closely related to visceral fat tissue [5]. In particular, abdominal obesity is a better predictor of cardiovascular events than obesity measured by BMI alone [6]. Higher cardiometabolic risk is also related to the location of excess fat in visceral adipose tissue and ectopic reservoirs (such as muscle and liver), and when the ratio of fat to lean body mass

increases (for example, normal body weight for metabolic obesity) [7]. These data indicate that obesity may be more common than large epidemiological studies have shown and requires more urgent attention. Relying solely on BMI to assess its prevalence may hinder future interventions for obesity prevention and control. However, long-term research on WC related to predictive value is still very limited [8]. Moreover, the risk threshold of WC to predict cardiovascular death remains controversial [9] [10] [11]. Therefore, this study ought to investigate the prognostic value of WC on the cardiovascular death during a long-term follow up.

Methods

2.1 The study population

From January 2010 to December 2020, a total of 2162 people in Wan Shou Lu Street were included in the study. Among these participants, patients had a history of stroke or cardiovascular disease or cancer were excluded from the study. In the current study, patients were excluded if the patients could not give informed consent, had comorbidities lead to changes abdominal circumference measurement (secondary to chronic ascites, liver disease, cancer, intestinal obstruction, abdominal mass in the abdomen, pre-existing abdominal stoma, incisional hernia). Participants were also excluded if their blood sample or detailed data were not available. Candidates lost follow up were also excluded from the study. Finally, a total of 1521 patients were included in the present study. The flowchart of the study is listed in Figure 1.

2.2 Baseline measurement

The measures of the participants were collected at the time of registration. Anthropometric measurements include weight in kilograms, height in meters, hips circumference and WC. WC was measured on the mid-axillary line between the lowest border of the thoracic cage and the top of the iliac crest to the nearest 0.1 cm. BMI is calculated as $\text{weight}/\text{height}^2$. After resting for 5 minutes, use the right arm in a sitting position and use standard or automated equipment to measure blood pressure, and use the average of the two blood pressure measurements as the final blood pressure. The patient was considered to have hypertension with BP>140/90 mmHg or received antihypertensive medication. Hyperlipidemia is defined as a known but untreated dyslipidemia or current treatment with lipid-lowering drugs. Diabetes mellitus (DM) is defined as the presence of diabetic symptoms and resting plasma glucose concentration $\geq 200\text{mg/dL}$, fasting plasma glucose concentration $\geq 126\text{mg/dL}$, or 2-hour plasma glucose concentration $\geq 200\text{mg/dL}$, or the use of an oral hypoglycemic agent or insulin at the time of admission. If a current smoker report smoking in the last 30 days, it is defined as smoking.

Baseline data was collected at the time of enrollment. Blood samples were collected in the early morning. On the basis of protocol, the blood was obtained by the EDTA-anticoagulated plastic tubes. All the blood samples were centrifuged at 1000 g for 10 min and serum samples were stored at -80°C . Enzyme colorimetry was used to assess total cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C) levels. A two-point kinetic assay kit (CH 9702, Randox Laboratories Ltd, UK) was used

to determine the level of low-density lipoprotein cholesterol (LDL-C). The blood glucose level was measured using commercial reagents following standard procedures.

2.3 Outcome assessment

Patients were followed up until December 2020 or until the occurrence of cardiovascular death. All participants were followed up by analyses of clinical materials and telephone contact quarterly. The endpoint was cardiovascular death. Cardiovascular death was defined as deaths caused by coronary heart disease or stroke, as well as deaths that cannot be classified without evidence of the source. Unless a clear non-cardiac cause is established, all deaths are considered cardiac deaths. Based on the International Classification of Diseases (ICD) codes for confirmation of each cause of death or routine death registration. Excluding the participants who were lost to follow-up, we obtained follow-up of all patients until the primary outcome or date of censoring. The follow-up time was calculated from the date of cardiac death onset to the date of mortality occurrence or the date of the last follow-up. Written informed consent was obtained from all study participants, and the study was approved by the ethics committee of Chinses PLA General Hospital.

2.4 Statistical analysis

Patients were divided into three groups according to the levels of WC. Variables with a normal distribution are expressed as the mean $\pm$ SD, and in the case of non-normality, the medians are presented. Categorical data are expressed in counts or percentages. Differences in baseline characteristics between the three groups were evaluated by chi-square tests (categorical variables), analysis of variance as appropriate. The Kaplan-Meier method was used to evaluate the cumulative risk of outcome at different WC levels, and compared by log-rank tests. Cox multivariate analyses were used to evaluate the association of baseline WC levels with the study endpoints. The results are presented as the hazard ratios (HRs) and 95% confidence intervals (CIs) according to levels of WC. Two multivariate proportional hazards models were fitted. Model 1 contains the following variables: age, sex, BMI, current smokers, hypertension, diabetes, TC, TG, HDL-C, LDL-C. Variables were input into the model according to their statistical significance in univariate analysis. Model 2 was based on model 1, with the addition of WC. The relationship between the WC levels and the outcome is represented by the COX proportional hazard model with WC as a continuous variable and WC as a categorical variable. Increase of (area under the curve) AUC in ROC (receiver operating characteristic) curve was used to compare the predictive power of the target parameter. In addition, when WC was added to the established model, continuous net weight classification index (NRI) and comprehensive discrimination improvement (IDI) were generated to assess any improvement in prognostic prediction. SPSS 22.0 and R 4.0.0 (R Foundation for Statistical Computing) were used for descriptive data analysis. All statistical tests were 2-tailed, and p values <0.05 were considered statistically significant.

Results

3.1 Baseline characteristics

Baseline measurements of WC were available in 1521 patients. We divided the patients into three groups based on the levels of WC. The baseline clinical and laboratory characteristics of the study patients are presented in Table 1. The patients with a higher WC level were older, more likely to be female, more likely to be smokers. Moreover, they had a higher BMI, hips circumference, fasting blood glucose, 2hPBG level, HbA1c level, uric acid level. Also, they had a higher systolic blood pressure (SBP) and diastolic blood pressure (DBP) level. However, they had a lower HDL-C as well as TC level.

Table 1

Baseline characteristics of the subjects by the waist circumference level

Variables	Total (n=1521)	Waist circumference level			p value for trend
		≤82cm (n=438)	82-94cm (n=752)	94cm (n=331)	
Age, years	70.96±7.18	69.98±7.23	70.86±6.90	72.49±7.48	0.000
Male, n%	900(59.4)	336(76.71)	430(57.18)	134(40.48)	0.000
Current smokers, n (%)	449(29.52)	106(24.20)	215(28.59)	128(38.67)	0.000
Hypertension, n (%)	785(51.61)	174(39.73)	410(54.52)	201(60.73)	0.411
DM, n (%)	253(16.63)	84(19.18)	113(15.03)	56(16.92)	0.531
BMI (kg/m ²)	24.88±3.46	21.98±2.84	25.01±2.31	28.39±2.98	0.000
Hips circumference (cm)	97.97±8.36	90.88±7.40	98.54±5.24	106.06±7.35	0.000
Fasting blood glucose (mmol/L)	5.68±0.99	5.46±0.77	5.67±1.02	5.97±1.12	0.000
2hPBG (mmol/L)	8.08±3.29	7.19±2.73	8.19±3.32	9.00±3.61	0.000
HbA1c	5.86±1.045	5.70±0.62	5.87±1.17	6.04±1.17	0.000
SBP (mmHg)	137.42±19.13	132.77±17.30	138.43±17.89	141.36±19.51	0.000
DBP (mmHg)	76.97±9.96	73.84±8.74	77.79±9.28	79.23±9.21	0.000
TC (mmol/L)	5.29±0.99	5.37±0.97	5.29±1.01	5.16±0.95	0.016
HDL-C(mmol/L)	1.44±0.39	1.62±0.42	1.40±0.34	1.30±0.39	0.000
LDL-C(mmol/L)	3.26±0.85	3.24±0.84	3.30±0.92	3.17±0.85	0.055
TG (mmol/L)	1.63±0.82	1.42±0.76	1.65±0.82	1.85±0.82	0.341
Creatinine (umol/L)	74.46±22.22	70.64±24.98	74.42±19.77	79.62±22.64	0.271
Uric acid (umol/L)	309.06±89.27	282.59±74.79	307.99±91.22	346.61±89.54	0.000

3.2 Association between WC and prognosis of cardiovascular death

During the median follow up of 9.2 years, 265 participants had the occurrence of cardiovascular death. The death rate of the higher WC group was significantly higher than the lower group (23.3% VS 16.0% VS 15.0%, $p < 0.05$). Kaplan–Meier curves were used to show the cumulative event curves for cardiovascular death stratified according to WC levels: patients with higher level of WC were more likely to have a higher cardiovascular death rate (log-rank test, $p < 0.05$) (Fig. 2). Table 2 shows the univariate and multivariate Cox regression analysis of cardiovascular death predictors. After adjusting for potential confounding factors, higher WC level group ($WC > 94$ cm) had a significantly higher risk of cardiovascular death than that of lower WC level group (adjusted HR = 3.02; 95% CI: 1.88 to 3.83; $p = 0.001$). Age, BMI, hypertension, hips circumference, fasting blood glucose, 2hPBG, HbA1c, SBP, DBP, current smoking, Hypertension, DM, TC, TG, HDL-C, LDL-C, Creatinine, Uric acid were included in the confounding factors.

Table 2
Univariate and multivariate Cox regression analysis of cardiovascular death predictors

Variables	Univariable analysis			Multivariable analysis		
	Crude HR	95%CI	P Value	Crude HR	95%CI	P Value
Age, years	1.18	1.16 to 1.20	0.00	1.16	1.13 to 1.18	0.00
Male, n (%)	1.02	0.96 to 1.03	0.12	–	–	
BMI (kg/m ²)	0.97	0.96 to 0.98	0.00	0.95	0.90 to 1.01	0.09
Hips circumference(cm)	0.99	0.97 to 1.00	0.07	–	–	
Fasting blood glucose (mmol/L)	1.08	1.03 to 1.14	0.00	1.09	0.92 to 1.30	0.34
2hPBG(mmol/L)	1.03	0.99 to 1.06	0.14	–	–	
HbA1c	1.11	1.04 to 1.19	0.00	1.04	0.92 to 1.19	0.53
SBP (mmHg)	1.01	1.002 to 1.012	0.01	1.00	0.99 to 1.01	0.25
DBP (mmHg)	0.99	0.98 to 0.99	0.03	0.99	0.97 to 1.00	0.11
Current smokers, n (%)	1.56	1.45 to 1.69	0.00	1.59	1.46 to 1.77	0.00
Hypertension, n (%)	1.07	0.89 to 1.26	0.00	1.08	0.85 to 1.22	0.06
DM, n (%)	1.02	0.89 to 1.07	0.18	–	–	
TC (mmol/L)	0.75	0.68 to 0.85	0.00	1.28	0.64 to 2.57	0.49
HDL-C(mmol/L)	0.97	0.74 to 1.28	0.84	–	–	
LDL-C(mmol/L)	0.78	0.69 to 0.88	0.00	0.75	0.36 to 1.55	0.44
TG (mmol/L)	0.71	0.61 to 0.83	0.00	0.58	0.42 to 0.80	0.00
Creatinine (umol/L)	1.01	1.008 to 1.013	0.00	1.01	1.01 to 1.02	0.00
Uric acid (umol/L)	1.003	1.002 to 1.004	0.00	1.002	1.001 to 1.004	0.00
Group						
WC<82cm	1	–	–	1	–	–
WC:82-94cm	1.06	0.53 to 1.32	0.05	1.68	1.18 to 2.41	0.01
>94cm	1.59	0.97 to 2.57	0.07	3.02	1.88 to 3.83	0.00

3.3 Construction of the prediction models and the model performance.

We adjusted two prediction models to evaluate the prognostic value. Model 1 was adjusted by the clinical confounders (age, sex, BMI, hips circumference, fasting blood glucose, 2hPBG, HbA1c, SBP, DBP, Current smokers, hypertension, DM, TC, HDL-C, LDL-C, TG, creatinine, uric acid). Model 2 included model 1 with the addition of WC. Finally, to evaluate the effect of WC on the accuracy of cardiovascular death risk assessment, discriminatory abilities were compared the between models with and without WC. As shown in Table 3, adding WC can significantly increase the static value of C from 0.693 (95%CI:0.656–0.730) to 0.812 (95%CI:0.782–0.842). Compared with the clinical model, there is a significant difference ($p < 0.01$). In addition, adding WC category to Model 1 can significantly improve NRI = 0.178 (95% CI: 0.094–0.262, $p < 0.001$); IDI = 0.009 (95% CI: 0.003–0.014, $p < 0.001$) (Table 3).

Table 3
Reclassification and discrimination statistics for clinical outcomes by WC level

		Estimate (95%CI)	P value
C static	Model1	0.693 (95%CI:0.656–0.730)	<0.01
	Model1 + WC	0.812 (95%CI:0.782–0.842)	
NRI	Model1	REF	<0.001
	Model1 + WC	0.178 (95% CI: 0.094–0.262)	
IDI	Model1	REF	>0.001
	Model1 + WC	0.009 (95% CI: 0.003–0.014)	

ROC curve was used to analyze the predictive value of WC. ROC curve analysis indicated that, AUC was 0.693 (95%CI:0.656-0.730) for clinical modelmodel1, 0.812 (95%CI: 0.782-0.842) for clinical model including WCmodel2. ROC curve analysis showed there was a significant difference compared to the clinical model with WC ($p<0.001$) (Figure 4).

Discussion

4.1 Principal findings

Our study established that higher WC was a significant and independent predictor of cardiovascular death. In the current study, we observed that the WC level94 cm, was associated with future risk of cardiovascular death in about 1521 Chinese adults without cancer and cardiovascular disease at

baseline (Figure 5). The association is still significant after adjustment of other clinical confounders. In addition, our results suggested that the addition of WC to established cardiovascular risk factors may further improve risk stratification in general population. Our results provided updated information about the long-term prognostic role of WC in general population.

4.2 Limitations of BMI

Adipose tissue is now considered to be a key organ for the fates of excessive dietary lipids, which may determine whether it will maintain body homeostasis (metabolic healthy obesity) or whether it will produce inflammation/insulin resistance, which can have harmful cardiovascular consequences. Obesity, especially visceral obesity, can also cause various structural adaptations/changes in the structure/function of CV. Adipose tissue can now be regarded as an endocrine organ that coordinates important interactions with vital organs and tissues (such as the brain, liver, skeletal muscle, heart, and blood vessels themselves).

The most commonly used anthropometric tool for assessing relative weight and classifying obesity is BMI, which is expressed as the ratio of total body weight to the square of height (kg/m^2). Individuals with a BMI $<18.5 \text{ kg/m}^2$ are considered underweight, while individuals with a BMI between 18.5 and 24.9 kg/m^2 are classified as normal or acceptable weight. Individuals with a BMI between 25 and 29.9 kg/m^2 are classified as overweight, while those with a BMI of $\geq 30 \text{ kg/m}^2$ are obese. BMI itself is associated with clinical outcomes and mortality in a U or J type [12]. This inverse relationship has triggered controversy in the literature, called the "obesity paradox" [13]. Compared with non-obese patients, patients with elevated BMI with chronic diseases have a higher survival rate and fewer cardiovascular (CV) events [14]. Moreover, previous research reported that patients with an increased BMI were found to show lower mortality [15]. In addition, BMI cannot distinguish between weight gain due to high levels of lean body mass and fat body mass. Generally, excess body fat (BF) is more often associated with metabolic abnormalities than high levels of lean body mass. Another explanation for this paradox is the lack of control over the main individual differences in the regional BF distribution. Therefore, more and more scholars believe that the BMI has its limitations to fully capture cardiometabolic risk. It is partially related to the fact that BMI in isolation is an insufficient biomarker of abdominal adiposity [5]. By using the BMI, one must rely on the assumption that adipose tissue is distributed evenly over the body, which does not take into account the heterogeneity of regional body fat deposition [16].

The level of obesity must be considered in the risk stratification. In a recent meta-analysis of 2.88 million people, all levels of obesity combined were associated with increased mortality, with a hazard ratio of 1.18 (95% CI, 1.12-1.25). However, when analyzing separately, grade 1 obesity (table 1) is associated with a higher risk of death with a hazard ratio of 0.97 (95% CI, 0.90–1.04), compared with normal body weight. In contrast, serious obesity (grades 2 and 3) and risk of death (hazard ratio 1.34 – 95% CI, 1.21 – 1.47) [17]. The prognostic value of BMI needs to pay attention to the length of follow-up time. There was a J-type association between BMI and sudden cardiac death, and the lowest risk was observed within the normal weight range. However, in studies with a longer follow-up period, the increased risk of low BMI

was attenuated [18]. In other words, the obesity phenotype may change over time to reflect the increase in abdominal obesity. For example, Ian Janssen and colleagues studied the changes in waist circumference for a given BMI over a 30-year period in a Canadian sample³⁵. It is worth noting that for a given BMI, Canadians had a larger waist circumference in 2007 than in 1981. Specifically, the researchers observed that between 1981, men with a BMI of 25 kg/m² increased their waist circumference by 1.1 cm, and women with a BMI of 25 kg/m² increased theirs by 4.9 cm and 2007. Similarly, Sandra Albrecht and colleagues studied 36 long-term changes in waist circumference in the United States (1988-2007), the United Kingdom (1992-2008), China (1993-2011), and Mexico (1999-2012), and reported significant statistics Academic significance in all countries and in most subgroups, waist circumference values have increased relative to BMI. The result of one study involving more than 58 000 elderly persons, during a 5-year-follow-up, showed that increased mortality risks for elderly people with an increased WC—even across BMI categories— and for those who were classified as ‘underweight’ using BMI. Part of the reason why BMI cannot fully capture cardiometabolic risks is that BMI alone is an insufficient biomarker for the whole body. More importantly, the central abdominal fat mass does not explain the extreme changes in intra-abdominal (visceral) fat mass. Fat distribution between individuals [19]. Compared with BMI, waist circumference has a higher predictive value for cardiovascular death [20].

4.3 Visceral adipose tissue and the underlying mechanisms

Visceral adipose tissue has been proved to be independently associated with elevated CVD (cardiovascular disease) risk [21]. Data from several past epidemiological studies 30 years of experience shows that VAT is an independent sign of morbidity and mortality [22]. In some populations, WC has been found to be more predictive of overall mortality, coronary heart disease (CHD), and CVD mortality than BMI, However, prospective data on the impact of abdominal obesity on CVD incidence is still scarce. Many experimental studies support the potential connection between VAT and biological pathways that are important in the pathogenesis of multiple disease outcomes. Adipokines are biologically active molecules secreted by adipose tissue and are key components of these pathways, including inflammatory cytokines, angiogenic factors, lipid metabolites, and extracellular matrix components [23]. The secretion of adipokines among specific fat depots appears to be different [24], and compared with subcutaneous adipose tissue (SAT), VAT exhibits more pro-inflammatory and pro-angiogenic gene expression. In addition, compared with SAT, small arteries in VAT are more likely to exhibit endothelial dysfunction [25], indicating that VAT has a potentially toxic effect on the vasculature. Visceral adipocytes differ from subcutaneous adipocytes in that they release secreted proteins that are known or potential risk factors for CHD. In at least one study, visceral fat expressed and released more plasminogen activator inhibitor-1, a fibrinolysis inhibitor, than subcutaneous fat [26]. Angiotensinogen is a potential blood pressure regulator and is also highly expressed in visceral adipose tissue.

There are currently multiple methods to assess body fat distribution. The most accurate method is costly and time-consuming. It is not suitable for large-scale population research. Since routine access to CT, magnetic resonance imaging (MRI) may too expensive to be feasible for many clinicians, and the use of these methods to image visceral and ectopic fat has historically been reserved for research purposes,

Perhaps the most widely used and these measurements are taken for waist circumference. Ashwell and colleagues were the first to show that there is a correlation between visceral fat mass and waist-to-hip ratio. However, compared with waist-to-hip ratio, waist circumference has a stronger correlation with visceral fat mass [27].

4.4 Different cutoff values for waist circumference

The result of A study which included 22,882 patients after a first myocardial infarction showed that larger WC ($WC \geq 106.3$ cm) was associated with recurrent atherosclerotic cardiovascular disease [28]. A recent study of patients with type-2 diabetes mellitus showed that compared with those in the first quartile of WC, male patients in the fourth quartile of WC ($WC \geq 126$) had a HR of 1.24 (95% CI 1.05–1.46) for MACEs; female patients in the fourth quartile of WC ($WC \geq 122$ cm) had an HR of 1.22 (95% CI 0.96–1.56) for MACEs [29]. The NCEP study showed that, there was a significant trend for a higher risk of CVD mortality across a higher WC categories ($WC \geq 102$ cm) [30]. In a study, WC values >88 cm for women and >102 cm for men were considered high and a high WC was a predictor of mortality in peritoneal dialysis patients [31]. The result of A meta-analysis of 29 cohorts involving more than 58 000 elderly persons during a 5-year-follow-up showed that a large waist (≥ 102 cm, men; ≥ 88 cm, women) was consistently associated with all-cause and CVD mortality [32]. Our result demonstrated that a larger WC ($WC \geq 94$ cm) was a significant and independent predictor of cardiovascular death in general population. The association is still significant after adjustment of other clinical confounders. Our results provided updated information about the long-term prognostic role of WC in general population.

Limitations of our study

The limitation of our study is the relatively small cohort patients; therefore, a larger sample of research is needed. Another limitation would be the lack of "gold standard" methods for abdominal obesity, such as computed tomography (CT) or MRI, may be another limitation of current research. However, the effectiveness of WC has previously been confirmed in cross-sectional studies and prospective studies.

Conclusion

In conclusion, our study demonstrated that an increased WC ($WC \geq 94$ cm) was associated with an increased cardiovascular death in general population. WC may provide incremental prognostic value beyond traditional risks factors.

List Of Abbreviations

AUC	area under the curve
BF	body fat
BMI	body mass index
BP	blood pressure
CI	confidence intervals
CT	computed tomography
CV	cardiovascular
CVD	cardiovascular disease
CHD	coronary heart disease
DBP	diastolic blood pressure
DM	diabetes mellitus
HDL-C	high-density lipoprotein cholesterol
HR	hazard ratio
ICD	international classification of diseases
IDI	integrated discrimination improvement
LDL-C	low-density lipoprotein cholesterol
MACEs	major adverse cardiovascular events
MRI	magnetic resonance imaging
NRI	net reclassification index
ROC	receiver operating characteristic
SAT	subcutaneous adipose tissue
SBP	systolic blood pressure
SD	standard deviation
TC	total cholesterol
TG	triglyceride
VAT	visceral adipose tissue
WC	waist circumference

Declarations

Ethics approval and consent to participate

This study was approved by the Ethics Board of the Chinese PLA General Hospital.

Consent for publication

Written informed consent for publication was obtained from each author and each patient.

Availability of data and material

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests

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Authors' contributions

Ping Zhu made substantial contributions to the conception or design of the work. Man Li and Shu-xia Wang contributed equally to the work, they contributed to the data collection, data interpretation, and critical review and drafting of the manuscript. Yong-kang Su, Jin Sun, An-hang Zhang, Bo-kai Cheng, Shuang Cai contributed to the data collection. All of the authors read and approved the final manuscript.

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Figures

Figure 1

The flowchart of the study

Figure 2

Kaplan–Meier survival rate of the participants according to the different WC levels

Figure 3

Cox-adjusted event-free survival curves for freedom of cardiovascular death in the 3 groups after adjustment for potential clinical confounders

Figure 4

Comparison of different predictive models for predicting cardiovascular death. Model 1 include clinical risk factors only. Model 2 include Model 1 and WC.

Figure 5

Associations between WC and the prevalence of cardiovascular death.