BMI (35-39.9 kg/m²) is associated with a significantly lower risk of cardiovascular morbidity compared with a normal BMI

Mengjie Hong
The Seventh Affiliated Hospital of Sun Yat-sen University

Yafei Chang
Sun Yat-sen University

Peipei Jiang
The Fourth People's Hospital of Urumqi City

Ling Sun
Fuyang Tumor hospital

Yitong Ma
First Affiliated Hospital of Xinjiang Medical University

Xiang Ma
First Affiliated Hospital of Xinjiang Medical University

Qinghua Yuan (✉ yuanqinghua2013@sina.com )
The Seventh Affiliated Hospital of Sun Yat-sen University

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Abstract

Objective

Obesity has been considered as a major risk factor for coronary heart disease (CHD), but the association between CHD and the degree of obesity is unclear. The objective of this study is to explore these relationships.

Methods

A total of 8299 patients from Xinjiang, China were enrolled in this study. BMI was categorized as normal (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), obesity class I (30.0–34.9 kg/m²), class II (35.0–39.9 kg/m²) and class III (≥ 40 kg/m²). We used logistic regression models to investigate differences in CHD risk between BMI categories and performed subgroup analysis in post-menopausal women.

Results

In this study, 62.1% of patients were overweight or obese, and only 37.9% had normal BMI. Logistic regression analysis showed that age [OR = 1.04, 95%CI (1.04–1.05), P < 0.01], and diabetes Yes vs. No [OR = 2.27, 95%CI (1.88–2.74), P < 0.01] might be independent predictors of CHD. Alcohol consumption Yes vs. No [OR = 0.70, 95%CI (0.56–0.88), P < 0.01] may decrease the risk of CHD. Lower risk was found in the obesity class II (BMI 35.0–39.9 kg/m²) group [OR = 0.62, 95%CI (0.41–0.93), P = 0.02] compare to people with normal weight. Postmenopausal women with a BMI of 35.0–39.9 kg/m² also had a lower risk for CHD compared to women with normal weight.

Conclusion

We found that individuals with lower BMI might not necessarily have a lower risk for CHD, obese individuals with BMI between 35-39.9 kg/m² might have a lower CHD risk compared to individuals who are of normal weight. Obesity evaluation and CHD risk prediction should require a more comprehensive assessment.

1. Introduction

The global obesity epidemic has been confirmed, according to the Global Burden of Disease (GBD) Obesity Collaborators [1], obesity affected 603.7 million (588.2- 619.8) adults worldwide in 2015, obesity prevalence has doubled since 1980 in more than 70 countries and continuously increased in most other countries. As stated by the WHO, 39% of the world’s adult population was overweight, and 13% was obese in 2016[2].

Coronary heart disease (CHD) is a complicated disease with a poor prognosis, which imposes a significant burden on global healthcare. It accounts for the greatest proportion of cardiovascular disease (CVD), which remains a dominant cause of death throughout the world [3, 4]. The incidence of CHD is also increasing in China. A previous study conducted in China revealed that vascular diseases had become one of the leading causes of death among Chinese adults [5]. It is crucial to timely assess the potential risks of CHD.

Obesity has been considered a major risk factor for most cardiovascular diseases, including coronary heart disease, hypertension, heart failure, and so on [6, 7, 8]. The associations between overweight and obesity with higher all-cause mortality were broadly consistent [9]. Higher BMI was shown to be strongly associated with cardiovascular diseases and mortality in a large body of previous studies [10, 11, 12, 13]. However, in recent years, studies have found contrary results,
they found the association of obesity between the risk of coronary heart disease and survival in coronary heart disease patients remains controversial, which has been described as the “Obesity Paradox”. Obesity degree is usually measured by body mass index (BMI), which is calculated as weight in kilograms divided by height in meters squared (kg/m²) [2]. In 1996, Ellis et al [14] claimed that low-normal or high BMI was a newly described and powerful risk factor for in-hospital death after percutaneous coronary intervention (PCI). Since then, numerous studies [15, 16, 17] showed this “overweight paradox” phenomenon, many types of cardiovascular diseases may have better prognosis among overweight or obese populations. A cross-sectional study of 491,773 individuals in the USA [18] indicated that being underweight might be another risk factor for cardiovascular disease. A study by Wang C et al performed among people with diabetes and hypertension implied that BMI and coronary heart disease risk showed a smile curve relationship [19]. A similar result was also found among cancer patients [20]. The association between BMI and cardiovascular outcomes is complex and contentious.

Despite these studies, the relationship between BMI and CHD is still not fully understood. Most of the previous studies focused on the association between BMI and CHD mortality, but only a limited number of studies explored the relationship between BMI and the risk of incident CHD. Using a large population-based cohort in China, our study aimed to examine the relationship between BMI and the risk of CHD in patients with coronary heart disease. Considering the appearance of controversy may due to a variety of BMI classification methods, in this study, we chose the World Health Organization criteria [2] to define overweight as a BMI ≥ 25 and < 30 kg/m² and obesity as a BMI ≥ 30 kg/m².

2. Method

2.1 Study design and population

This study was cross-sectional, and the population included patients hospitalized in the First Affiliated Hospital of Xinjiang Medical University. This study was approved by the Ethics Committee of the First Affiliated Hospital of Xinjiang Medical University and conducted according to the standards of the Declaration of Helsinki. Informed consent was obtained from all the participants present in the study.

The inclusion criteria:

1. Patients met the diagnostic criteria for CHD confirmed by coronary angiography or coronary computed tomography (at least one major coronary artery stenosis ≥ 50%).
2. Patients older than 18 years and voluntarily agreed to participate.

The exclusion criteria:

1. Missing BMI information.
2. Severe cognitive impairment.

2.2 Data collection

Ethnicity was categorized into three groups: Han, Uygur, and Kazakh. Socioeconomic (age, sex, and ethnicity), medical history (hypertension, diabetes mellitus, atherosclerosis, smoking, and alcohol consumption), health status (weight, height, abdominal circumference, blood pressure, heart rate, fasting glucose, cholesterol, creatinine, and uric acid) information were collected. Weight, height, abdominal circumference, blood pressure, and heart rate were measured by trained nurses using uniform standards. All blood samples were analyzed by the accredited laboratory of the First Affiliated Hospital of Xinjiang Medical University.

2.3 Definition of Variables
CHD was defined as the documented diagnosis of coronary heart disease confirmed by invasive or non-invasive methods (≥ 50% vascular stenosis), including angina and myocardial infarction, a history of percutaneous coronary intervention (PCI), or bypass surgery.

Our study recruited patients mainly from three ethnic groups, including Han, Uygur, and Kazakh. Scientists identified four major ancestral components in Xinjiang's Uyghurs, including harboring European, South Asian, Siberian, and East Asian [21]. Given the unique genetic backgrounds of Uygur people, BMI was classified according to the WHO criteria: normal weight (BMI 18.5-24.9kg/m2), overweight (BMI 25-29.9kg/m2), obesity class I (BMI 30-34.9kg/m2), obesity class II (BMI 35.0–39.9 kg/m2), obesity class III (BMI ≥ 40 kg/m2) [22].

The menopausal status of women was collected by querying themselves and then they were categorized as postmenopausal or premenopausal status. Menopause was defined as the absence of menstruation for more than consecutive 12 months before the interview.

### 2.5 Statistical Analysis

Data analysis was performed using SPSS software for Windows, version 26.0. Variables with continuous characteristics were described by means and standard deviations (SDs), and discontinuous variables were presented as proportions. Associations between BMI and the risk of CHD were evaluated using the logistic regression models and expressed with odds ratios (ORs). To investigate the relationship between body mass index and CHD, we conducted three logistic regression models: model 1 adjusted for BMI; model 2 adjusted for BMI, age, and ethnicity (Han, Uygur, Kazakh); model 3 adjusted for BMI, age, ethnicity, abdominal circumference, total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and fasting blood glucose. To examine the relationship between BMI and CHD, we used patients with BMI 18.5–24.9 kg/m2 as the reference, which was taken as controls (OR = 1).

### 3. Results

#### 3.1 Demographic characteristics and clinical features

A total of 8299 individuals (male 69.7%) were enrolled in this study. Patients were divided into five groups according to the BMI cutoff value. A summary of demographic and clinical characteristics is listed in Table 1. Table 1 shows most of the patients were overweight or obese (62.1%) and only 37.9% had normal BMI. The highest proportion of subjects in the study was overweight. The normal weight group had older mean ages than the overweight and obese BMI groups. The HDL, LDL, fast glucose, total cholesterol, triglyceride, and diabetes were not significantly different between the 5 groups.

#### Table 1 Baseline characteristics of the patients according to BMI
<table>
<thead>
<tr>
<th>Items</th>
<th>(18.5-24.9)</th>
<th>(25-29.9)</th>
<th>30-34.9</th>
<th>35-39.9</th>
<th>≥40</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>59.65±11.20</td>
<td>57.37±11.10</td>
<td>55.43±10.61</td>
<td>53.55±10.0</td>
<td>54.00±10.85</td>
<td>0.01</td>
</tr>
<tr>
<td>Sex(male)</td>
<td>1944(23.4)</td>
<td>2927(35.3)</td>
<td>785(9.5)</td>
<td>111(1.3)</td>
<td>14(0.2)</td>
<td>0.01</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Han</td>
<td>2562 30.9</td>
<td>2838 34.2</td>
<td>525 6.3</td>
<td>52 0.6</td>
<td>4 0</td>
<td>0.01</td>
</tr>
<tr>
<td>Uygur</td>
<td>483 5.8</td>
<td>922 11.1</td>
<td>390 4.7</td>
<td>72 0.9</td>
<td>7 0.1</td>
<td>0.01</td>
</tr>
<tr>
<td>Kazakh</td>
<td>98 1.2</td>
<td>204 2.5</td>
<td>112 1.3</td>
<td>19 0.2</td>
<td>8 0.1</td>
<td>0.01</td>
</tr>
<tr>
<td>Abdominal circumference</td>
<td>87.87±10.95</td>
<td>96.16±11.27</td>
<td>105.78±14.53</td>
<td>113.14±16.54</td>
<td>107.50±18.76</td>
<td>0.01</td>
</tr>
<tr>
<td>HR</td>
<td>73.31±10.86</td>
<td>74.32±10.73</td>
<td>76.37±10.64</td>
<td>77.11±10.77</td>
<td>76.89±13.32</td>
<td>0.01</td>
</tr>
<tr>
<td>Systolic BP, mmHg</td>
<td>137.83±28.39</td>
<td>142.59±27.97</td>
<td>147.59±28.27</td>
<td>154.68±26.37</td>
<td>163.93±33.41</td>
<td>0.01</td>
</tr>
<tr>
<td>Diastolic BP, mmHg</td>
<td>85.25±18.51</td>
<td>89.34±19.67</td>
<td>93.63±21.14</td>
<td>98.35±19.51</td>
<td>114.21±32.05</td>
<td>0.01</td>
</tr>
<tr>
<td>Creatinine</td>
<td>77.77±42.28</td>
<td>81.47±47.59</td>
<td>78.90±33.25</td>
<td>86.04±65.57</td>
<td>76.11±28.64</td>
<td>0.01</td>
</tr>
<tr>
<td>SUA, umol/L</td>
<td>286.95±110.77</td>
<td>304.85±131.46</td>
<td>321.92±120.01</td>
<td>327.92±131.28</td>
<td>313.89±136.41</td>
<td>0.01</td>
</tr>
<tr>
<td>HDL, mmol/L</td>
<td>6.67±24.43</td>
<td>8.47±28.18</td>
<td>6.58±24.26</td>
<td>6.88±24.58</td>
<td>7.45±23.98</td>
<td>0.11</td>
</tr>
<tr>
<td>LDL-C, mmol/l</td>
<td>3.19±15.29</td>
<td>2.84±8.53</td>
<td>2.81±9.34</td>
<td>2.53±0.95</td>
<td>2.69±1.19</td>
<td>0.87</td>
</tr>
<tr>
<td>Fast glucose, mmol/l</td>
<td>6.94±17.67</td>
<td>7.55±20.97</td>
<td>6.83±13.80</td>
<td>6.55±2.98</td>
<td>6.00±1.51</td>
<td>0.77</td>
</tr>
<tr>
<td>Total Cholesterol, mmol/L</td>
<td>4.72±5.86</td>
<td>5.02±9.48</td>
<td>5.12±11.71</td>
<td>4.69±1.95</td>
<td>6.50±6.81</td>
<td>0.64</td>
</tr>
<tr>
<td>Triglyceride, mmol/L</td>
<td>4.08±17.64</td>
<td>4.58±17.46</td>
<td>4.03±9.89</td>
<td>3.92±6.53</td>
<td>5.60±10.50</td>
<td>0.85</td>
</tr>
<tr>
<td>Atherosclerosis (%)</td>
<td>1143(13.8)</td>
<td>1341(16.2)</td>
<td>369(4.4)</td>
<td>62(0.7)</td>
<td>11(0.1)</td>
<td>0.01</td>
</tr>
<tr>
<td>Hypertension</td>
<td>2044(24.6)</td>
<td>2309(27.8)</td>
<td>541(6.5)</td>
<td>62(0.7)</td>
<td>6(0.1)</td>
<td>0.01</td>
</tr>
<tr>
<td>Diabetes</td>
<td>426(5.1)</td>
<td>621(7.5)</td>
<td>154(1.9)</td>
<td>22(0.3)</td>
<td>1(0)</td>
<td>0.16</td>
</tr>
<tr>
<td>Smoking</td>
<td>574 6.9</td>
<td>894 10.8</td>
<td>281 3.4</td>
<td>44 0.5</td>
<td>8 0.1</td>
<td>0.01</td>
</tr>
<tr>
<td>Alcohol drinker</td>
<td>365 4.4</td>
<td>631 7.6</td>
<td>209 2.5</td>
<td>34 0.4</td>
<td>5 0.1</td>
<td>0.01</td>
</tr>
</tbody>
</table>

### 3.2 Association of cardiovascular disease risk factors with coronary heart disease

Table 2 shows logistic regression between some risk factors and risk for coronary heart disease. Logistic regression results revealed that age [OR = 1.04, 95%CI (1.04-1.05), P<0.01], and diabetes Yes vs. No [OR = 2.27, 95%CI (1.88-2.74), P<0.01] may be independent predictors of CHD. Alcohol consumption Yes vs. No [OR = 0.70, 95%CI (0.56-0.88), P<0.01] may decrease the risk of CHD. Lower risk was found in the obesity class II (BMI 35-39.9kg/m2) group [OR = 0.62, 95%CI (0.41-0.93), P = 0.02] compared to people with normal weight (BMI 18.5-24.9 kg/m2).
Table 2 Associations between CHD and risk factors

<table>
<thead>
<tr>
<th>Variables</th>
<th>CHD</th>
<th>OR (95%CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td></td>
<td>1.04</td>
<td>1.04-1.05</td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td>1.13</td>
<td>0.98-1.31</td>
</tr>
<tr>
<td>SBP</td>
<td></td>
<td>1.00</td>
<td>0.99-1.00</td>
</tr>
<tr>
<td>DBP</td>
<td></td>
<td>1.00</td>
<td>1.00-1.01</td>
</tr>
<tr>
<td>Diabetes, Yes vs. No</td>
<td></td>
<td>2.27</td>
<td>1.88-2.74</td>
</tr>
<tr>
<td>BMI (kg/m^2)</td>
<td></td>
<td>0.98</td>
<td>0.96-0.99</td>
</tr>
<tr>
<td>Q2 vs. Q1</td>
<td></td>
<td>1.00</td>
<td>0.88-1.13</td>
</tr>
<tr>
<td>Q3 vs. Q1</td>
<td></td>
<td>1.03</td>
<td>0.86-1.25</td>
</tr>
<tr>
<td>Q4 vs. Q1</td>
<td></td>
<td>0.62</td>
<td>0.41-0.93</td>
</tr>
<tr>
<td>Q5 vs. Q1</td>
<td></td>
<td>0.54</td>
<td>0.17-1.66</td>
</tr>
<tr>
<td>Current smoking, Yes vs. No</td>
<td></td>
<td>1.17</td>
<td>0.95-1.44</td>
</tr>
<tr>
<td>Current drinking, Yes vs. No</td>
<td></td>
<td>0.70</td>
<td>0.56-0.88</td>
</tr>
</tbody>
</table>

Table 3 presents adjusted odd ratios (OR) for associations between CHD and BMI levels. Relative to individuals with normal weight, overweight individuals might have an increased risk of CHD (OR=1.12, P=0.03) in model 1. Adjustment for BMI, age and the ethnic group showed similar results (OR=1.22, P<0.01). After the adjustment for the potentially confounding factors of abdominal circumference, total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and fasting blood glucose, we found obese class II individuals had a lower risk of CHD [OR =0.62, 95%CI (0.41-0.93), P =0.02].

Table 3 Adjusted odds ratios (95% CI) for associations between CHD and BMI levels

<table>
<thead>
<tr>
<th>Model</th>
<th>Q1</th>
<th>P</th>
<th>Q2</th>
<th>P</th>
<th>Q3</th>
<th>P</th>
<th>Q4</th>
<th>P</th>
<th>Q5</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>1</td>
<td>-</td>
<td>1.12</td>
<td>0.03</td>
<td>1.02</td>
<td>0.80</td>
<td>0.75</td>
<td>0.09</td>
<td>0.42</td>
<td>0.06</td>
</tr>
<tr>
<td>2</td>
<td>1</td>
<td>-</td>
<td>1.22</td>
<td>0.01</td>
<td>1.16</td>
<td>0.06</td>
<td>0.89</td>
<td>0.50</td>
<td>0.18</td>
<td>0.10</td>
</tr>
<tr>
<td>3</td>
<td>1</td>
<td>-</td>
<td>1.00</td>
<td>1.00</td>
<td>1.03</td>
<td>0.73</td>
<td>0.62</td>
<td>0.02</td>
<td>0.54</td>
<td>0.28</td>
</tr>
</tbody>
</table>

Model 1 Adjusted for BMI, Model 2 adjusted for model 1 + age, and ethnic group, Model 3 adjusted for model 2 + other factors abdominal circumference, total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and fasting blood glucose.

3.3 CHD risk in post-menopausal women

We performed subgroup analysis in post-menopausal women and found similar results. Table 4 presents adjusted odd ratios (OR) for associations between CHD with post-menopausal women and BMI levels. Postmenopausal women with
BMI 35-39.9 kg/m² had a lower risk for CHD compared to women with normal weight in model 2 (OR = 0.43, P = 0.047) and model 3 (OR = 0.33, P = 0.03).

Table 4 Adjusted odds ratios (95% CI) for associations between CHD with Post-menopausal women and BMI levels

<table>
<thead>
<tr>
<th>Model</th>
<th>Q1</th>
<th>P</th>
<th>Q2</th>
<th>P</th>
<th>Q3</th>
<th>P</th>
<th>Q4</th>
<th>P</th>
<th>Q5</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
<td>1</td>
<td>-</td>
<td>1.10</td>
<td>0.34</td>
<td>1.01</td>
<td>0.95</td>
<td>0.51</td>
<td>0.11</td>
<td>1.06</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>1</td>
<td>-</td>
<td>1.05</td>
<td>0.62</td>
<td>0.89</td>
<td>0.48</td>
<td>0.43</td>
<td>0.047</td>
<td>0.85</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>1</td>
<td>-</td>
<td>0.95</td>
<td>0.66</td>
<td>0.79</td>
<td>0.22</td>
<td>0.33</td>
<td>0.03</td>
<td>1.81</td>
</tr>
</tbody>
</table>

Model 1 Adjusted for BMI, Model 2 adjusted for model 1 + age, and ethnic group, Model 3 adjusted for model 2 + other factors abdominal circumference, total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and fasting blood glucose.

4. Discussion And Conclusion

This cross-sectional study included 8299 patients with CHD in Xinjiang, China, patients with different BMI levels were classified into 5 groups according to the WHO criteria. Our study found that obese individuals with BMI between 35-39.9 kg/m² might have a lower CHD risk than those of normal weight [OR = 0.62, 95% CI (0.41–0.93), P = 0.02]. In the subgroup analysis of post-menopausal women, we obtained a similar result, the risk of CHD was the lowest in the obesity class II group with OR 0.43 in model 2 and OR 0.33 in model 3. Furthermore, this study suggested that alcohol consumption might reduce the risk of CHD.

Some previous studies support our observations, they found that the risk of CHD increased in people with low body weight [18, 20]. A previous study indicated a U-shaped association between BMI and the risk of CHD among women with diabetes [23]. Meanwhile, opposite opinions also exist, a large number of studies showed that higher BMI might confer an increased risk of CHD [24–27].

The reason for this difference is worthy of study. Several possible mechanisms could account for lower risk in obese people. First of all, the limitation of BMI as a measurement of adiposity should be considered. BMI cannot distinguish lean versus adipose tissue compartments [28]. A higher BMI doesn't necessarily mean a lower fitness level. A previous study showed that CRF (cardiorespiratory fitness) is significant in the prevention of cardiovascular disease [29]. High-level CRF could largely offset the effect of obesity and other traditional risk factors [30], and some researchers described this phenomenon as the “Fat but Fit paradox” [31]. Secondly, it is possible that pathophysiological processes are different among people with different adiposity levels. A study in PCI patients identified an inverse correlation between BMI and index lesion calcification [32]. Parsa and Jahanshahi used SYNTAX and Duke scores to evaluate the severity of coronary artery disease (CAD) and reported a negative correlation between BMI and the severity of CAD [33]. Furthermore, CHD is often accompanied by other chronic diseases, such as hypertension, diabetes, and hyperlipidemia. These patients may consciously attempt to lose weight because of clinicians’ pieces of advice, which could influence our results. As a final point, as we know, cigarette smokers showed a significantly increased risk of developing CHD in comparison to nonsmokers. Cigarette smoking was found to have a significant association with being underweight [34]. Smoking cessation could result in a substantial increase in weight [35].

In the subgroup analysis of post-menopausal women, the risk of CHD was the lowest in the obesity class II group too. Previous studies showed estrogen increased with increasing BMI in post-menopausal women [36]. Obesity could
increase estrogen concentrations. Estrogen might reduce the risk of coronary heart disease by influencing plasma lipoprotein levels [37].

Moreover, our study also suggested that alcohol consumption may reduce the risk of CHD. Some studies reported alcohol consumption in moderation was associated with a reduced risk of CHD risk [38, 39, 40]. Moreover, there was study suggested that moderate alcohol consumption might reduce the risk of CVD [41] and light drinking had a reduced risk of mortality from total cardiovascular disease [42] in women, while heavy drinking might increase these risks. Some studies have demonstrated that alcohol was associated with increased HDL-C and decreased TRG, which may impact CVD risk [43]. New research about flora revealed that drinking in moderation tended to have a positive impact on metabolic profiles and commensal flora, which may explain its benefits on cardiovascular health [44]. However, the amount of alcohol consumption was not evaluated in our study.

In this cross-sectional study, clinical data and lifestyle information were analyzed to investigate the relationship between BMI and CHD risk. We found that individuals with lower BMI might not necessarily have a lower risk for CHD. Obese individuals with BMI between 35-39.9 kg/m2 might have a lower CHD risk when compared to individuals of normal weight. Obesity evaluation and CHD risk prediction should require a more comprehensive assessment.

**Limitation**

First, BMI is widely used to measure the degree of obesity, but it has serious limitations when used as an indicator of the percent of body fat mass [45]. Second, we did not measure the levels of female hormones, which might associate with the risk of CHD. Third, we did not record the severity of alcohol use. There were no underweight individuals in our study, so we cannot study this type of population. At last, as a retrospective cohort study, our study has all inherent limitations for retrospective analyses.

**Declarations**

**Ethics approval and consent to participate**

The present study was approved by the Ethics Committee of the First Affiliated Hospital of Xinjiang Medical University and conducted according to the standards of the Declaration of Helsinki. Written informed consent was obtained from all participants. All patients underwent a physical examination and biochemical screening at baseline.

**Consent for publication**

Consent for publication is obtained from participants for manuscripts that include information or images that could lead to the identification of study participants (Not applicable).

** Availability of data and materials**

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

**Competing interests**

All authors have declared no conflicts of interest. All authors state to consent to publish.

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Authors' contributions:

Mengjie Hong and Qinghua Yuan contributed to the study design and manuscript writing and revision. Yafei Chang, Peipei Jiang and Ling Sun contributed to the data collection statistical analysis, and experiment implementation. Xiang Ma and Yitong Ma contributed to the study design, and manuscript revision and approval.

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