

Obstructive Sleep Apnea Related to Abdominal Adipose Tissue Distribution Assessed by Bioelectrical Impedance Analysis in Obese Patients

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Abstract

Background: The aim of this study was to identify the association between obstructive sleep apnea syndrome (OSAS) and abdominal fat distribution in obese individuals.

Methods: A total of 94 obese patients were enrolled in the study from January 2018 to July 2020. Demographic data were collected. OSAS was diagnosed based on the results of overnight polysomnography, and the abdominal fat distribution was measured by bioelectrical impedance analysis (BIA). Univariate and multivariate logistic regression analyses were used to investigate the association between OSAS and the distribution of abdominal fat.

Results: 1) The mean age (SD) of the patients included in this study was 32.44 (11.81) years and the majority were women (62.77%). The overall incidence rate of OSAS was 51.06%, and 24 (25.53%) patients had mild OSAS, 10 (10.64%) had moderate OSAS, and 14 (14.89%) had severe OSAS. 2) The incidence of OSAS among men was higher than in women (62.86% vs. 44.07%). The apnea hypopnea index (AHI) of men was significantly higher than that of women (5.50, interquartile range [IQR] 3.80-30.6 vs. 4.2, IQR 1.4-12 events/hour, $P=0.014$). Additionally, men had a significantly higher visceral fat area (180.29 ± 51.64 vs. 143.88 ± 53.42 cm², $P=0.002$). 3) Patients with OSAS had a significantly higher visceral fat area than patients without OSAS (178.28 ± 59.89 vs. 135.68 ± 40.58 cm², $P=0.013$). Multivariate analysis demonstrated that abdominal fat area and fasting plasma glucose were independent risk factors for OSAS (odds ratio, 1.016; 95% confidence interval, 1.005-1.026, $P=0.005$; odds ratio, 1.618; 95% confidence interval, 1.149-2.278, $P=0.006$).

Conclusions: In obese patients, the abdominal visceral adipose deposit was an independent risk factor for OSAS. Therefore, improving the distribution of abdominal fat may contribute to alleviating the severity of OSAS.

Introduction

Due to the development of the society and changes in the lifestyle, the prevalence of obesity has increased significantly among the global population [1]. Obesity as a disease has become a global public health problem and is associated with several other diseases, such as type 2 diabetes mellitus (T2DM), hypertension, nonalcoholic fatty liver disease, and obstructive sleep apnea syndrome (OSAS) [2]. With regard to OSAS, obesity is one of the most important risk factors, and its prevalence is increasing in parallel with the severity of obesity [3, 4]. In addition, OSAS also promotes weight gain [5]. Consequently, obesity and OSAS interact with each other.

OSAS is characterized by repetitive upper airway obstruction during sleep, recurrent oxygen desaturation, and frequent arousal from sleep [6]. The overall population prevalence ranges from 9% to 38% and is higher in men. It increases with age, and in some elderly groups, the incidence of OSAS was reported to be as high as 90% in men and 78% in women [7]. OSAS not only decreases the quality of life (QOL) of the patients, but also increases the societal burden [8]. Several studies have shown that OSAS is associated

with numerous adverse health outcomes such as hypertension, cognitive impairment, stroke, and Alzheimer's disease [9, 10]. A new meta-analysis has demonstrated that blood pressure control can benefit from the treatment of OSAS [11].

OSAS results from a combination of anatomic features that narrow the upper airway along with the permissive effect of insufficient neuromuscular compensation during sleep. With an increasing volume of the upper airway structures, the severity of OSAS also increases [12]. In addition, abdominal adipose deposits due to obesity may be a causative factor for OSAS because of decreased lung volume and traction on the pharynx [13]. Research on abdominal adipose deposits and OSAS is relatively limited; therefore, we designed this study to further illuminate this relationship. In this study, we mainly aimed to evaluate the incidence of OSAS in the obese population as well as to investigate the association between OSAS and abdominal fat distribution.

Methods

This was a prospective cross-sectional study that enrolled 94 obese patients from the Inpatient Department of Endocrinology, Shanghai Tenth People's Hospital between July 2018 and January 2020. The body mass index (BMI) of the enrolled cohort was ≥ 30 kg/m², and all patients underwent an examination for the abdominal fat distribution and overnight polysomnography. The exclusion criteria were as follows: (1) patients without results of the overnight polysomnography examination and (or) abdominal fat distribution; (2) patients who did not agree to participate in this study. The study was approved by the ethics committee of Shanghai Tenth People's Hospital. All participants included in the study provided written informed consent.

Data collection

In this study, we collected demographic data including age, sex, height, weight, and BMI. We also recorded the circumference of the neck, waist, and hip, which were measured with a tape. Every measurement was performed twice, and the average was chosen as the final result to reduce error. Additionally, venous blood samples were collected after overnight fasting for the measurement of fasting plasma glucose, 2 hours postprandial plasma glucose, glycated hemoglobin, serum total cholesterol, triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and free fatty acids (FFA). Systolic blood pressure and diastolic blood pressure data were collected and recorded. Before measuring the blood pressure, all patients were asked to rest for 10 minutes and to avoid smoking and drinking coffee or tea.

Measurement and diagnosis of OSAS

We used overnight polysomnography (SOMNOlab2, Weinmann, Germany) in our study to assess whether the patients had OSAS. The result of the overnight polysomnography was presented as an apnea hypopnea index (AHI), measuring the total number of apneas and hypopneas per hour of sleep. It is considered the gold standard for the diagnosis of OSAS and is widely used in clinical practice [14]. Based on the AHI values, OSAS was divided into three categories, including mild OSAS (AHI 5 to 15

events/hour), moderate OSAS (AHI 15 to 30 events/hour), and severe OSAS (AHI \geq 30 events/hour) [15]. One day before the polysomnography was performed, patients were instructed to stop taking sedative and hypnotic agents such as alcohol and caffeine.

Examination of the abdominal fat distribution

We utilized a fat measurement device (DHS-2000, Omron, Japan) to measure the abdominal fat distribution, which uses the theory of bioelectrical impedance analyses (BIA) to measure the area of every abdominal component. The cross-sectional image at the L3 level of the lumbar vertebra was selected to measure the abdominal fat distribution, and the results were exhibited in cm². Moreover, this measurement does not use radiation and can be repeated multiple times.

Statistical analyses

Normally distributed continuous data were presented as means \pm standard deviations (SD), non-normally distributed data were presented as medians (quartile, third quartile), and categorical variables were presented as numbers (percentages). Normally distributed continuous data were compared using the independent samples t-test. Non-normally distributed continuous data were compared using the Mann – Whitney U test. Pearson's χ^2 test or Fisher's exact test were used to examine the differences between categorical variables. Univariate and multivariate logistic regression analyses were used to evaluate the risk factors for OSAS. $P < 0.05$ was considered statistically significant. All statistical analyses were performed using SPSS version 22 (IBM Corp, Armonk, NY, USA).

Results

General clinical characteristics of the participants

Ninety-four obese patients were enrolled in this study with a mean (SD) age of 32.44 (11.81) years and mean (SD) BMI of 38.88(5.87) kg/m². The detailed baseline characteristics are shown in Table 1. The overall incidence of OSAS was 51.06%, including 24 cases of mild OSAS (25.53%), 10 of moderate OSAS (10.64%), and 14 of severe OSAS (14.89%). The majority of patients were women (62.77%), but the incidence of OSAS in women was lower than in men (44.07% vs. 62.86%, $P=0.078$). Furthermore, the AHI in men was significantly higher than in women (5.50, IQR 3.80-30.6 vs. 4.2, IQR 1.4-12 events/hour, $P=0.014$); the results are shown in Figure 1. In addition, patients with hypertension ($P=0.030$) or diabetes ($P=0.019$) also exhibited a significantly higher AHI. Neck circumference, waist circumference, and visceral fat area showed a positive association with the AHI (all $P < 0.05$). Compared with women, men had a significantly higher visceral fat area (180.29 \pm 51.64 vs. 143.88 \pm 53.42 cm², $P=0.002$), as illustrated in Figure 2.

Univariate analysis of factors associated with OSAS

Based on the AHI values, the cohort was divided into two groups: the non-OSAS (AHI <5 events/hour) and the OSAS group (AHI ≥5 events/hour). Compared with the non-OSAS group, patients with OSAS had a significantly higher age (33.00, IQR 27.25-37.00 vs. 28.00, IQR 23.00-34.75y, $P=0.007$). In the OSAS group, the visceral fat area was significantly higher than in the non-OSAS group (178.28 ± 59.89 vs. 135.68 ± 40.58 cm², $P=0.013$). However, no significant differences were found in the subcutaneous fat area and BMI. The fasting plasma glucose, 2 hours postprandial plasma glucose, and glycated hemoglobin were all significantly higher in the OSAS group than in the non-OSAS group (all $P < 0.05$). While the HDL of the OSAS group was significantly lower than that of the non-OSAS group (0.96 ± 0.21 vs. 1.06 ± 0.27 mmol/L, $P=0.008$), the waist circumference of the OSAS group was significantly higher (121.21 ± 12.69 vs. 113.08 ± 13.03 cm, $P=0.043$). Although the results did not reach statistical significance, the mean neck circumference and hip circumference of the OSAS group were higher compared to those of the non-OSAS group. Other detailed results are shown in Table 2.

Results of the multivariate analysis

According to the results of the univariate analysis, the factors with $P < 0.05$, including age, waist circumference, visceral fat area, HDL, fast plasma glucose, 2 hours postprandial plasma glucose, and glycated hemoglobin, were further analyzed in the multivariate analysis. The results are shown in Table 3. Visceral fat area (odds ratio, 1.016; 95% confidence interval, 1.005-1.026; $P=0.005$) and fasting plasma glucose (odds ratio, 1.618; 95% confidence interval, 1.149-2.278; $P=0.006$) were found to be the independent risk factors for OSAS.

Discussion

The findings of our study revealed that the prevalence of OSAS was higher in obese patients and was associated with the deposition of abdominal visceral adipose tissue. Furthermore, abdominal visceral adipose accumulation was an independent risk factor for OSAS.

The incidence of obesity is increasing year by year and has become a public health issue. Obesity is strongly linked with many metabolic diseases such as type 2 diabetes, hypertension, cardiovascular diseases, dyslipidemia, non-alcoholic fatty liver disease, chronic kidney disease, obstructive sleep apnea, and hypoventilation syndrome. According to the results of our study, over half of the obese patients had OSAS and male obese patients demonstrated a higher incidence of OSAS. Therefore, the management of weight can play a crucial role and weight loss has been shown to be an effective treatment for OSAS [16]. In a study by Del Genio et al., OSAS patients were followed up for 5 years and it was reported that sleeve gastrectomy could improve OSAS [17].

BMI is the basis for the World Health Organization classification of obesity and has been used to assess the degree of obesity. However, due to individual differences, people may have the same BMI but a different distribution of fat and muscle tissue. Hence, BMI alone cannot accurately reflect the distribution of body fat in obese patients in the clinic [18]. In this study, we analyzed the association between BMI and

OSAS. The results were not statistically significant, possibly because the enrolled patients were Asians, who more commonly exhibit central obesity with a normal BMI [19]. Central obesity may also be one of the reasons why waist circumference was significantly associated with OSAS, but hip and neck circumference were not. In contrast to some studies that reported that neck circumference was associated with the incidence of OSAS [20], univariate analysis in our study did not show a significant relationship between neck circumference and OSAS. The main reason for this may be that we defined AHI ≥ 5 events/hour as the group standard whereas many other studies designated AHI < 15 events/hour as the cutoff. Additionally, when we further compared the different degrees of OSAS, we found that the neck circumference of severe OSAS patients was significantly higher than that of non-OSAS patients.

Furthermore, on measuring the abdominal adipose tissue distribution, we found that visceral adipose tissue was significantly associated with OSAS. Abdominal adiposity is known to be associated with decreased lung volumes and hypoventilation [20]. Increased abdominal visceral adiposity decreases the lung volumes, including the functional residual volume, which reduces traction on the pharynx, and this may subsequently result in increased pharyngeal collapsibility and, thus, OSAS [13]. In addition, abdominal fat distribution was assessed by body impedance analyses. This was the first study to evaluate the relationship between OSAS and abdominal fat distribution. The results of our study were similar to those of a study by C. D. Turnbull, who explored the relationship between fat distribution assessed by magnetic resonance imaging (MRI) and OSAS [12]. In his study, abdominal visceral fat at the L2-3 level was significantly associated with OSAS ($P=0.02$). Similarly, Kritikou et. al used computer tomography (CT) to assess visceral fat and proved that visceral adiposity was significantly associated with OSAS [21]. Hence, in the assessment of fat distribution, BIA can have the same effect as MR and CT. Compared with CT and MR, BIA is relatively cheap, portable, has no radiation, and consequently, may be more suitable for the screening of OSAS.

Several studies have shown that OSAS is significantly associated with T2DM, and a linear association has been found [22]. In our study, we also discovered that the levels of fasting plasma glucose and glycated hemoglobin were significantly higher in obese patients with OSAS than in patients without OSAS. Besides blood glucose, OSAS is also associated with other components of metabolic syndrome, such as blood lipids and blood pressure [23]. A previous study has revealed that serum triglyceride levels were significantly associated with OSAS [24]. However, our study did not present a similar result, which may be due to a difference in the cohort.

However, there are also some limitations of our study. One is that the sample size is comparatively small because the measurement of OSAS requires special equipment and conditions, and not everyone consent to the examination. The other one is that our study is a retrospective study, we do not investigate whether the improvement in obesity and the reduction of abdominal fat can lead to an improvement in OSAS. Larger scale and well-designed randomized controlled trials are necessary in the future.

Conclusion

The prevalence of OSAS was high in obese patients and was associated with the deposition of abdominal fat, especially visceral adipose tissue. Abdominal visceral adipose accumulation was an independent risk factor for OSAS. Obesity as a significant risk factor for OSAS was linked to a reduction in both sleep quality and quantity. In obese patients, especially those with visceral adipose deposits, attention should be paid to the presence of OSAS. Our study investigated the association between abdominal visceral adipose tissue and OSAS in obese patients, which has important clinical significance in the assessment and treatment of OSAS.

Abbreviations

T2DM: Type 2 diabetes mellitus; OSAS: Obstructive sleep apnea syndrome; QOL: The quality of life; BMI: Body mass index; TG: Triglycerides; HDL: high-density lipoprotein; LDL: low-density lipoprotein; FFA: free fatty acids; AHI: apnea hypopnea index; SD: standard deviations ()

Declarations

Acknowledgements

Not applicable.

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Availability of data and materials

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

Authors' Contributions

Conceptualization, LS L and S Q; Formal Analysis, BW M; Investigation, HH M and SL W; Data Curation, XC W; Writing-Original Draft Preparation, BW M and XC W; Writing – Review & Editing, DL Z; Supervision, L D and Y C; Funding Acquisition, S Q.

Competing interests

The authors declare that they have no competing interests.

Consent for publication

Not applicable.

Ethics approval and consent to participate

This study was approved by the ethics committee of Shanghai Tenth People's Hospital.

Endnotes

Not applicable.

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Tables

Table1 Patients demographic characteristics and correlation with AHI

	Total N=94	R	P
Age (y)	31.00(24.00-37.00)	0.268	0.001*
Sex			0.014*
Male	35(37.23%)		
Female	59(62.77%)		
Height (m)	1.68(0.09)	0.155	0.137
Weight (kg)	103.40(92.60-124.25)	0.197	0.056
BMI (kg/m ²)	38.00(34.59-41.92)	0.306	0.003*
Neck circumference (cm)	42.35(4.71)	0.380	0.001*
Waist circumference (cm)	117.44(13.41)	0.328	0.002*
Hip circumference (cm)	120.26(11.41)	0.189	0.086
OSAS			0.001*
None	46(48.94%)		
Mild	24(25.53%)		
Moderate	10(10.64%)		
Severe	14(14.89%)		
Visceral fat area (cm ²)	157.44(55.39)	0.420	0.001*
Subcutaneous fat area (cm ²)	411.99(115.46)	0.010	0.094
Diabetes			0.019*
Yes	30(31.91%)		
No	64(68.09%)		
Hypertension			0.030*
Yes	69(73.40%)		
No	25(26.60%)		
Triglyceride (mmol/L)	1.64(1.19-2.42)	0.135	0.200
Cholesterol (mmol/L)	4.66(0.91)	0.081	0.443
LDL-C (mmol/L)	2.84(0.77)	0.142	0.179
HDL-C (mmol/L)	1.01(0.24)	0.170	0.106

FFA (mmol/L)	0.66(0.23)	0.052	0.625
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OSAS, obstructive sleep apnea syndrome; AHI, apnea hypopnea index; BMI, body mass index; HDL-C, high-density lipoprotein; LDL-C, low-density lipoprotein; FFA, free fatty acids

*Statistically significant ($P < 0.05$).

Table2 Univariate analysis of factors for OSAS

	OSAS N=48	Non-OSAS N=46	<i>P</i>
Age (y)	33.00(27.25-37.00)	28(23.00-34.75)	0.007*
Sex			0.078
Male	22(62.86%)	13(37.14%)	
Female	26(44.07%)	33(55.93%)	
Height (m)	1.70(0.08)	1.66(0.10)	0.092
Weight (kg)	115.20(98.15-131.08)	99.95(89.70-118.00)	0.078
BMI (kg/m ²)	39.55(35.40-45.25)	37.18(33.98-40.37)	0.257
Neck circumference (cm)	43.72(4.66)	40.77(4.29)	0.132
Waist circumference (cm)	121.21(12.69)	113.08(13.03)	0.043*
Hip circumference (cm)	121.40(12.39)	118.93(10.18)	0.559
Systolic pressure (mmHg)	143.35(19.43)	135.98(21.79)	0.146
Diastolic pressure (mmHg)	86.19(12.85)	81.17(11.82)	0.116
Visceral fat area (cm ²)	178.28(59.89)	135.68(40.58)	0.013*
Subcutaneous fat area (cm ²)	417.56(106.48)	406.18(125.07)	0.970
Fasting plasma glucose (mmol/L)	6.05(5.13-8.18)	5.2(4.88-6.00)	0.007*
2 hours postprandial plasma glucose (mmol/L)	9.50(7.18-16.08)	7.75(6.50-9.50)	0.012*
Glycated hemoglobin (%)	6.89(5.70-8.00)	5.90(5.60-6.23)	0.001*
Triglyceride (mmol/L)	1.78(1.31-2.71)	1.45(1.14-2.38)	0.122
Cholesterol (mmol/L)	4.63(0.93)	4.69(0.90)	0.052
LDL-C	2.79(0.76)	2.90(0.79)	0.698
HDL-C	0.96(0.21)	1.06(0.27)	0.008*
FFA	0.67(0.22)	0.65(0.24)	0.855

OSAS, obstructive sleep apnea syndrome; AHI, apnea hypopnea index; BMI, body mass index; HDL-C, high-density lipoprotein; LDL-C, low-density lipoprotein; FFA, free fatty acids

*Statistically significant ($P < 0.05$).

Table3 Multivariate logistic regression analysis of factors for OSAS

	Odds ratio	95% CI	<i>P</i>
Visceral fat area	1.016	1.005-1.026	0.005*
Fast plasma glucose	1.618	1.149-2.278	0.006*

*Statistically significant (*P* < 0.05).

Figures

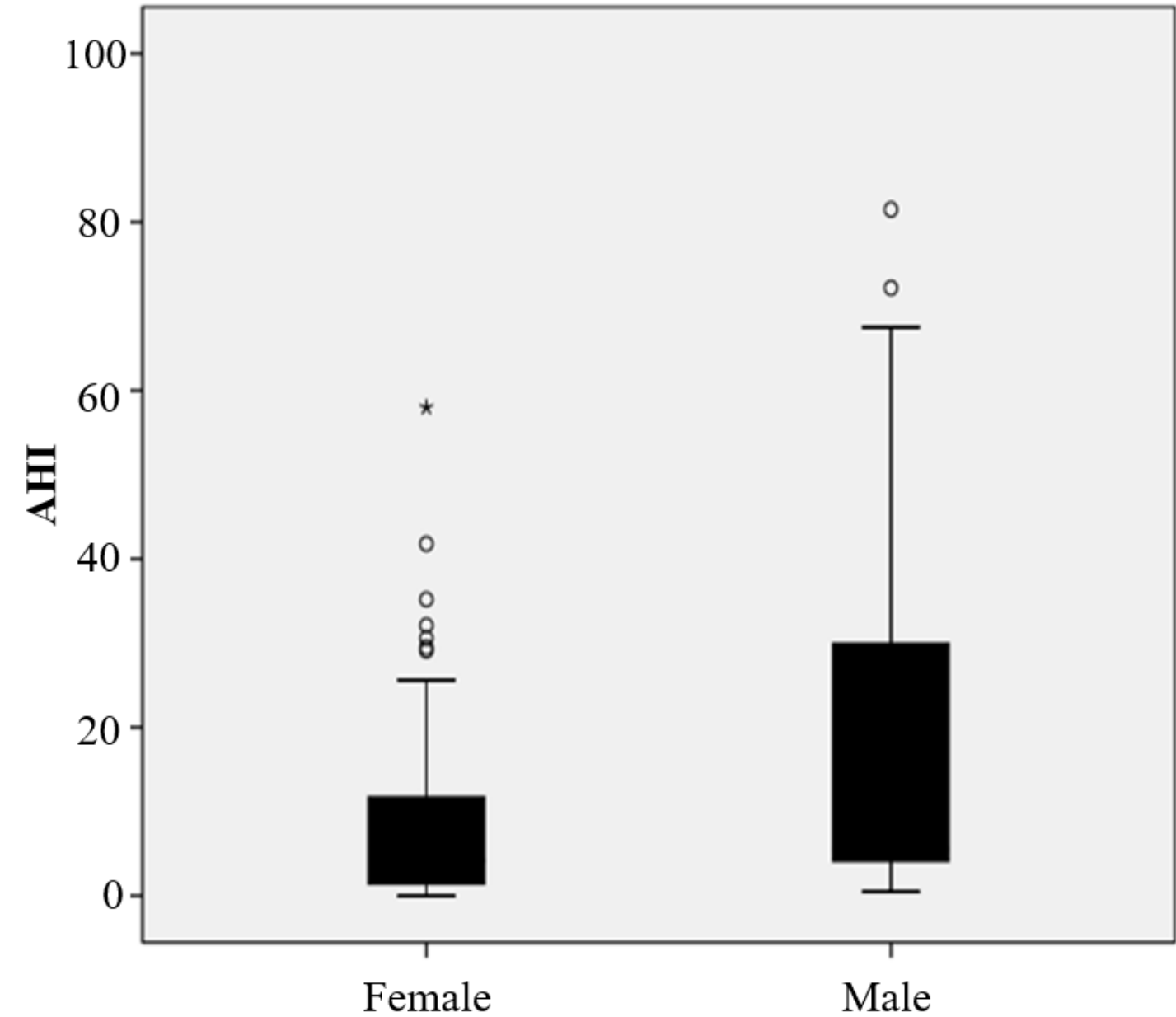


Figure 1

The value of AHI in female and male patients(P=0.014).

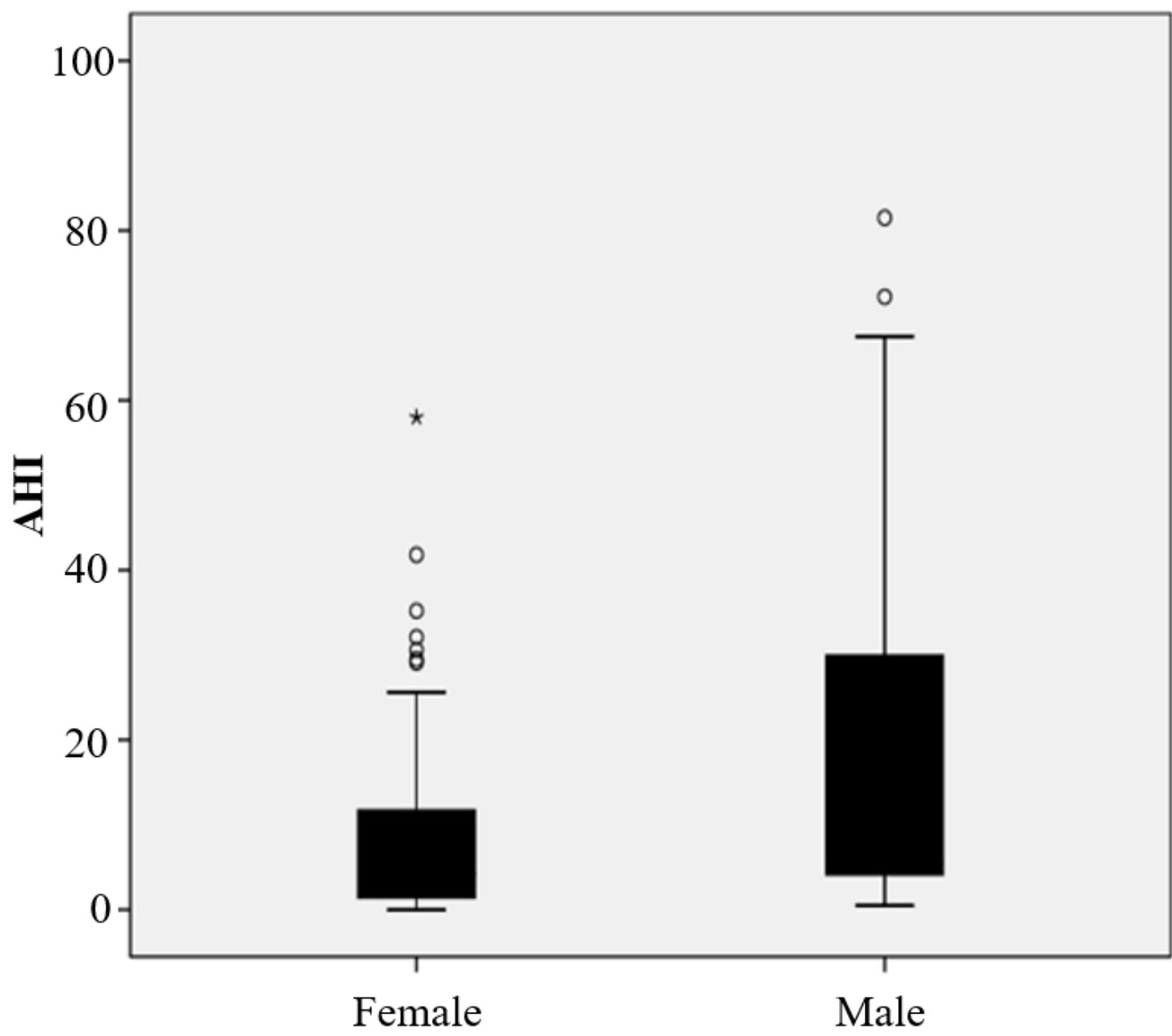


Figure 1

The value of AHI in female and male patients(P=0.014).

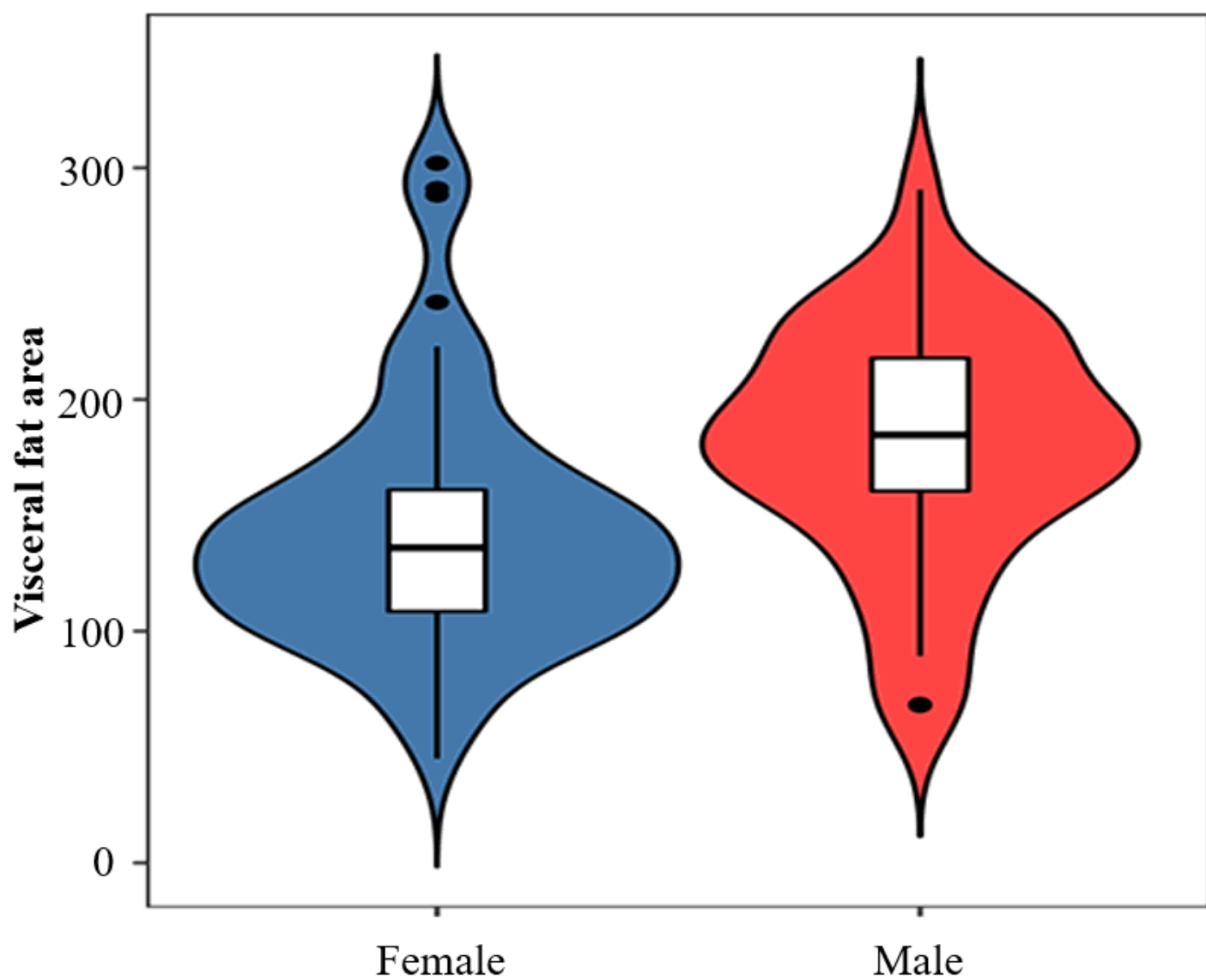


Figure 2

The visceral fat area in female and male patients($P=0.002$).

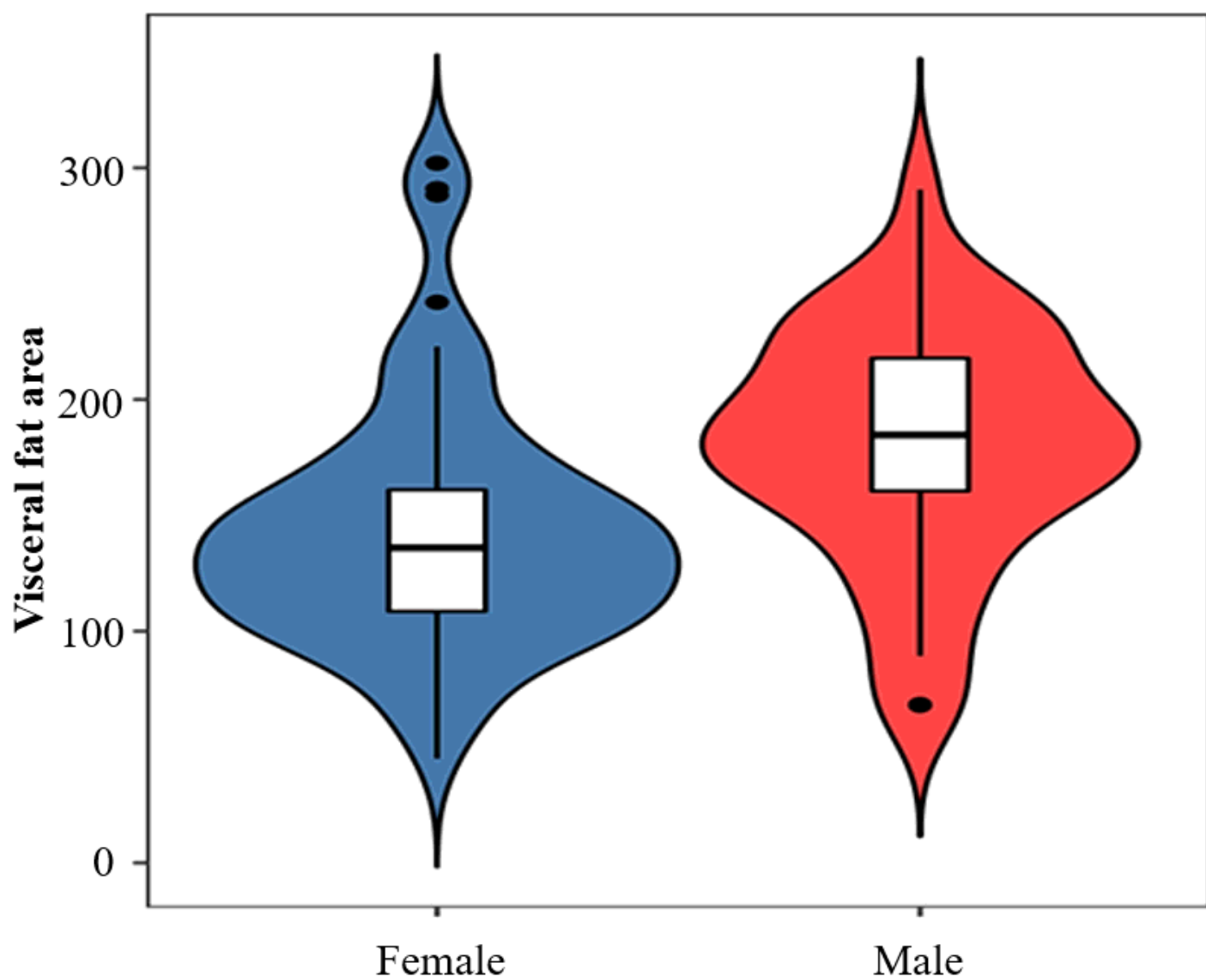


Figure 2

The visceral fat area in female and male patients($P=0.002$).