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Association of the Visceral Adiposity Index and Body Roundness Index with Prostate Cancer Risk: A Single-Center Case-Control Study

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Abstract

Background

Current research indicates that prostate cancer (PCa) is the second most common cancer worldwide, and a growing body of evidence suggests an association between visceral obesity and prostate cancer. Therefore, this study investigates the association between the Chinese Visceral Adiposity Index (CVAI) and the Body Roundness Index (BRI) and the risk of prostate cancer.

Methods

After propensity score matching, 200 prostate cancer patients and 200 healthy participants were included in the study. The association between CVAI and BRI and the risk of prostate cancer was assessed using logistic regression models, with odds ratios (OR) and 95% confidence intervals (CI) reported. The dose-response relationship between CVAI, BRI, and prostate cancer was evaluated using restricted cubic spline plots (RCS). Receiver Operating Characteristic curves (ROC) were used to evaluate the predictive value of the two indices for prostate cancer.

Results

In the multivariable logistic regression model, CVAI and BRI were positively correlated with the risk of prostate cancer ($P < 0.05$); the OR for the CVAI Q3 group was 3.47 (95% CI: 1.85–6.51), and the OR for the BRI Q4 group was 2.17 (95% CI: 1.20–3.94). Both CVAI and BRI showed a linear relationship with the risk of prostate cancer, with area under the ROC curve (AUC) values of 0.802 ($P < 0.05$) and 0.791 ($P < 0.05$), respectively.

Conclusion

There is a correlation between CVAI and BRI—as indices for assessing visceral obesity—and the risk of prostate cancer.

1 Introduction

Prostate cancer (PCa) is the second most common cancer worldwide and the fifth leading cause of cancer-related death among men[1]. According to the 2022 Global Cancer Statistics Report, there were 1.46 million new cases of prostate cancer and over 390,000 deaths[1]. Prostate cancer predominantly affects men over the age of 50[2]. With the global ageing population grows[3], the incidence of prostate cancer is increasing annually, posing a significant threat to the health of older men[4].Consequently, prostate cancer has become a major public health concern affecting men worldwide.

The causes of prostate cancer are complex[2]. The International Agency for Research on Cancer (IARC) has established a causal link between body fat and 13 types of cancer[5]. Obesity, as a pathological consequence of abnormal body fat distribution, has also been shown to be closely linked to cancer. According to research by Giorgio Gandaglia et al., apart from age and family history—which are recognised as non-modifiable risk factors for prostate cancer—obesity is now recognized as a modifiable risk factor[6]. Nevertheless, in recent years, studies on the association between obesity and prostate cancer have yielded negative results or found no association[7–13]. Furthermore, the findings of a Mendelian randomisation study do not provide strong evidence of a causal relationship between general obesity markers (BMI) and prostate cancer[14, 15]. The relationship between obesity and prostate cancer remains complex and controversial. Body Mass Index (BMI) is a commonly used measure of obesity; however, as it is calculated solely on the basis of height and weight, it cannot distinguish between total body weight and fat mass, or between subcutaneous adipose tissue (SAT) and visceral adipose tissue (VAT). Consequently, further investigation into the impact of obesity on prostate cancer requires the incorporation of visceral fat markers.

Common methods for assessing visceral fat include dual-energy X-ray absorptiometry (DEXA), computed tomography (CT), magnetic resonance imaging (MRI) and bioelectrical impedance analysis (BIA)[16, 17]. Due to radiation exposure, time constraints and high costs, these methods are not suitable for routine clinical practice in the general population[18, 19].

A number of indices have now been established to assess abdominal obesity, including the Visceral Adiposity Index (VAI) and the Body Roundness Index (BRI). There is a strong positive correlation between abdominal obesity and visceral fat area[19]. Studies have demonstrated that VAI and BRI are reliable indicators of visceral fat distribution[20–22]. To better estimate visceral fat in Chinese adults, Xia et al. incorporated age into the VAI formula. After validating their model against CT values, they established the Chinese Visceral Adiposity Index (CVAI) and confirmed its strong correlation with visceral fat[23]. BRI is a newer method for measuring obesity developed by Thomas et al[22]. It provides a more accurate estimate of body fat percentage and visceral fat percentage; using BRI as a measurement indicator is more sensitive in identifying individuals with visceral obesity[22]. At the same time, the BRI has been shown to be superior to other measurement indicators in predicting the risk of clinical conditions such as cardiometabolic diseases, kidney disease, and cancer[24–28]. However, CVAI and BRI remain relatively new areas of research in the field of oncology, although studies have already demonstrated a significant association between the risk of benign prostatic hyperplasia (BPH) and CVAI and BRI[29, 30]. However, much of the research is still in its exploratory phase, and no studies have yet reported on the relationship between CVAI and BRI and the risk of developing prostate cancer.

This study aims to investigate the relationship between central visceral adiposity (CVAI, BRI) and the risk of prostate cancer, thereby enhancing our understanding of the mechanisms underlying the development and progression of prostate cancer and providing a crucial basis for more accurate risk prediction, screening, intervention, prevention, and treatment of prostate cancer in the future.

2 Subjects and Methods

2.1 Subject of the study: We retrospectively reviewed data from 31,433 men who visited the Urology Department or Physical Examination Center of a hospital in Xinjiang between March 2019 and October 2024. Of these, 316 patients were diagnosed with prostate cancer via transrectal ultrasound-guided biopsy. Following 1:1 propensity score matching for age, 200 cases and 200 controls from a pool of 5,519 healthy examinees were included in the final analysis.

2.1.1 Inclusion and exclusion criteria for the case group: (1) Patients meeting the indications for prostate biopsy and diagnosed with prostate cancer via histopathological and immunohistochemical confirmation, aged ≥ 50 years, were included from March 2019 to October 2024; (2) Prostate cancer patients who were able to read, understand and provide a consent form, as well as a complete medical record; (3) Patients with a history of other types of cancer were excluded; (4) Patients with incomplete test data were excluded.

2.1.2 Inclusion and exclusion criteria for the control group:(1) Including males aged ≥ 50 years who underwent physical examination during the same period; (2) Participants who were able to read, understand and provide a written consent form; (3) Participants with incomplete examination data were excluded; (4) No evidence of current or past cancer.

This study has been approved by the Ethics Committee of the First Affiliated Hospital of Xinjiang Medical University (20180223-93). All study participants signed an informed consent form and fully understood the objectives of the study.

2.2 Data collection and measurement: This study is a single-center case-control study. General demographic data, such as the age and height of the participants, were obtained from medical records at hospitals and health check-up centers, while clinical data—including triglycerides, high-density lipoprotein, urea, uric acid, creatinine, and total cholesterol—were also collected.

Fasting blood samples were collected between 8:00 and 9:00 a.m. on the day after admission and were immediately sent for biochemical analysis. Patients meeting the criteria for prostate biopsy were scheduled for a transrectal prostate biopsy on the day following the examination. Biopsies were performed by several senior urologists using BARD biopsy needles (USA), employing the 12 + X puncture technique[31], wherein 1–2 needles were used as ‘X’ needles to target areas identified as suspicious on magnetic resonance imaging (MRI) or ultrasound, whilst the remaining areas were sampled using the standard 12-needle puncture method. Each tissue needle was placed individually into a fixation vial containing a 10% methanol-water solution. The vials were labelled with the patient’s admission number and the date of the biopsy, and were sent collectively to the Department of Pathology at the First Affiliated Hospital of Xinjiang Medical University for examination, where senior pathologists recorded and diagnosed the histopathological results.

2.2.1 Formula calculation

$$CVAI = -267.93+0.68\times \text{age}+0.03\times BMI+4.00\times WC+22.00\times log10(TG)-16.32\times HDL-C$$

$$BRI = 364.2-365.5\times \sqrt{1-\left(\frac{WC(m)/2\pi}{0.5\times BH(m)}\right)^2}$$

2.3 Statistical analysis

Continuous variables that followed a normal distribution were described as mean ± standard deviation, and t-tests were used for intergroup comparisons. Data that did not follow a normal distribution were described using the median and interquartile range, and nonparametric tests were used for intergroup analysis. Categorical data were described as percentages, and chi-square tests were used for between-group comparisons. Binary logistic regression was used to analyze the relationship between CVAI, BRI, and prostate cancer, and restricted cubic spline (RCS) plots were used to assess the dose-response relationship between CVAI, BRI, and prostate cancer. ROC curves were used to evaluate the predictive value of these two indices for prostate cancer. The significance level was set at α = 0.05. Propensity score matching was performed, with age as the matching variable, to pair the case group with the control group in a 1:1 ratio to reduce the influence of confounding factors. Subgroup analyses were conducted to compare differences in the effects of CVAI and BRI on prostate cancer between patients with and without hypertension; differences were considered statistically significant at $P < 0.05$.

3 Results

3.1 Analysis of baseline data

Prior to propensity score matching, the case group included 316 patients with prostate cancer, while the control group consisted of 5,519 participants who had undergone physical examinations. The median ages of the two groups were 54 and 68 years, respectively. Statistically significant differences in CVAI and BRI were observed between the groups ($P < 0.05$) (Table 1).

Table 1
Analysis of baseline data (pre-matching)

Variables	Total (n = 5835)	control group (n = 5519)	case group (n = 316)	Z/χ^2	*P*
Age at admission (years), M (Q_1, Q_3)	54.00 (52.00, 58.00)	54.00 (52.00, 57.00)	68.00 (62.00, 76.00)	-21.22	< 0.001
Urea (mmol/L), M (Q_1, Q_3)	5.39 (4.57, 6.39)	5.35 (4.53, 6.32)	6.12 (4.96, 7.20)	-7.64	< 0.001
Creatinine (μmol/L), M (Q_1, Q_3)	77.41 (68.28, 87.03)	77.62 (68.50, 87.08)	74.06 (62.36, 86.02)	-3.57	< 0.001
uric acid (mmol/L), M (Q_1, Q_3)	356.22 (307.00, 412.00)	358.00 (310.22, 413.66)	302.30 (255.00, 373.00)	-10.07	< 0.001
Triglycerides (mmol/L), M (Q_1, Q_3)	1.51 (1.07, 2.19)	1.51 (1.07, 2.21)	1.42 (1.07, 1.91)	-2.68	0.007
Total cholesterol (mmol/L), M (Q_1, Q_3)	5.14 (4.52, 5.82)	5.18 (4.58, 5.86)	4.21 (3.49, 4.80)	-16.41	< 0.001
High-density lipoprotein cholesterol (mmol/L), M (Q_1, Q_3)	1.14 (0.98, 1.33)	1.15 (0.98, 1.34)	1.04 (0.86, 1.21)	-7.54	< 0.001
BMI(kg/□), n(%)				12.46	0.006
Q1	1451 (24.87)	1359 (24.62)	92 (29.11)		
Q2	1454 (24.92)	1398 (25.33)	56 (17.72)		
Q3	1470 (25.19)	1376 (24.93)	94 (29.75)		
Q4	1460 (25.02)	1386 (25.11)	74 (23.42)		
CVAI n(%)				10.74	0.013
Q1	1459 (25.00)	1397 (25.31)	62 (19.62)		
Q2	1458 (24.99)	1390 (25.19)	68 (21.52)		
Q3	1459 (25.00)	1365 (24.73)	94 (29.75)		
Q4	1459 (25.00)	1367 (24.77)	92 (29.11)		
BRI n(%)				3.10	0.377
Q1	1459 (25.00)	1371 (24.84)	88 (27.85)		
Q2	1458 (24.99)	1390 (25.19)	68 (21.52)		
Q3	1455 (24.94)	1379 (24.99)	76 (24.05)		

Variables	Total (n = 5835)	control group (n = 5519)	case group (n = 316)	Z/χ^2	*P*
Q4	1463 (25.07)	1379 (24.99)	84 (26.58)		

Following propensity score matching, there were no differences in age between the groups. This study included a total of 400 participants, comprising 200 prostate cancer patients in the case group and 200 individuals in the control group. Baseline characteristics showed statistically significant differences ($P < 0.05$) between the two groups in BMI, BRI, CVAI, urea, uric acid, total cholesterol, and HDL-cholesterol. However, no statistically significant differences ($P > 0.05$) were observed in creatinine and triglyceride levels (Table 2).

Table 2
Analysis of baseline data (after matching)

Variables	**Total (n = 400)**	**control group (n = 200)**	**case group (n = 200)**	**Z/χ²**	***P***
Urea(mmol/L), M (Q_1, Q_3)	5.70 (4.68, 6.90)	5.33 (4.58, 6.42)	5.98 (4.79, 7.21)	-3.68	< 0.001
Creatinine(μmol/L), M (Q_1, Q_3)	76.34 (65.74, 87.00)	76.39 (67.00, 87.05)	76.00 (63.13, 86.02)	-1.20	0.229
uric acid(μmol/L), M (Q_1, Q_3)	333.69 (283.16, 396.92)	344.00 (297.00, 400.25)	321.08 (267.18, 384.38)	-3.17	0.002
triglycerides(mmol/L), M (Q_1, Q_3)	1.36 (1.02, 1.94)	1.25 (0.95, 1.91)	1.50 (1.07, 1.98)	-1.83	0.067
Total cholesterol(mmol/L), M (Q_1, Q_3)	4.68 (3.88, 5.32)	5.12 (4.46, 5.71)	4.25 (3.57, 4.80)	-7.92	< 0.001
High-density lipoprotein cholesterol(mmol/L), M (Q_1, Q_3)	1.10 (0.92, 1.30)	1.19 (0.99, 1.36)	1.04 (0.84, 1.20)	-5.29	< 0.001
BMI (kg/□), n(%)				14.00	0.003
Q1	100 (25.00)	57 (28.50)	43 (21.50)		
Q2	100 (25.00)	61 (30.50)	39 (19.50)		
Q3	100 (25.00)	38 (19.00)	62 (31.00)		
Q4	100 (25.00)	44 (22.00)	56 (28.00)		
CVAI, n(%)				14.67	0.002
Q1	99 (24.75)	63 (31.50)	36 (18.00)		
Q2	101 (25.25)	55 (27.50)	46 (23.00)		
Q3	99 (24.75)	40 (20.00)	59 (29.50)		
Q4	101 (25.25)	42 (21.00)	59 (29.50)		
BRI, n(%)				8.00	0.046
Q1	98 (24.50)	55 (27.50)	43 (21.50)		
Q2	102 (25.50)	45 (22.50)	57 (28.50)		
Q3	98 (24.50)	57 (28.50)	41 (20.50)		
Q4	102 (25.50)	43 (21.50)	59 (29.50)		

3.2 The relationship between visceral fat index and the risk of prostate cancer

To evaluate the association between visceral obesity indices and PCa risk, we constructed logistic regression models. In the quartile grouping analysis of CVAI, compared with Q1, Q3 and Q4 were significantly associated with an increased risk of prostate cancer in the multivariate analysis ($P < 0.05$), with odds ratios (OR) of 3.47 (95% CI: 1.85–6.51) and 3.33 (95% CI: 1.79–6.20), respectively. Similarly, in the quartile analysis of BRI, Q4 was positively correlated with prostate cancer compared to Q1 ($P < 0.05$), and this correlation persisted in the multivariate analysis ($P < 0.05$), with an OR of 2.17 (95% CI: 1.20–3.94) (Table 3).

Table 3
Relationship between visceral adiposity index and the risk of prostate cancer

Variables	Univariate		Multivariate	
	OR (95%CI)	*P*	OR (95%CI)	*P*
CVAI				
Q1	1.00		1.00	
Q2	1.46 (0.83 ~ 2.58)	0.188	1.78 (0.97 ~ 3.28)	0.063
Q3	2.58 (1.45 ~ 4.58)	0.001	3.47 (1.85 ~ 6.51)	< 0.001
Q4	2.46 (1.39 ~ 4.34)	0.002	3.33 (1.79 ~ 6.20)	< 0.001
BRI				
Q1	1.00		1.00	
Q2	1.62 (0.93 ~ 2.83)	0.090	1.68 (0.92 ~ 3.05)	0.088
Q3	0.92 (0.52 ~ 1.62)	0.773	1.20 (0.65 ~ 2.19)	0.559
Q4	1.76 (1.01 ~ 3.07)	0.049	2.17 (1.20 ~ 3.94)	0.011

3.3 RCS Curve Analysis of the Relationship Between Visceral Adiposity and Prostate Cancer Risk.

Analysis of the results from the restricted cubic spline analysis revealed that both CVAI and BRI exhibited a linear dose-response relationship with the risk of prostate cancer (overall P-value < 0.001; P-value for nonlinearity > 0.05) (Fig. 2).

3.4 ROC curve analysis evaluates the predictive value of the two indices for prostate cancer.

Analysis of the ROC curves revealed that the comparisons between the visceral adiposity indices (CVAI and BRI) in terms of the area under the curve (AUC) for prostate cancer were all statistically significant ($P < 0.05$). The AUC values for CVAI and BRI in the prostate cancer ROC curves were 0.802 ($P < 0.05$) and 0.791 ($P < 0.05$), respectively (Fig. 3).

3.5 Subgroup analysis

Based on the cut-off points for the area under the ROC curve for uric acid, urea, and cholesterol, subgroup analyses were conducted according to different levels. The results showed that the association between CVAI and BRI and prostate cancer was stronger in groups with lower levels of urea and uric acid. However, in the group with high cholesterol levels, the impact of CVAI and BRI on prostate cancer was more pronounced (Tables 4, 5 and 6).

Table 4
Effects of different levels of urea on prostate cancer

Variables	Univariate		Multivariate	
	OR (95%CI)	***P***	**OR (95%CI)**	***P***
Urea ≥ 5.7 mmol/L				
CVAI				
Q1	1.00		1.00	
Q2	2.12 (0.95 ~ 4.74)	0.068	2.75 (1.10 ~ 6.90)	0.031
Q3	1.74 (0.79 ~ 3.83)	0.166	1.88 (0.78 ~ 4.55)	0.161
Q4	2.25 (1.01 ~ 5.01)	0.047	2.12 (0.86 ~ 5.22)	0.103
BRI (Q4)				
Q1	1.00		1.00	
Q2	2.58 (1.11 ~ 6.03)	0.028	3.07 (1.20 ~ 7.90)	0.020
Q3	1.46 (0.67 ~ 3.21)	0.342	1.49 (0.62 ~ 3.57)	0.373
Q4	1.21 (0.55 ~ 2.65)	0.633	1.16 (0.48 ~ 2.82)	0.739
Urea<5.7 mmol/L				
CVAI				
Q1	1.00		1.00	
Q2	1.06 (0.44 ~ 2.54)	0.898	1.65 (0.56 ~ 4.87)	0.366
Q3	3.02 (1.32 ~ 6.93)	0.009	4.15 (1.42 ~ 12.14)	0.009
Q4	2.37 (1.04 ~ 5.44)	0.041	4.93 (1.62 ~ 15.08)	0.005
BRI				
Q1	1.00		1.00	
Q2	0.86 (0.37 ~ 1.98)	0.726	0.98 (0.35 ~ 2.70)	0.964
Q3	0.72 (0.31 ~ 1.66)	0.437	1.10 (0.38 ~ 3.18)	0.866
Q4	2.58 (1.15 ~ 5.81)	0.022	4.30 (1.43 ~ 12.94)	0.009

Table 5
The effect of different levels of uric acid on prostate cancer

Variables	Univariate		Multivariate	
	OR (95%CI)	***P***	**OR (95%CI)**	***P***
uric acid ≥ 333.69mmol/L				
CVAI				
Q1	1.00		1.00	
Q2	1.61 (0.70 ~ 3.69)	0.262	2.54 (0.99 ~ 6.51)	0.052
Q3	2.84 (1.25 ~ 6.44)	0.012	2.60 (1.05 ~ 6.41)	0.038
Q4	1.83 (0.80 ~ 4.18)	0.149	2.01 (0.80 ~ 5.01)	0.136
BRI				
Q1	1.00		1.00	
Q2	1.40 (0.62 ~ 3.14)	0.415	1.19 (0.48 ~ 2.95)	0.710
Q3	1.24 (0.55 ~ 2.79)	0.612	1.09 (0.44 ~ 2.67)	0.856
Q4	1.63 (0.72 ~ 3.67)	0.240	1.52 (0.62 ~ 3.76)	0.360
uric acid<333.69mmol/L				
CVAI				
Q1	1.00		1.00	
Q2	2.08 (0.93 ~ 4.62)	0.073	2.39 (0.93 ~ 6.14)	0.071
Q3	3.17 (1.40 ~ 7.17)	0.006	3.75 (1.39 ~ 10.11)	0.009
Q4	3.81 (1.66 ~ 8.75)	0.002	5.84 (2.05 ~ 16.60)	< .001
BRI				
Q1	1.00		1.00	
Q2	2.06 (0.91 ~ 4.66)	0.082	2.88 (1.10 ~ 7.54)	0.032
Q3	0.89 (0.41 ~ 1.94)	0.767	1.17 (0.44 ~ 3.08)	0.757
Q4	1.94 (0.87 ~ 4.35)	0.107	2.74 (1.01 ~ 7.45)	0.048

Table 6
The effect of different cholesterol levels on prostate cancer

Variables	Univariate		Multivariate	
	OR (95%CI)	*P*	OR (95%CI)	*P*
Cholesterol ≥ 4.68mmol/L				
CVAI				
Q1	1.00		1.00	
Q2	2.00 (0.78 ~ 5.12)	0.149	2.44 (0.89 ~ 6.70)	0.083
Q3	5.88 (2.38 ~ 14.53)	< .001	8.24 (2.97 ~ 22.90)	< .001
Q4	2.77 (1.11 ~ 6.93)	0.029	5.30 (1.85 ~ 15.18)	0.002
BRI				
Q1	1.00		1.00	
Q2	2.44 (1.03 ~ 5.76)	0.042	2.88 (1.14 ~ 7.28)	0.026
Q3	2.31 (0.99 ~ 5.38)	0.053	3.62 (1.41 ~ 9.27)	0.007
Q4	1.62 (0.68 ~ 3.88)	0.274	2.56 (0.99 ~ 6.65)	0.053
Cholesterol<4.68mmol/L				
CVAI				
Q1	1.00		1.00	
Q2	1.09 (0.48 ~ 2.45)	0.837	1.36 (0.55 ~ 3.36)	0.508
Q3	1.56 (0.68 ~ 3.60)	0.293	2.32 (0.92 ~ 5.84)	0.073
Q4	1.72 (0.74 ~ 4.00)	0.205	2.52 (0.98 ~ 6.50)	0.056
BRI				
Q1	1.00		1.00	
Q2	0.69 (0.30 ~ 1.59)	0.382	0.76 (0.31 ~ 1.88)	0.550
Q3	0.78 (0.33 ~ 1.83)	0.566	1.03 (0.41 ~ 2.61)	0.947
Q4	1.02 (0.43 ~ 2.46)	0.958	1.30 (0.49 ~ 3.44)	0.592

3.6 Sensitivity analysis

To demonstrate the robustness of our results, we conducted a sensitivity analysis using Poisson regression. The findings indicate that the association between CVAI, BRI, and prostate cancer remains consistent (Table 7).

Table 7
Sensitivity Analysis

Variables	Univariate		Multivariate	
	OR (95%CI)	***P***	**OR (95%CI)**	***P***
CVAI				
Q1	1.00		1.00	
Q2	1.25 (0.81 ~ 1.95)	0.312	1.33 (0.86 ~ 2.07)	0.201
Q3	1.64 (1.09 ~ 2.50)	0.020	1.75 (1.15 ~ 2.68)	< 0.05
Q4	1.61 (1.07 ~ 2.45)	0.025	1.78 (1.17 ~ 2.73)	< 0.05
BRI				
Q1	1.00		1.00	
Q2	1.27 (0.86 ~ 1.96)	0.231	1.23 (0.83 ~ 1.86)	0.301
Q3	0.95 (0.62 ~ 1.46)	0.827	1.09 (0.70 ~ 1.68)	0.701
Q4	1.32 (0.89 ~ 1.96)	0.168	1.44 (0.97 ~ 2.15)	0.072

4 Discussion

Visceral adiposity is considered a more significant indicator of obesity-related metabolic risk than generalized obesity, and is associated with a higher risk of cancer[32]. Given its ability to reflect metabolic risk and distinguish differences in fat distribution, a growing body of research suggests that visceral obesity is associated with prostate cancer[33–35]. Although CVAI and BRI are non-invasive, low-cost indices for assessing visceral obesity, few studies have examined their association with prostate cancer.

This study retrospectively investigated the association between CVAI, BRI, and the risk of prostate cancer. Propensity score matching was used to match the case group with the control group based on age as a matching variable in order to reduce the influence of confounding factors. The results showed a linear dose-response relationship between CVAI and BRI and the risk of prostate cancer in the RCS curves. Analysis using logistic regression indicated that the Q3 group of CVAI had a 2.58-fold higher risk of prostate cancer than the Q1 group, and the BRI Q4 group had a 1.76-fold higher risk of developing prostate cancer compared to the Q1 group. This association was further strengthened in the multivariate analysis, indicating that visceral obesity, as represented by CVAI and BRI, has a significant impact on the development and progression of prostate cancer. To further investigate the impact of different metabolic indicators on the risk of prostate cancer, this study conducted subgroup analyses based on ROC curve cut-off points for different levels of urea, uric acid, and cholesterol. The results showed that in the groups with lower urea and uric acid levels, CVAI and BRI were more strongly associated with prostate cancer,

whereas cholesterol exhibited a stronger association in the higher-level groups. Furthermore, we analyzed the predictive value of CVAI and BRI for prostate cancer risk. Using ROC curves, we found that the AUCs for CVAI and BRI were 0.802 and 0.791, respectively. The results indicate that CVAI and BRI have strong predictive value for prostate cancer risk. To test the robustness of these findings, the study reanalyzed the data using Poisson regression, confirming the results' stability.

CVAI is a new visceral obesity index developed by Xia et al.[23]. Furthermore, numerous studies have confirmed that it is closely linked to a range of obesity-related conditions[36–39]. Unlike VAI, CVAI incorporates age—a well-established risk factor for prostate cancer—which may enhance its predictive value for this disease. This study found that CVAI, as an index of visceral obesity, is associated with prostate cancer, and the risk of prostate cancer increases as CVAI levels rise. CVAI includes triglyceride and high-density lipoprotein cholesterol levels.The Jungyo Suh report found a positive correlation between high triglyceride levels and high-grade prostate cancer[40]. At the same time, multiple studies have found that low levels of high-density lipoprotein cholesterol are associated with an increased risk of prostate cancer and poor prognosis[41, 42]. This may be because high-density lipoprotein cholesterol influences the proliferation and metastasis of prostate cancer through cholesterol transport proteins[43]. Importantly, elevated CVAI levels, as well as high triglyceride and high-density lipoprotein cholesterol levels, reflect abnormalities in fatty acid metabolism. Compared to normal tissues and other cancers, prostate cancer cells highly express acyl-CoA synthase 1 and 3 (ACSM1 and ACSM3) in the fatty acid anabolic pathway[44]. ACSM1 and ACSM3 drive fatty acid oxidation (FAO) for energy production by catalyzing the conversion of medium-chain fatty acids (MCFAs) into acyl-CoA derivatives; this process is a central pathway in the energy metabolism of prostate cancer cells. Furthermore, under the regulatory action of the androgen receptor (AR), AR can directly upregulate the expression of ACSM1 and ACSM3, forming an AR-ACSM metabolic axis and promoting tumor growth[45]. A study by Shrestha et al. indicated that ACSM1 and ACSM3 not only promote prostate cancer cell proliferation by enhancing energy production through fatty acid oxidation but also enable prostate cancer cells to resist the toxic effects of MCFAs and promote resistance to ferroptosis-inducing drugs and AR antagonists[44].

The BRI is a body roundness index that reflects height-related body shape and provides a more accurate estimate of visceral fat levels than traditional anthropometric indices. Individuals with a high BRI are likely to develop an accumulation of visceral fat[22]. The development of insulin resistance is closely linked to this[46]. Visceral obesity leads to elevated levels of insulin and free insulin-like growth factor-1 through insulin resistance[47]. Insulin-like growth factor-1 (IGF-1) is a polypeptide hormone; when the body is in a state of insulin resistance, the bioavailability of insulin-like growth factor-1 (IGF-1) increases[48–50]. This activates the p21 RAS/MAPK and PI3K/AKT pathways, promoting cell growth, differentiation, and mitosis, and thereby facilitating the proliferation of prostate cancer cells[51, 52].

Compared with previous studies, this study identifies visceral fat as a key risk factor for the impact of obesity on prostate cancer and elucidates the effects of the novel visceral obesity indices CVAI and BRI on prostate cancer. Given that the early symptoms of prostate cancer are not readily apparent, early screening is of particular importance[53].

This study also has certain limitations. As a single-center case-control study, although we identified associations between CVAI, BRI, and prostate cancer, we were unable to establish a causal relationship. Despite adjusting for several confounding factors, we cannot rule out the possibility that unmeasured or residual confounding factors may have influenced the results. Furthermore, due to the lack of data on prostate cancer subtypes and staging, we were unable to analyze the specific effects of histological type and tumor staging. The study sample lacked data from patients across different geographic regions, limiting the generalizability of the findings. In the future, we plan to strengthen collaboration with hospitals in various regions to collect more comprehensive data and conduct multicenter studies to further validate these findings. Finally, since this study did not include patient follow-up, we were unable to assess prostate cancer prognosis; we intend to conduct relevant cohort studies in the future to address this.

5 Conclusion

This study indicates that increases in CVAI and BRI are associated with an elevated risk of prostate cancer. CVAI and BRI are expected to serve as effective tools for the clinical assessment of prostate cancer risk. However, as cross-sectional studies cannot establish causality, further research is required. The study also highlights the role of visceral fat in the risk of prostate cancer and encourages people to actively lose weight through dietary control, regular exercise, and other methods, with a particular focus on reducing visceral fat deposits.

Declarations

6.1 Ethics approval and consent to participate

This study involved human participants and it was reviewed and approved by the Ethics Committee of the First Affiliated Hospital of Xinjiang Medical University(approval number: 20180223-93). Considering the retrospective nature of data review, informed consent was waived for this study. Clinical trial number: not applicable.This study was performed in accordance with the principles outlined in the Declaration of Helsinki.

6.2 Consent for publication

Not applicable.

6.3 Availability of data and materials

The original data supporting the conclusions of this article will be provided by the authors without undue reservation.

6.4 Competing interests

The authors declare that they have no competing interests.

6.5 Funding

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6.6 Author Contributions

1. Tianci He conceived and designed the experiments,contributed to the data research,prepared figures and/or tables and wrote the manuscript.

2. Mingyue Chen wrote the manuscript,prepared figures and/or tables and approved the final manuscript.

3.Jinru Wang analyzed the data,prepared figures and approved the final manuscript.

4. Yijie Zhou contributed to the discussion.

5.Ning Tao conceived and designed the experiments and approved the final manuscript.

6.Hengqing An contributed to the data research and analyses and approved the final manuscript.

6.7 Acknowledgments

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Figures

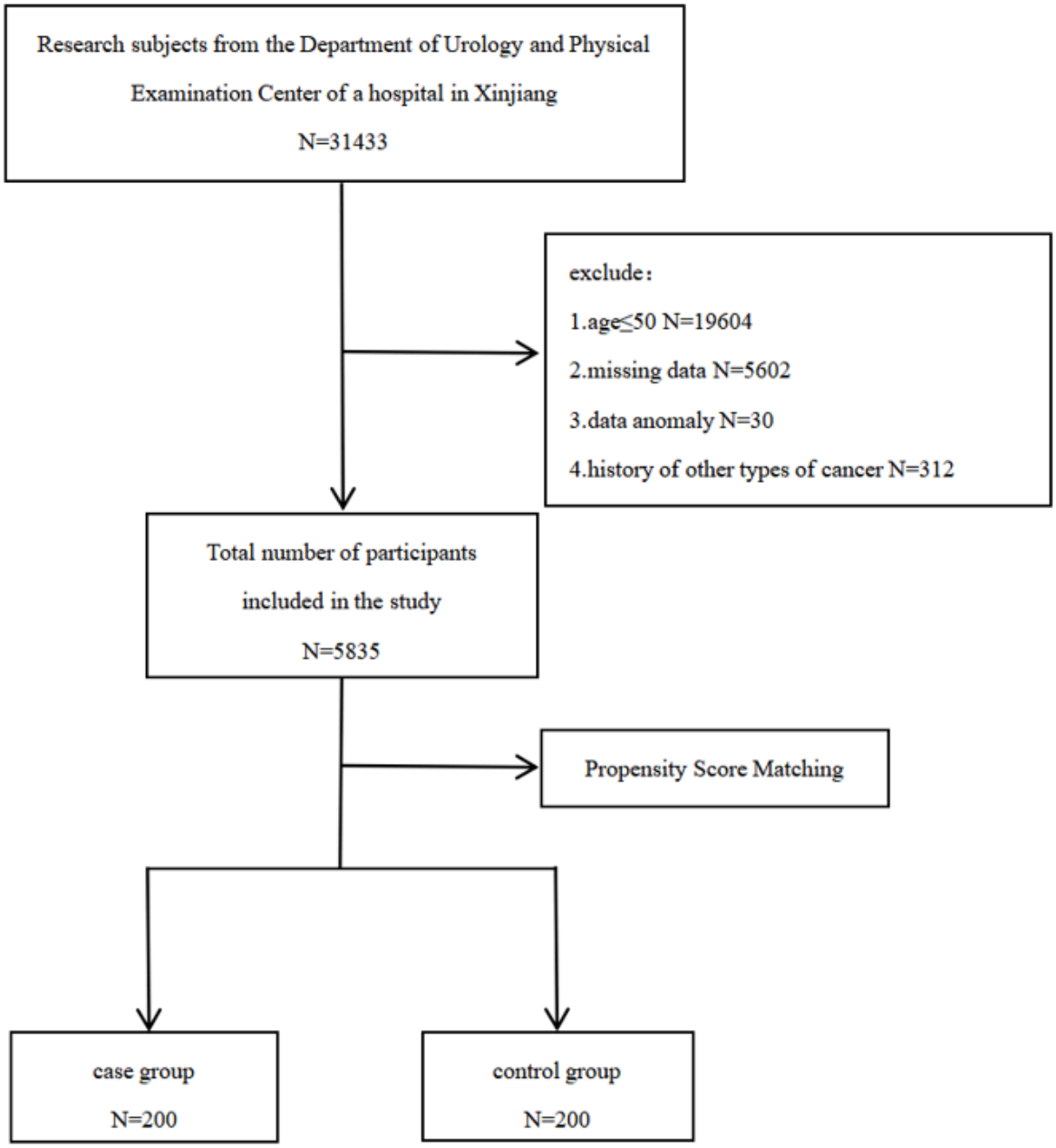


Figure 1

Flowchart of inclusion and exclusion of study participants

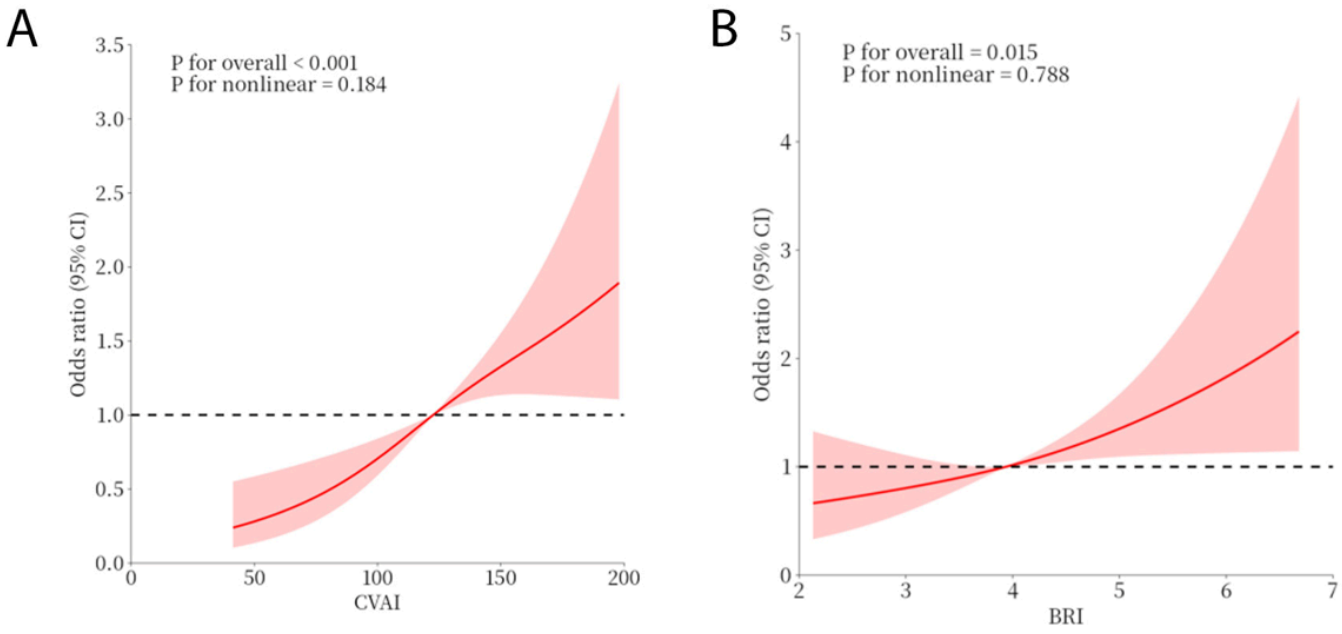


Figure 2

(A)Analysis of the RCS curve between China visceral obesity index (CVAI) and prostate cancer;(B)RCS curve analysis of Body Roundness Index (BRI) and prostate cancer

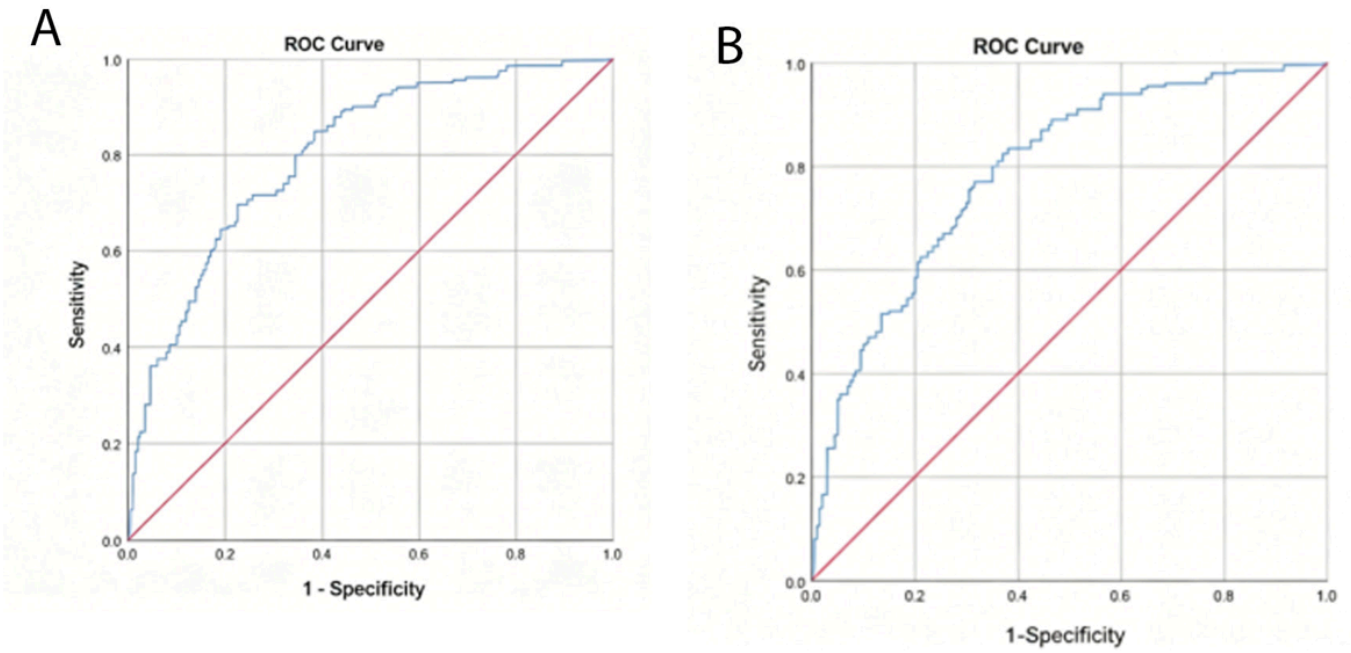


Figure 3

(A)Analysis of the ROS curve between China visceral obesity index (CVAI) and prostate cancer;(B)ROC curve analysis of Body Roundness Index (BRI) and prostate cancer