



OPEN Exploring the predictive values of ABSI and BRI in premature coronary artery disease based on a real world study

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Exploring predictors of premature coronary artery disease (PCAD) based on a real-world study. We employed 50,190 individuals and collected their baseline characteristics, including medical history, anthropometric measurements, and blood biochemistry indicators from 2001 to 2018. PCAD were screened strictly based on diagnostic and exclusion criteria, and ultimately 14,469 individuals (including 387 in coronary angiography for the diagnosis of PCAD and 14082 in the control who thought that the degree of coronary stenosis did not meet diagnostic criteria) were included in analyzed. Propensity score matching (PSM) was used to assess these patients. We used multivariable Cox proportional hazards regression to identify risk factors and developed a nomogram to estimate the risk of major adverse cardiac events (MACE) over the 36-month follow-up period. Receiver operating characteristic (ROC) analysis was used to evaluate the diagnostic performance of the risk factors. All patients were followed up for 36 months, and a survival curve was plotted. Before PSM matching, PCAD patients exhibited significantly higher levels of several risk factors, including body mass index (BMI), waist circumference (WC), triglycerides (TG), hemoglobin A1c (HbA1c), glucose, and various cardiovascular indicators such as systolic blood pressure (SBP), diastolic blood pressure (DBP), and pulse pressure (PP), compared to the control group ($P < 0.001$). After PSM matching, only the body roundness index (BRI) remained significantly higher in PCAD patients ($P < 0.001$). In Cox regression analysis and nomogram development, gender, age, smoking, serum creatinine, aspartate aminotransferase (AST), ABSI, and BRI were identified as significant risk factors for PCAD ($P < 0.01-0.05$), both before and after PSM matching. The ROC curve analysis showed that combining ABSI and BRI improved diagnostic performance, with an area under the curve (AUC) of 0.73. Survival analysis revealed that high ABSI and BRI significantly predicted poorer prognosis in PCAD patients, regardless of PSM matching ($P < 0.05$). Further survival analysis combining ABSI and BRI demonstrated that, after PSM matching, high ABSI consistently indicated a worse prognosis, irrespective of BRI levels. ABSI and BRI are high-risk factors for PCAD, and ABSI combined with BRI with a higher diagnosis. High ABSI is associated with poor prognosis in PCAD patients, and the exact mechanism needs to be further explored.

Keywords PCAD, Real-world study, Propensity score matching, BRI, ABSI

Abbreviations

PSM	propensity score matching
ROC	receiver operating characteristic
CAD	coronary artery disease
PCAD	premature coronary artery disease

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DM	diabetes mellitus
COPD	Chronic Obstructive Pulmonary Disease
RWS	Real World Study
BMI	Body Mass Index
DBP	diastolic blood pressure
SBP	systolic blood pressure
RWD	real-world data
RCTs	Randomized Controlled Trials
ABSI	associations of a body shape index
BRI	body roundness index
WC	waist circumference
TC	total cholesterol
TG	triglyceride
LDL	low-density lipoprotein
HDL	high-density lipoprotein
MACE	major adverse cardiac events
TyG	Triglyceride-glucose index
MetS	metabolic syndrome
PP	Pulse Pressure
AUC	receiver operating characteristic curve area under curve

Ischemic heart disease remains one of the leading causes of death worldwide¹. With the advancement of medicine, significant breakthroughs have been made in the prevention and treatment of coronary artery disease. However, the incidence of CAD continues to increase worldwide, and there is a trend towards younger individuals being affected². Currently, secondary prevention is very common. PCAD is an aggressive form of CAD, and despite our active participation in secondary prevention, its mortality and recurrence rates remain high. We still know little about primary prevention and long-term disease progression of these young CAD patients³. Race, accompanying inflammatory diseases, lifestyle habits, and inadequate control of risk factors are associated with poor prognosis. Predicting risk and disease development is challenging, and cardiovascular disease is no exception. Accurately determining disease risk in young CAD populations is crucial for taking precise prevention measures. Although the effects of gender, diabetes mellitus, smoking, high blood pressure, physical inactivity and other factors on the prognostic risk of PCAD have been studied in some studies, this issue is still complex and controversial. Importantly, relevant prior studies have been limited to small populations⁴.

The latest developments have brought a new era of concept to the applicability of real-world studies (RWS) in clinical medicine, such as the first approval of IBRANCE's label expansion for male breast cancer based on RWS⁵. RWS is defined as a series of clinical evidence derived from analyzing real-world data (RWD), including risk assessment, the benefits and drawbacks of a technology, potential gains, disease prognosis, and more⁶. The most common bias in real-world event studies is the inevitable generation of systematic errors⁷. RWS offers advantages over RCTs, such as larger sample sizes, longer observation periods, and better control of confounding factors. It is a cost-effective approach that provides valuable insights into treatment effectiveness and patient outcomes. Under normal circumstances, having taken measures to minimize bias 'by design', the next step is to implement appropriate analytical techniques (for example, propensity scores) to minimize the remnant potential biases^{6,7}. These characteristics make RWS a valuable tool in studying PCAD, as it helps overcome the limitations of previous studies and fosters a more comprehensive approach to understanding the disease. RWS opens up new avenues for research and provides a broader perspective in the field of PCAD.

There is currently no further evidence on the relationship between the disease characteristics of PCAD and traditional cardiovascular risk factors, as well as the effectiveness of secondary prevention of PCAD⁸. Past studies on PCAD often have many limitations, including small sample size, lack of cause-specific mortality rates, and short follow-up periods^{9,10}. To address the needs of PCAD research, we require evidence from real-world studies to help identify high-risk populations and make more reasonable secondary prevention for high-risk PCAD patients¹¹.

Methods

Study population

We enrolled 50,190 patients for the study, including patients hospitalized in Liuzhou People's Hospital Affiliated to Guangxi Medical University due to chest tightness, shortness of breath and chest pain from 2001 to 2018. The diagnostic criteria and exclusion criteria for coronary artery disease are the same as previous studies¹². Diagnosis criteria for PCAD: confirmed diagnosis of CAD; CAD manifested in males < 55 years old and in females < 65 years old was defined a PCAD^{4,13}. Exclusion criteria are as follows: having a major illness with a life expectancy of less than 6 months, participating in another clinical study at the same time, and researchers having difficulties in tracking and following up participants due to speech impairment, mental disorders, long-term travel, or lack of effective contact information. After strict screening, we enrolled 14,469 individuals for the analysis (including 387 in coronary angiography for the diagnosis of PCAD and 14082 in the control who thought that the degree of coronary stenosis did not meet diagnostic criteria). The study protocol was reviewed and approved by the Medical Ethics Committee of Liuzhou People's Hospital (Approval No. KY2025-009-01), and written informed consent was obtained from all participants prior to enrollment. For the specific analysis process, see Fig. 1.

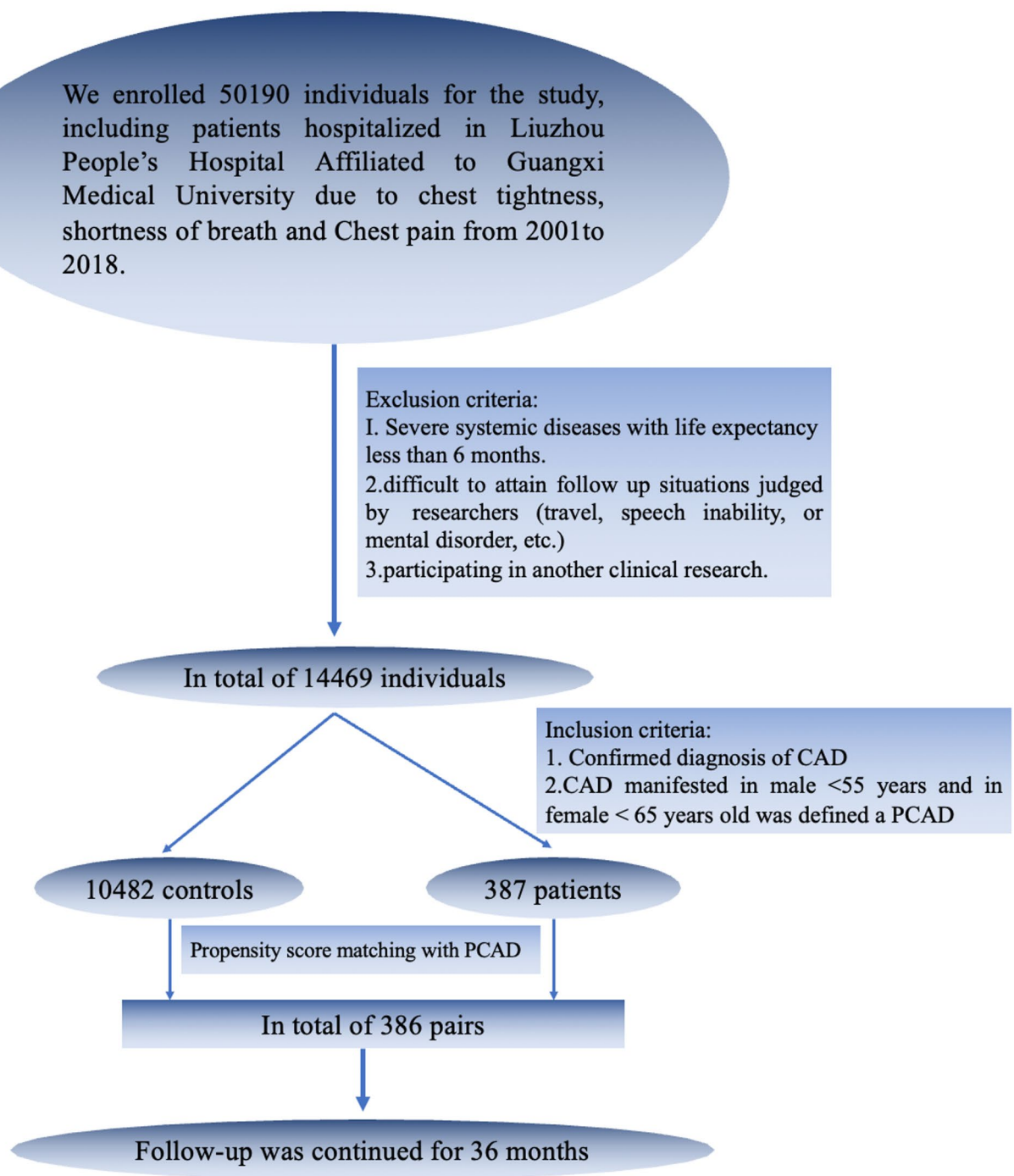


Fig. 1. Process of recruiting patients for the study. CAD, coronary artery disease; PCAD, premature coronary artery disease;

Data collection

We collected baseline clinical data from patients using electronic medical records, including age, gender, smoking habits (current, never smoked, former smoker), alcohol consumption (mean intake > 50 g/day for over 1 year), systolic blood pressure (SBP), diastolic blood pressure (DBP), body mass index (BMI), waist circumference (WC), A Body Shape Index (ABSI), Body Roundness Index (BRI), comorbid conditions, and history of tumors. During baseline collection, professional researchers used standardized protocols to obtain anthropometric measurements, including height, weight, waist circumference, and so on. ABSI was calculated using the formula $ABSI = WC / (BMI^{2/3} \times height^{1/2})^{14}$. BRI is defined as $364.2 - 365.5 \times \{1 - [(WC/2\pi)/(0.5 \times height)]^2\}^{0.5}$. BRI is a predictor of body fat percentage and visceral adiposity tissue, and its values range from 1 to 16¹⁵. Additionally, laboratory tests were conducted, including fasting blood glucose, glycosylated hemoglobin,

complete blood count, total cholesterol, triglycerides, HDL cholesterol, LDL cholesterol, creatinine, and blood urea nitrogen. Data on patients' lifestyle habits, such as smoking, alcohol consumption, and medical history, including hypertension, diabetes, coronary artery disease, COPD, autoimmune diseases, and cancer therapy, were obtained through medical records or questionnaires. The data collection procedures, instruments used, and reference ranges were consistent with our previous research¹².

Treatment and follow-up

There were no predefined principles of antiplatelet therapy in the protocol. All treatments for cardiovascular disease, including antiplatelet therapy, interventional therapy, anticoagulation, etc., were jointly determined by two senior cardiovascular physicians. Patients were followed up regularly at the 3rd, 6th, 9th, and 12th months after enrollment, including via telephone, rehospitalization, and outpatient clinic visits.

The time of follow-up was 36 months in total. No advice on cardiovascular treatment was provided to patients during regular telephone follow-up unless they were in an emergency situation. Follow-up was conducted as scheduled in general, and post-discharge patient data were collected, including control of risk factors, relief of symptoms, treatment, laboratory tests, and examinations.

Endpoint definitions

The primary outcome was patient-oriented composite endpoints at 36 months. Patients who cannot complete follow-up for any reason will not be included in the final analysis. The main endpoint was major adverse cardiac events (MACE), a composite of cardiac death, MI and ischemic stroke.

Statistical methods

We statistically analyzed the data using the SPSS 22.0 package and GraphPad Prism 9.5. Categorical variables were expressed in percentages (%), and chi-square tests were performed to analyze differences. Quantitative variables were expressed as mean and standard deviation, and differences were analyzed using the independent unpaired student's t-test. $P < 0.05$ was considered statistically significant. We performed propensity score matching (PSM) using propensity scores estimated from a multivariable model including age, sex, smoking, alcohol use, hypertension, diabetes, metabolic syndrome, cancer, nicotine metabolites, pulse pressure, systolic/diastolic blood pressure, Charlson Comorbidity Index, triglycerides, HbA1c, glucose, blood urea nitrogen, AST, ALT, creatinine, LDL, HDL, and total cholesterol. Matching was conducted with a caliper width of 0.2 of the standard deviation of the logit of the propensity score. Covariate balance after matching was assessed using standardized mean differences. We performed 1:1 propensity score matching. Standardized mean differences (SMDs) were used to assess covariate balance after PSM; an SMD < 0.10 was considered indicative of adequate balance. We used the 'table one' package in R to design a propensity matching score design for the selected population sample. A multivariable Cox proportional hazards model was used to determine the risk of PCAD disease and potential significant prognostic factors. According to the final Cox proportional hazard regression model, using the 'rms' package in R, the nomogram model was constructed. The model was validated by plotting the predicted standard curve. The higher the fit between the predicted curve and the standard curve, the more accurate and excellent the predictive model results. We calculated the area under the ROC curve (AUC) value to determine the diagnostic performance of these variables. Patients were divided based on the optimal parameter of the ROC curve into two groups (high and low). The survival curves were plotted using the Kaplan-Meier method, and the log-rank test was used to compare the curves.

Results

General clinical features of patients hospitalized for PCAD

A total of 14,469 individuals participated in this study. Patients who meet the diagnostic criteria for PCAD form one group, the one who not meeting diagnostic criteria by coronary angiography served as the Control group. There were 387 patients with PCAD and 14,082 individuals in the Control group. Using PSM matching, 386 patients with PCAD and 386 members of the normal population were included in the study. Before PSM matching (Table 1), the proportion of female, drink, cotinine, cancer, smoking, hypertension, diabetes, metabolic syndrome (MetS) and chronic obstructive pulmonary disease; the levels of body mass index (BMI), waist circumference (WC), triglycerides (TG), hemoglobin A1c (HbA1c), glucose, the proportion of smoking ($P < 0.001$), pulse pressure (PP), systolic blood pressure (SBP), diastolic blood pressure (DBP), charlson comorbidity index (CCI), body roundness index (BRI), associations of a body shape index (ABSI) and Triglyceride-glucose index (TyG) were higher in PCAD patients than in the normal group ($P < 0.001-0.0014$). After PSM matching (Table 2), only the levels of BRI were higher in PCAD patients ($P < 0.001$). Thus, whether PSM is performed or not, only the levels of BRI were higher in PCAD patients than in the control. Balance before and after matching. Before PSM, baseline imbalance was substantial: among 29 covariates, only 8 had a SMD < 0.10 , 17 were ≥ 0.20 , the mean |SMD| was 0.28 (maximum 0.79), and 21 variables showed statistically significant differences ($P < 0.05$). After PSM, 386 matched pairs were obtained; all covariates had SMD < 0.10 (22 were < 0.05), the mean |SMD| decreased to 0.03 (maximum 0.09), and none of the covariate tests were significant ($P \geq 0.05$). Collectively, these findings indicate that PSM markedly improved baseline balance, reduced potential confounding, and enhanced the comparability and robustness of subsequent outcome analyses.

Nomogram model of PCAD

A nomogram model that included the important predictors in the Cox analysis was established to predict the prognosis of PCAD (Fig. 2). Before PSM matching, smoking, drinking, gender, age, COPD, Ast, CCI, BUN, Hypertension, SBP, Scr, Cotinine, Glu, BRI and ABSI ($P < 0.05-0.001$) were identified as significant risk factors

Parameter	Control	PCAD	P	SMD
Number	14,082	387		
Gender = Male (%)	7720 (54.8)	122 (31.5)	<0.0001	0.484
BMI (mean (SD))	28.74 (6.47)	32.10 (8.34)	<0.0001	0.45
WC (mean (SD))	98.82 (15.91)	106.06 (17.56)	<0.0001	0.432
Drink = Yes (%)	2358 (16.7)	81 (20.9)	<0.0001	0.107
Cotinine (mean (SD))	61.82 (130.94)	131.96 (168.96)	<0.0001	0.464
Cancer = Yes (%)	1308 (9.3)	46 (11.9)	<0.0001	0.085
LDL (mean (SD))	2.97 (0.92)	2.99 (1.05)	0.71	0.018
HDL (mean (SD))	1.39 (0.41)	1.36 (0.47)	0.21	0.088
TC (mean (SD))	4.99 (1.05)	5.07 (1.18)	0.18	0.073
TG (mean (SD))	1.36 (0.77)	1.61 (0.86)	<0.0001	0.3
BUN (mean (SD))	4.85 (1.99)	4.79 (2.89)	0.68	0.023
Alt (mean (SD))	25.53 (24.10)	26.21 (21.99)	0.55	0.029
Ast (mean (SD))	25.45 (17.54)	26.03 (20.47)	0.58	0.03
HbA1c (mean (SD))	5.69 (0.99)	6.11 (1.57)	<0.0001	0.327
Glu (mean (SD))	107.38 (31.91)	119.53 (53.95)	<0.0001	0.274
Age (mean (SD))	49.65 (17.71)	50.17 (9.51)	0.3	0.037
Scr (mean (SD))	94.10 (22.68)	91.82 (22.84)	0.053	0.1
Smoke (%)			<0.0001	0.52
former	3993 (28.4)	90 (23.3)		
never	6872 (48.8)	118 (30.5)		
now	3217 (22.8)	179 (46.3)		
Hypertension = yes (%)	5800 (41.2)	276 (71.3)	<0.0001	0.637
DM = no (%)	12,080 (85.8)	277 (71.6)	<0.0001	0.352
MetS = yes (%)	4367 (31.0)	204 (52.7)	<0.0001	0.451
COPD = yes (%)	676 (4.8)	41 (10.6)	<0.0001	0.219
PP (mean (SD))	70.38 (12.29)	73.78 (13.17)	<0.0001	0.267
SBP (mean (SD))	123.08 (18.11)	126.18 (18.80)	0.0014	0.168
DBP (mean (SD))	69.87 (11.73)	73.22 (12.66)	<0.0001	0.275
CCI (mean (SD))	0.97 (1.45)	2.26 (1.81)	<0.0001	0.787
BRI (mean (SD))	5.32 (2.22)	6.65 (2.77)	<0.0001	0.53
TyG (mean (SD))	8.61 (0.62)	8.83 (0.69)	<0.0001	0.347
ABSI (mean (SD))	0.08 (0.00)	0.08 (0.00)	<0.0001	0.175

Table 1. General characteristics of patients before PSM matching. CAD, coronary artery disease; CAD, premature coronary artery disease; MI, Body Mass Index; DBP, diastolic blood pressure; SBP, systolic blood pressure; ABSI, associations of a body shape index; BRI, body roundness index; WC, waist circumference; TC, total cholesterol; TG, triglyceride; LDL, low-density lipoprotein; HDL, high-density lipoprotein; MACE, major adverse cardiac events; TyG, Triglyceride-glucose index; MetS, metabolic syndrome; PP, Pulse Pressure. Categorical variables were expressed in percentages (%) and grouped according to clinical regulations, and using chi-square were performed to analyze differences. The quantitative variables were presented as the mean \pm standard deviation and using the unpaired student's t-test. $p < 0.05$ was considered statistically significant. DM, diabetes mellitus; COPD, Chronic Obstructive Pulmonary Disease.

for PCAD (Fig. 2A). The model predicted individualized 3-year risks of MACE ranging from 0.2% to 60%. Draw the predicted calibration curve, the standard curve, and predicted calibration curve before PSM matching showed a good fit with a nearly perfect alignment (Fig. 2B). After PSM matching, gender, age, smoking, Scr, AST, ABSI and BRI ($P < 0.05$ – 0.01) were identified as important disease risks for PCAD (Fig. 2C). The model predicts that the proportion of PCAD occurrence within 3 years ranges from 0.2% to 90%. The fit of the standard curve decreased compared to before matching, but still meets the evaluation criteria (Fig. 2D). By comparing two nomograms, we found that gender, age, smoking, Scr, Ast, ABSI, and BRI are all high-risk factors for PCAD, both before and after PSM matching.

Diagnostic efficiency

The results revealed that many risk factors, including BMI, WC, ABSI, and BRI were significantly associated with PCAD, and we can use them to predict the occurrence of PCAD. Obesity is a significant risk factor for cardiovascular disease. BMI, WC, ABSI, and BRI are commonly used indicators for assessing obesity.

Next, we calculated the AUC values to determine the threshold selection and diagnostic performance for the following risk factors before PSM matching (Fig. 3): BMI (AUC = 0.618, Fig. 3A), WC (AUC = 0.612, Fig. 3B),

Parameter	Control	PCAD	P	SMD
Number	386	386		
Gender = Male (%)	119 (30.8)	122 (31.6)	0.815	0.017
BMI (mean (SD))	31.67 (7.97)	32.04 (8.27)	0.527	0.047
WC (mean (SD))	105.29 (17.81)	105.93 (17.39)	0.613	0.036
Drink = Yes (%)	67 (17.4)	81 (21.0)	0.234	0.092
Cotinine (mean (SD))	136.44 (196.64)	132.25 (169.08)	0.751	0.023
Cancer = Yes (%)	46 (11.9)	46 (11.9)	0.999	<0.001
LDL (mean (SD))	2.99 (1.00)	2.99 (1.04)	0.999	<0.001
HDL (mean (SD))	1.34 (0.38)	1.36 (0.47)	0.515	0.049
TC (mean (SD))	5.02 (1.09)	5.07 (1.18)	0.541	0.047
TG (mean (SD))	1.53 (0.83)	1.60 (0.85)	0.247	0.086
BUN (mean (SD))	4.70 (2.32)	4.78 (2.88)	0.67	0.033
Alt (mean (SD))	25.59 (18.35)	26.25 (22.00)	0.65	0.033
Ast (mean (SD))	25.23 (13.93)	26.06 (20.49)	0.51	0.047
HbA1c (mean (SD))	6.10 (1.48)	6.11 (1.57)	0.927	0.005
Glu (mean (SD))	118.66 (50.45)	119.55 (54.01)	0.813	0.017
Age (mean (SD))	50.34 (16.02)	50.18 (9.52)	0.866	0.012
Scr (mean (SD))	91.62 (24.43)	91.96 (22.70)	0.841	0.014
Smoke (%)			0.672	0.064
former	98 (25.4)	90 (23.3)		
never	108 (28.0)	118 (30.6)		
now	180 (46.6)	178 (46.1)		
Hypertension = yes (%)	274 (71.0)	275 (71.2)	0.999	0.006
DM = no (%)	276 (71.5)	277 (71.8)	0.999	0.006
MetS = yes (%)	206 (53.4)	203 (52.6)	0.999	0.016
COPD = yes (%)	36 (9.3)	41 (10.6)		0.043
PP (mean (SD))	73.58 (13.53)	73.73 (13.15)	0.875	0.011
SBP (mean (SD))	125.62 (19.83)	126.14 (18.81)	0.708	0.027
DBP (mean (SD))	73.40 (13.34)	73.17 (12.63)	0.805	0.018
CCI (mean (SD))	2.26 (2.16)	2.25 (1.80)	0.944	0.005
BRI (mean (SD))	6.49 (2.66)	6.93 (2.24)	0.013	0.05
TyG (mean (SD))	8.78 (0.68)	8.83 (0.68)	0.307	0.073
ABSI (mean (SD))	0.08 (0.01)	0.08 (0.00)	0.999	0.002

Table 2. General characteristics of patients after PSM matching. CAD, coronary artery disease; PCAD, premature coronary artery disease; BMI, Body Mass Index; DBP, diastolic blood pressure; SBP, systolic blood pressure; ABSI, associations of a body shape index; BRI, body roundness index; WC, waist circumference; TC, total cholesterol; TG, triglyceride; LDL, low-density lipoprotein; HDL, high-density lipoprotein; MACE, major adverse cardiac events; TyG, Triglyceride-glucose index; MetS, metabolic syndrome; PP, Pulse Pressure. DM, diabetes mellitus; COPD, Chronic Obstructive Pulmonary Disease.

BRI (AUC = 0.627, Fig. 3C), ABSI (AUC = 0.531, Fig. 3D). The AUC values range from 0 to 1, with values closer to 1 indicating higher diagnostic performance. The diagnostic performance of BRI is most prominent, followed by BMI, WC, and ABSI. The point that balances sensitivity and specificity and maximizes the model's classification performance is considered the threshold for these clinical indicators, as determined by the ROC curve (Fig. 3). Therefore, we determined the threshold selection for these risk factors: BMI (29.99, specificity: 0.638, sensitivity: 0.562, Fig. 3A); WC (101.15, specificity: 0.593, sensitivity: 0.592, Fig. 3B); BRI (5.83, specificity: 0.640, sensitivity: 0.567, Fig. 3C); ABSI (0.081, specificity: 0.443, sensitivity: 0.619, Fig. 3D). Based on the thresholds obtained, we divided these four risk factors into high and low groups. Based on whether the values of ABSI and BRI for the patients are higher than the thresholds, the patients are divided into four groups: Group 1 (BRI = low, ABSI = low), Group 2 (BRI = high, ABSI = low), Group 3 (BRI = low, ABSI = high), and Group 4 (BRI = high, ABSI = high). A follow-up was conducted for the grouped patients to record the time of the occurrence of the endpoint event.

After PSM matching, we recalculated the AUC values to determine the diagnostic performance of the following risk factors (Fig. 4A): BMI (AUC = 0.534), WC (AUC = 0.547), BRI (AUC = 0.565), ABSI (AUC = 0.725). It can be seen that ABSI has the highest diagnostic performance, followed by BRI ($P = 0.0326$). We calculated the diagnostic performance again by combining ABSI and BRI, resulting in an improved AUC of 0.73 (Fig. 4B). And ABSI and ABSI + BRI were compared, and $p = 0.023$ indicates statistical significance. Therefore, the combination of ABSI and BRI provided the best diagnostic performance. A BRI of more than 5.83 and an ABSI of more than 0.081 were good predictors of the onset of PCAD.

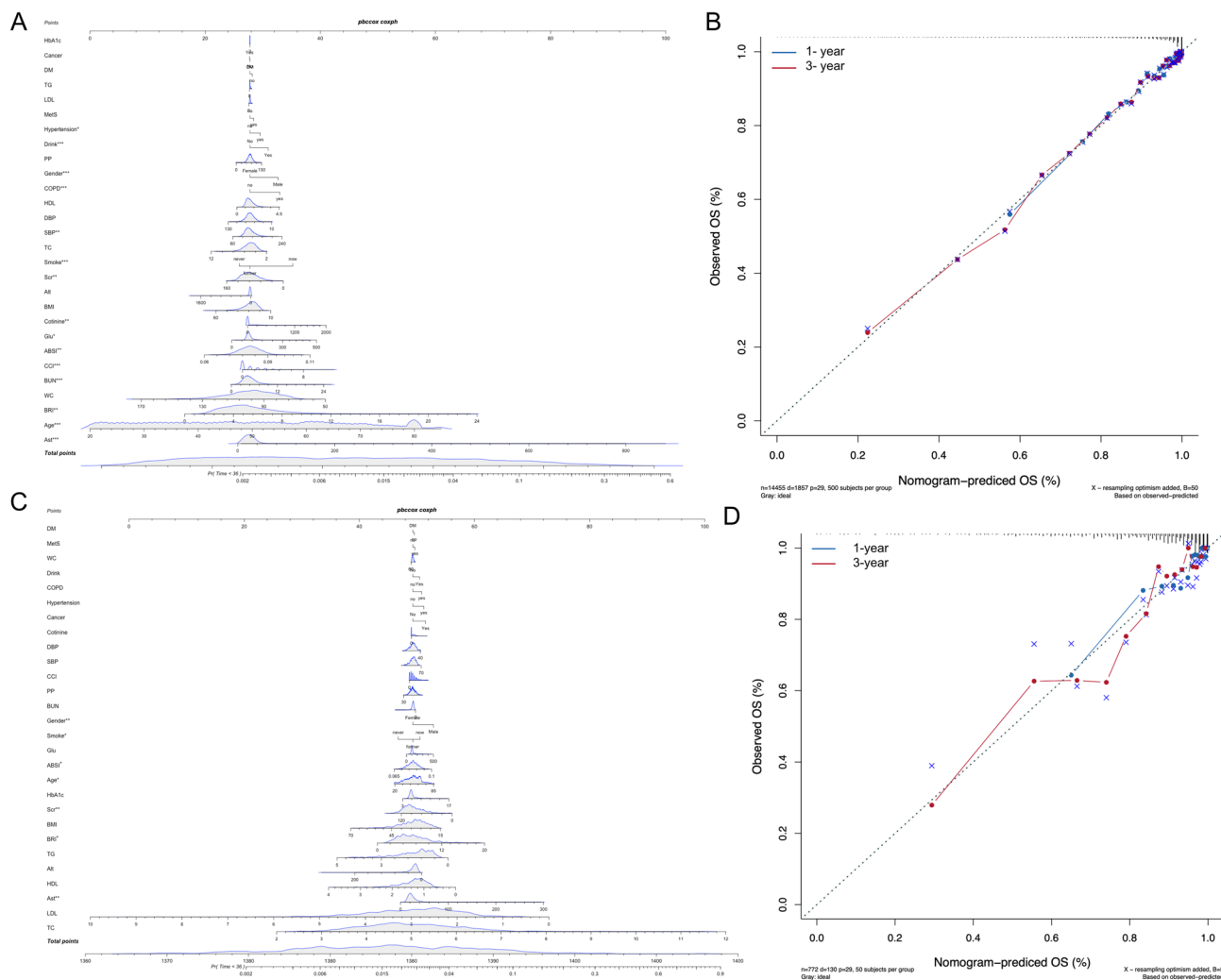


Fig. 2. Nomogram used for evaluating disease risk in PCAD patients to PSM matching. (A) shows the Nomogram before PSM matching, and (B) tests its accuracy. (C) shows the Nomogram after PSM matching, and (D) tests its accuracy. Each warning variable characteristic has a corresponding point value based on its position on the vertex scale and contribution to the model. The probability of PCAD for each subject is calculated by adding up the points for each variable, resulting in a total point value that corresponds to the probability of PCAD on the baseline scale. The variable data include diabetes, MetS, waist circumference, drink, COPD, hypertension, cancer, Cotinine, DBP, SBP, CCI, PP, BUN, gender, smoking, Glu, ABSI, age, glycated hemoglobin, Scr, BMI, BRI, TG, Alt, HDL, Ast, LDL, TC, TG. The discriminatory ability of the model is evaluated using the standard curve method.

Survival analysis of patients with PCAD

The follow-up endpoint was cardiovascular death and/or all-cause death. Survival curves were plotted for the endpoint events of patients followed up for 3 years (Fig. 5). Before PSM matching, there was no significant difference in survival time between the high and low BMI groups, although the *P*-value was 0.22 and lacked statistical significance (Fig. 5A). The number of endpoint events in the high WC group was higher than that in the low WC group ($P < 0.001$, Fig. 5B). The number of endpoint events in the high BRI group was higher than that in the low BRI group ($P < 0.001$, Fig. 5C), with shorter survival time. The number of endpoint events in the high ABSI group was significantly higher than that in the low ABSI group ($P < 0.001$, Fig. 5D).

After PSM matching (Fig. 6), the number of endpoint events in the high BMI group was higher than that in the low BMI group (Fig. 6A), but the *P*-value was 0.28 and lacked statistical significance. The number of endpoint events in the high WC group was higher than that in the low WC group at between 2 and 3 years of follow-up (Fig. 6B), but there was no difference in the total number of survivors at 3 years, and the *P*-value was 0.64 and lacked statistical significance. The number of endpoint events in the high BRI group was higher than that in the low BRI group ($P = 0.46$, Fig. 6C), with a shorter survival time. The number of endpoint events in the high ABSI group was significantly higher than that in the low ABSI group ($P < 0.001$, Fig. 6D).

ABSI+BRI has good diagnostic significance and can be used to assess the risk of PCAD. We conducted interaction survival curves for ABSI and BRI (Fig. 7). We define four groups: Group 1 (BRI=low, ABSI=low),

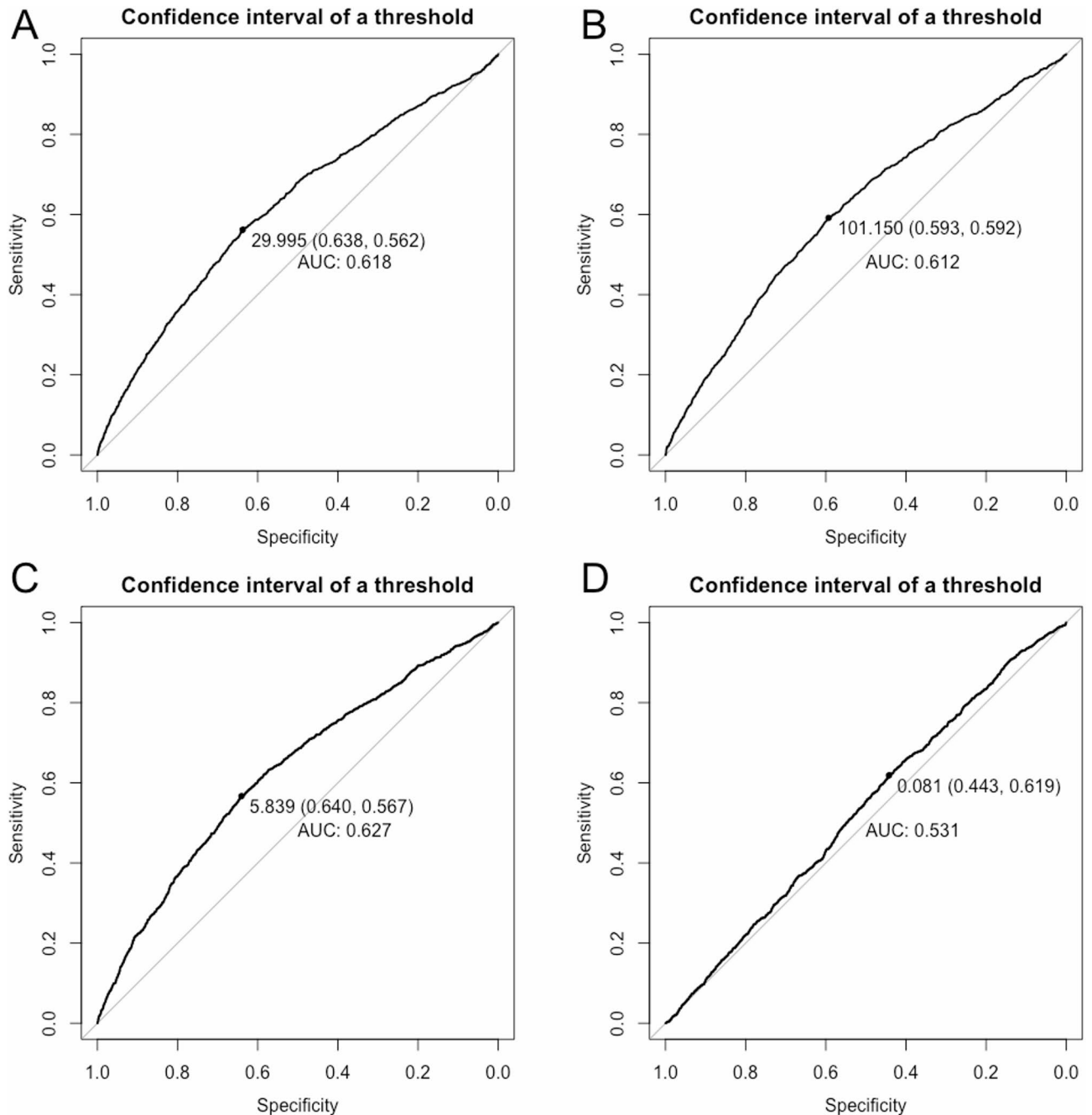


Fig. 3. Before PSM matching, ROC curves predicting the risk of PCAD disease. (A) BMI (AUC=0.618), body mass index; (B) WC (AUC=0.612), waist circumference; (C) BRI (AUC=0.627), body roundness index; (D) ABSI (AUC=0.531), associations of a body shape index; ROC, receiver operating characteristic.

Group 2 (BRI=high, ABSI=low), Group 3 (BRI=low, ABSI=high), and Group 4 (BRI=high, ABSI=high), where the values used for grouping are described earlier in the text. Before PSM matching (Fig. 7A), it can be seen that Group 4 (hazard ratio, HR=3.883) had the highest endpoint events over time, followed by Group 3 (HR=3.164), Group 1 (HR=1), and Group 2 (HR=0.779). It can be seen that the survival of the four groups (high BRI, high ABSI) is the shortest. The *p*-values for pairwise comparisons between groups were all less than 0.05, indicating statistical differences. After PSM matching (Fig. 7B), Group 3 (HR=2.712) and Group 4 (HR=2.793) had the highest incidence rate of endpoint events, but the difference between the two groups was not statistically significant. The incidence rate of endpoint events fluctuated, sometimes higher in one group and sometimes higher in another. The reasons for this phenomenon of cross-occurrence are currently unknown. Group 1 (HR=1.000) and Group 2 (HR=0.992) had similar prognoses, significantly lower than Group 3 and Group 4. The *P*-values for pairwise comparisons between groups were all less than 0.05, indicating statistically significant differences. After comprehensive appeal, After combining the above, patients with high BRI and

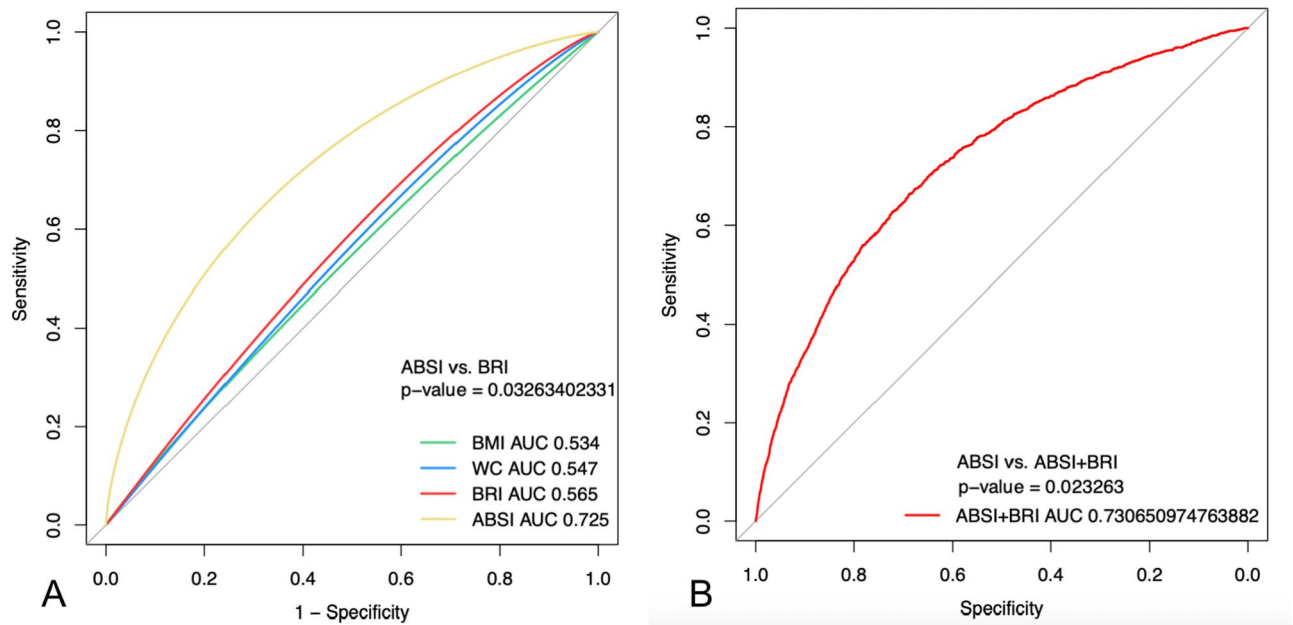


Fig. 4. After PSM matching, ROC curves predicting the risk of PCAD disease. (A) The ROC curves for ABSI (AUC = 0.565), BRI (AUC = 0.565), WC (AUC = 0.547), and BMI (AUC = 0.534) in predicting the risk of PCAD disease. (B) The ROC curves for ABSI + BRI, AUC = 0.725. ABSI, associations of a body shape index; BRI, body roundness index; WC, waist circumference; BMI, Body Mass Index.

ABSI had a poor prognosis before PSM matching. After PSM matching, patients with high ABSI had a poor prognosis regardless of whether their BRI was high or low. Therefore, ABSI is the best indicator for evaluating the prognosis of patients with PCAD.

Discussion

In this large-scale regression cohort study in the real world, risk factors and prognostic indicators for PCAD patients were revealed to a certain extent. In this study, we analyzed the baseline characteristics of the control and blank groups before and after PSM matching and assessed the disease risk of PCAD through COX analysis, ROC analysis, and survival analysis. We attempted to establish a predictive model for PCAD using a nomogram. After long-term follow-up, survival curves were plotted to assess whether the identified risk factors had an impact on the prognosis. We acknowledge that variables on the causal pathway—such as BMI and WC—were included in the PSM model, which may introduce post-treatment adjustment and potential collider bias. The persistent imbalance in BRI after matching indicates suboptimal covariate balance and possible residual confounding. Therefore, we obtained the following main results: both BRI and ABSI have significant meaning in the nomogram, regardless of PSM status, and are risk factors for PCAD. BRI has the greatest diagnostic performance before PSM matching, whereas ABSI has the greatest diagnostic performance after PSM matching. ABSI + BRI yields the best diagnostic performance, with an AUC of 0.73. Patients with high BRI and high ABSI have the poorest prognosis, and ABSI and BRI are the reasons for a worse prognosis in PCAD.

Although the association between dyslipidemia and cardiovascular disease has been well established¹⁶, the link between obesity and cardiovascular disease remains controversial¹⁷. On the one hand, obesity itself directly increases the risk of cardiovascular disease by leading to metabolic syndrome¹⁸. The essence of obesity is the impairment of fat storage and utilization¹⁹, which results in the accumulation of lipids in blood vessels and excessive release of fatty acids, lipids, and other substances that cause chronic inflammation²⁰. This not only initiates coronary heart disease but also promotes its progression²¹. Furthermore, the association between obesity and hypertension, diabetes, dyslipidemia, and sleep apnea syndrome has been proven to increase the incidence of cardiovascular disease²². Obesity patients generally have a poorer prognosis after developing coronary artery disease²², which is attributed to a larger body surface area, postoperative infections, in-stent restenosis, multiple other vascular complications, and an independent elevation of MACE risk^{16,23}.

Obesity has become an increasingly serious health problem worldwide, with the prevalence of obesity rising in both developed and developing countries²⁴. Waist circumference (WC) is a component of body shape measurement and reflects excessive obesity and health risks. However, there are still differences as to whether calibration is needed for different heights²⁵. Body Mass Index (BMI) is used to measure the degree of obesity; however, it does not provide information about fat distribution, which is highly relevant to cardiovascular risk. ABSI considers obesity indices for both overall and central obesity¹⁴, while BRI is used to predict the percentage of body fat and visceral fat tissue¹⁵, which allows for a comprehensive evaluation of obesity levels in the human body.

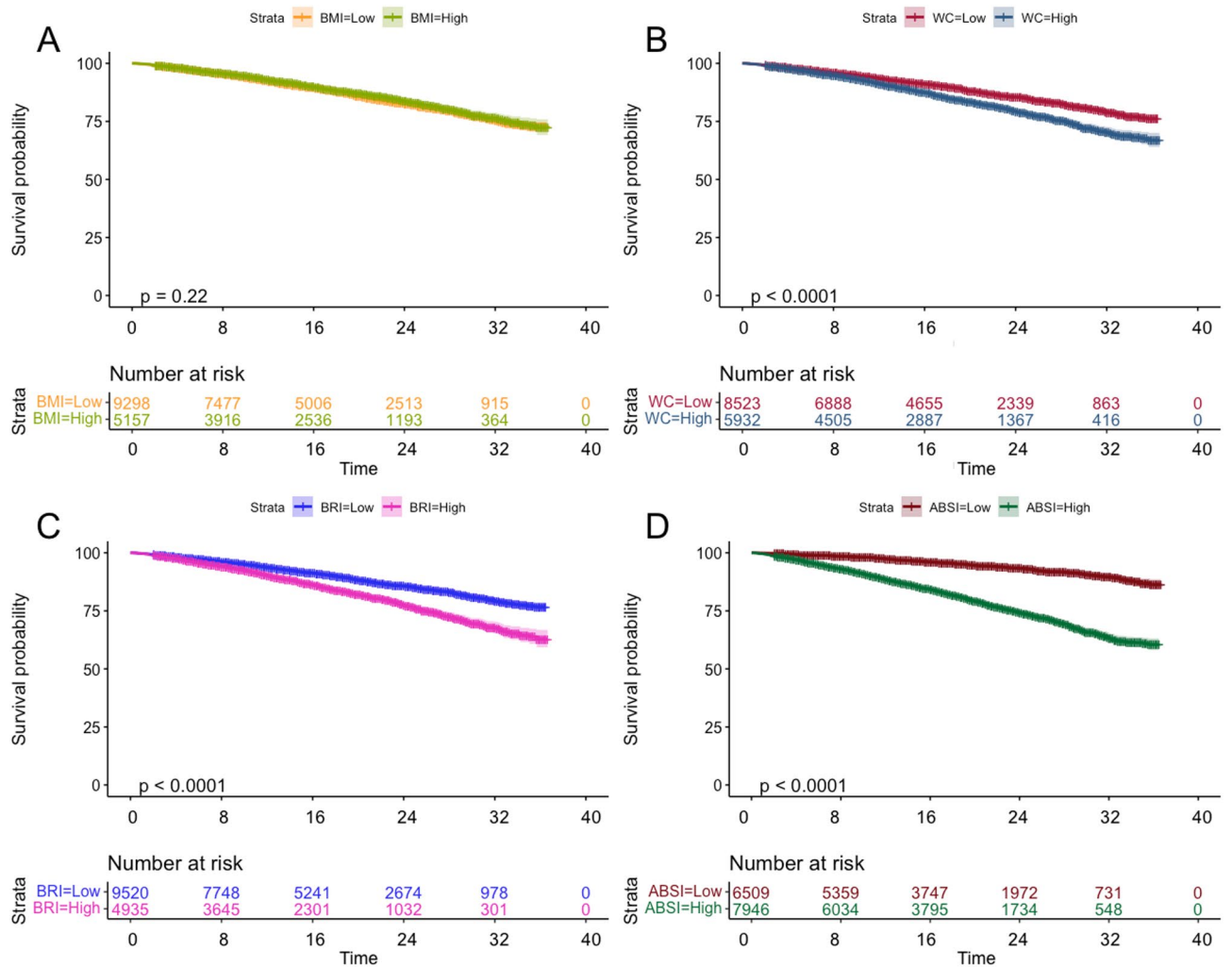


Fig. 5. Before PSM matching, the survival curves show the impact of ABSI, BRI, WC, and BMI on the prognosis of PCAD patients. (A) The follow-up of patients with different BMI group for 36 months. (B) The follow-up of patients with different WC group for 36 months. (C) The follow-up of patients with different BRI group for 36 months. (D) The follow-up of patients with different ABSI group for 36 months.

Yang Li et al. followed 500,090 participants for 8 years and evaluated the association between anthropometric measurements and CAD in each BMI category using logistic regression models. They found that BRI and WHR (waist-to-hip ratio) were better than other anthropometric methods in determining the presence of CAD²⁶. Gunisil Yalcin et al. found in their study that BRI was the most relevant parameter for important CAD. Moreover, BRI was more closely related to mortality during follow-up²⁷. In Maessen et al.'s study of 4,627 participants, BSI was not an appropriate parameter to determine the presence of cardiovascular disease and related risk factors²⁸. Wang Fei et al. studied the relationship between various body indices and the 10-year risk of coronary artery events in 11,247 patients and found that BRI and WHtR were the best indicators for evaluating the risk of coronary heart disease in women, with gender differences observed²⁹. Our study found that BRI was high in PCAD patients, and BRI was a good predictor of PCAD. Patients with high BRI were more likely to experience MACE events during long-term follow-up.

ABSI and BRI have recently garnered significant attention due to their association with the development of cardiovascular diseases and other adverse events. Some studies have found a stronger correlation between ABSI and premature mortality compared to body mass index or waist circumference^{15,26}. However, conflicting findings have been reported by studies conducted by Sen He, Yi Zheng, and others regarding the predictive ability of ABSI for chronic diseases and mortality rates³⁰. Maessen et al. have demonstrated that ABSI is not a suitable indicator for identifying Dutch cardiovascular diseases and risk factors²⁸. However, Wang Fei et al. which investigated the relationship between various anthropometric measures in 11,247 patients and the 10-year risk of coronary artery events, found ABSI was found to be the best indicator for assessing male coronary heart disease risk²⁶. Song et al. investigated the relationship between several anthropometric measures of obesity and cardiovascular/all-cause mortality rates in several European populations. They concluded that all anthropometric measures of abdominal obesity are positively linearly associated with cardiovascular disease-

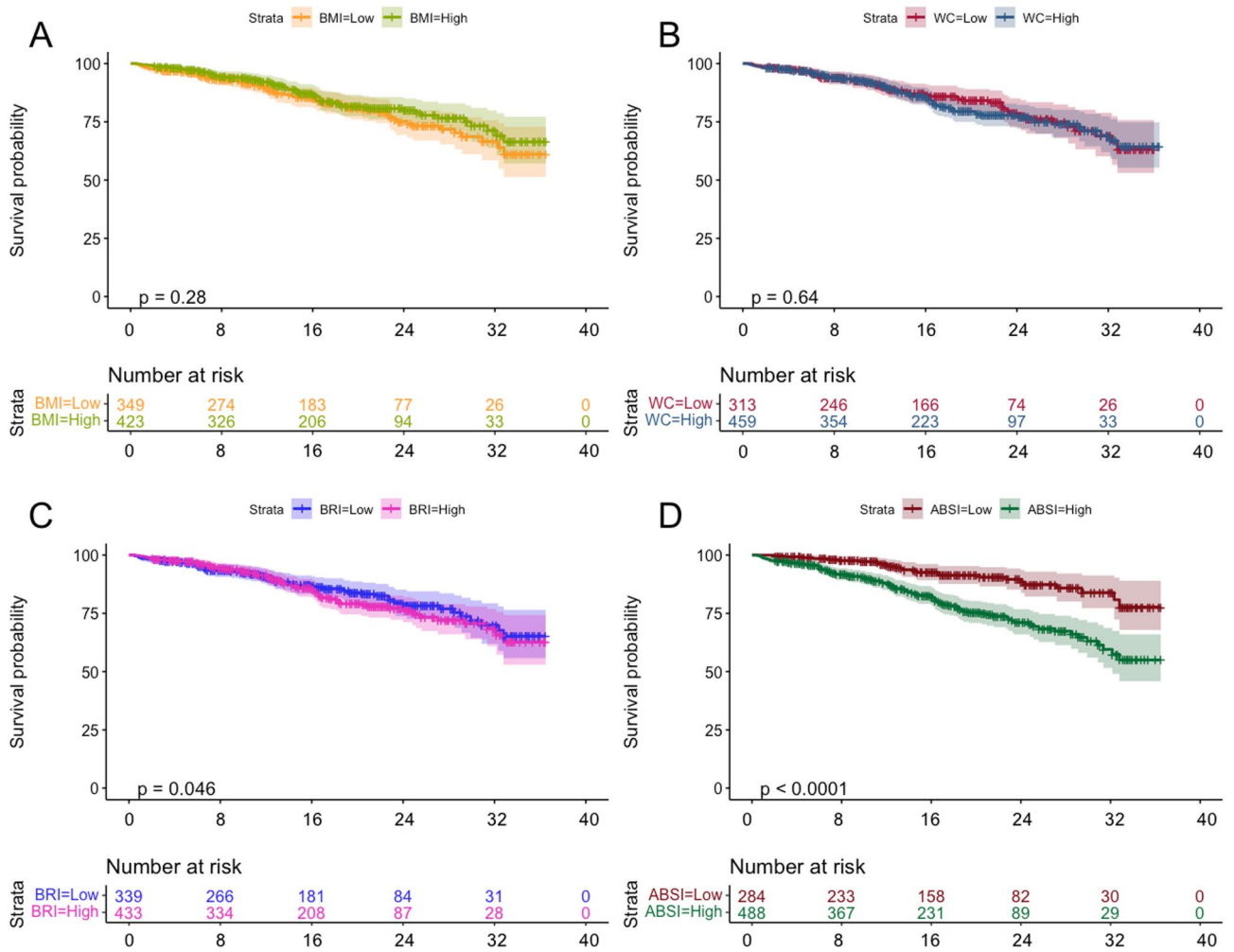


Fig. 6. After PSM matching, the survival curves show the impact of ABSI, BRI, WC, and BMI on the prognosis of PCAD patients. (A) The follow-up of patients with different BMI group for 36 months. (B) The follow-up of patients with different WC group for 36 months. (C) The follow-up of patients with different BRI group for 36 months. (D) The follow-up of patients with different ABSI group for 36 months.

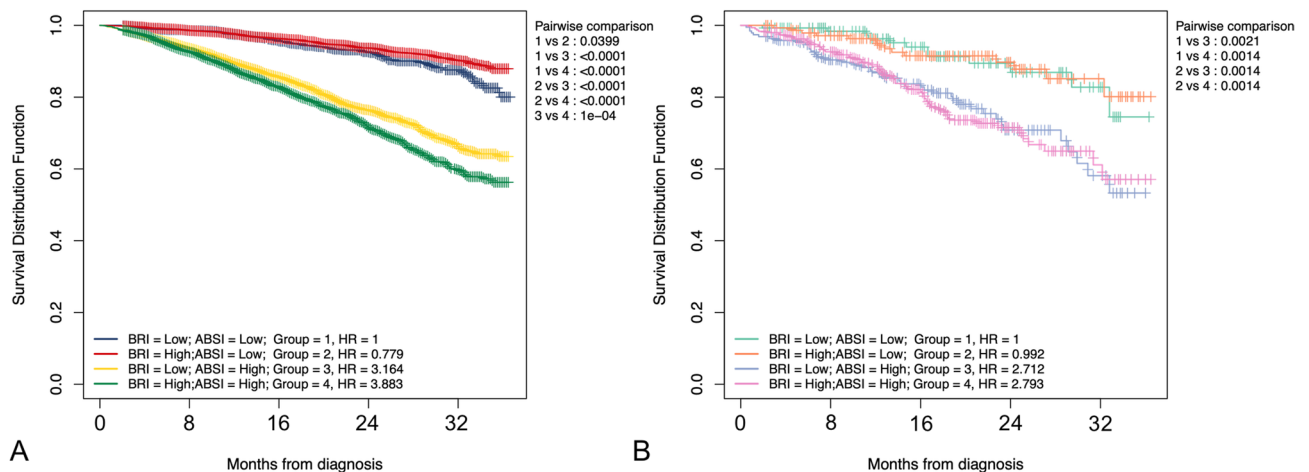


Fig. 7. Survival curves for the interaction between ABSI and BRI. (A) Before PSM matching, The follow-up of patients with different BRI + ABSI group for 36 months. (B) After PSM matching, The follow-up of patients with different BRI + ABSI group for 36 months.

related mortality, particularly ABSI³¹. In our study, ABSI not only predicts PCAD well, but also shows a stronger correlation with poor prognosis compared to BRI.

Wang Fei et al. found that BRI was a better indicator for evaluating the prognosis of female CAD patients, while ABSI was a better indicator for evaluating the prognosis of male CAD patients²⁶. Our study, through the interaction of survival curves with ABSI and BRI, found that prior to PSM matching, PCAD patients with high BRI and ABSI had the worst prognosis. However, after PSM matching, there was not much difference in prognosis between the high ABSI/high BRI group and the high ABSI/low BRI group. This suggests that after PSM matching, the impact of BRI on PCAD prognosis disappeared. This may be related to the elimination of some unknown biases through matching, such as gender, medical history, blood lipids, and other related indicators¹⁵. Further mechanisms need to be explored through more experiments.

This single-center study analyzed all enrolled participants without an external validation cohort. As a result, the ROC analyses and risk thresholds were derived and evaluated in the same sample, introducing a risk of overfitting and instability (optimism). The reversal in AUC rankings—BRI performing best before PSM, whereas ABSI ranked highest after matching—further suggests sensitivity to analytic choices and potential model instability. In Fig. 7, the hazard ratios for the ABSI–BRI interaction fluctuate, and the interaction surface indicates sparse events in several joint strata, limiting interpretability. These limitations may constrain generalizability. We plan a multicenter study with an independent validation cohort to corroborate these findings and to pre-specify and externally validate decision thresholds.

Conclusions

In this real-world study, 14,469 participants were included, comprising 387 PCAD. Propensity score matching, ROC analysis, nomogram construction, and survival analysis were applied to evaluate risk factors and outcomes. ABSI and BRI were identified as significant high-risk factors for PCAD, and their combined use showed superior diagnostic value. Moreover, elevated ABSI was associated with worse prognosis among PCAD patients. Further multicenter studies and mechanistic investigations are warranted to validate their predictive value.

Data availability

The datasets generated and/or analyzed during the current study are not publicly available due to the data belonging to the hospital database, but are available from the corresponding author on reasonable request.

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Author contributions

W.-X.S. and L.M. conceived and designed this study, participated in the experimental design, and drafted the manuscript. Y.L. and X.-Q.W. participated in patient follow-up, Y.-H.W. and Y.M. collected data, and Y.-L.L. and Y.-J.G. analyzed the data together. Y.-J.G. revised the paper. All authors read and approved the final manuscript.

Declarations

Competing interests

The authors declare no competing interests.

Ethics approval and consent to participate

The authors are accountable for all aspects of the work, ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The research design was approved by the Medical Ethics Committee of Liuzhou People's Hospital (Approval No. KY2025-009-01). All patients provided written informed consent.

Additional information

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