Abdominal fat thickness predicts the risk of pregnancy-associated hypertension: A prospective cohort study

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ABSTRACT

Background and Objectives: Pregnancy-associated hypertension (PAH) is a prevalent complication during pregnancy. Maternal obesity is a known risk factor for PAH. As nutrition is a key modifiable driver of obesity and metabolic health, this study aimed to evaluate whether early-pregnancy abdominal fat thickness and nutrient intake are associated with the development of PAH. **Methods and Study Design:** A prospective cohort study was conducted including pregnant women during 9 to 13+6 weeks of gestation. The abdominal visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT) were measured by ultrasound. Daily intake of key nutrients was assessed using a validated food frequency questionnaire. The Primary outcome was development of PAH, encompassing gestational hypertension and pre-eclampsia. **Results:** A total of 1912 pregnant women were recruited in this study. The overall incidence of PAH was 7.1% (136/1912). High VAT was associated with a higher PAH incidence than normal VAT (11.2% vs. 4.5%). Independent of BMI, high SAT (≥17.05 mm; adjusted odds ratios (aOR): 2.74, 95% confidence interval (CI): 1.89–3.96) and high VAT (≥28.85 mm; aOR: 2.42, 95% CI: 1.67–3.50) significantly increased risk of PAH. Comparatively, obesity (BMI ≥30 kg/m²) was associated with an aOR of 7.04 (95% CI: 3.94–12.6) for PAH, while the aOR for overweight was 3.16 (95% CI: 2.11–4.71). A predictive model incorporating these adiposity indicators demonstrated anarea under the ROC curve of 0.745 (95%CI: 0.703–0.788). In contrast, no significant associations were observed between daily nutrient intake and PAH risk. **Conclusions:** In this prospective cohort study, both early-pregnancy SAT and VAT are significant predictors of PAH independent of BMI. The developed predictive model facilitates the early identification of high-risk women, enabling timely interventions to reduce adverse perinatal outcomes.

Key Words: pregnancy-associated hypertension, adiposity, dietary nutrition, maternal health, visceral fat, clinical prediction models

INTRODUCTION

Pregnancy-associated hypertension (PAH) is a general classification of new hypertensive disorders after 20 weeks of gestation, including gestational hypertension (GH) and pre-eclampsia (PE).[1, 2] With a global incidence of 8%-10%, PAH stands as a predominant cause of maternal and fetal morbidity and mortality.[3, 4] Therefore, it is important to identify risk factors associated with PAH, such as obesity,[5] advanced maternal age,[6] multiple pregnancies, renal

disease,[7] pre-pregnancy diabetes and assisted reproductive technology (ART).[8] Of these, obesity is a prominent and modifiable risk factor for PAH.

In recent years, the prevalence of obesity has steadily risen globally.[9] It is defined by an excess of body mass index (BMI) in general, and is a major risk factor for PAH.[10-12] However, BMI alone is not sufficient to accurately evaluate central abdominal fat distribution and metabolic function.[13-15] Abdominal adipose tissue, comprising visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT), provides a more precise evaluation on central obesity.[16] Adipose tissue, as an endocrine and immune organ, releases signals to secret adipokines, inflammatory factors, proteins and macrophage cytokines (adiponectin, leptin, FABP4, resistin, IL-6 and TNFα) that can directly or indirectly affect hypertension, insulin resistance, dyslipidemia and high serum free fatty acid levels.[17, 18] Notably, the secretory profile and metabolic activity of adipose tissue, particularly VAT, are highly sensitive to dietary composition.

The accumulation of VAT responds dynamically to maternal nutritional intake. Diets high in saturated fats can exacerbate adipose tissue inflammation and dysregulation of adipokine secretion (e.g., decreased adiponectin, increased leptin).19 Conversely, diets rich in fiber, unsaturated fats, and essential micronutrients confer protective metabolic effects.[9] Furthermore, inadequate status of nutrients such as vitamin D and calcium has been independently associated with an adverse metabolic profile and a significantly increased risk of developing PAH.[20, 21] Thus, nutrient intake is a key modifiable factor that influences oxidative stress, systemic inflammation, and endothelial function—all central pathways implicated in the pathogenesis of PAH.[22]

CT scan is the gold standard for measuring abdominal fat thickness,[23] but is limited by its high cost and radiation exposure. Ultrasound is more suitable for pregnant women and has been proven to be safe and accurate.[24-26] Several studies have shown that abdominal fat thickness was strongly associated with an increased risk for complication during pregnancy, including gestational diabetes mellitus (GDM), newborn weight, and preterm delivery.[27-35] However, there are few studies on abdominal fat thickness and PAH.[36, 37] In addition, most existing evidence is derived from Western populations, leaving the relationship between dietary patterns, central obesity, and PAH risk in other demographics largely unexplored.

In this study, we aimed to investigate the associations of early-pregnancy maternal abdominal fat thickness and dietary factors with PAH development, and to evaluate the predictive value of this anthropometric measure. We further developed a clinical prediction

model for PAH based on abdominal fat thickness. The identification of high-risk individuals through this model could guide targeted nutritional counseling for primary prevention.

MATERIALS AND METHODS

Institutional review board statement

The study was approved by the Institutional Review Board of Qingdao Women and Children's Hospital Ethics (Number: QFELL-KY-2022-15).

Study design and participants

This prospective, observational study was conducted in Qingdao Women and Children's Hospital between Feb 24, 2023 and Dec 30, 2023. Pregnant women from 9 to 13 + 6 weeks of gestation were recruited, determined by the date of their last menstrual period, and their VAT and SAT were measured by ultrasound. The inclusion criteria were: at 9 to 13 + 6 weeks of gestation, >18 years old, planned for routine prenatal testing and give birth at the study hospital. The exclusion criteria were pre-existing hypertension before pregnancy or before 20 weeks of gestation, gynaecological diseases (e.g., cervical insufficiency, luteal insufficiency, mediastinal uterus which may lead to miscarriage), major medical conditions (e.g., heart, liver, brain, blood and other systemic diseases, congenital diseases), history of mental illness, and inability to complete follow-up as required by the programme. All women provided written informed consent.

Procedures

The baseline questionnaire was completed by trained professionals, including demographic characteristics (residence, maternal age and educational level), behavioral habits (smoking, alcohol consumption and physical activity), parity (multiparous or nulliparous), personal medical history (diabetes, hypertension, history of polycystic ovary syndrome (PCOS)), family history of related diseases (diabetes, hypertension), and pre-pregnancy weight.

Dietary intake was assessed using a semi-quantitative food frequency questionnaire (FFQ), which had been previously validated in Chinese pregnant women.[38] The validation study demonstrated acceptable reproducibility, with intra-class correlation coefficients ranging from 0.23 to 0.58 for major foods and nutrients. Relative validity against three 24-hour dietary recalls showed energy-adjusted and de-attenuated correlation coefficients ranging from 0.35 to 0.63 for most dietary components, with an average of 68% of participants classified into the same or adjacent quintiles and low extreme misclassification (<3%).

The FFQ assessed the frequency and portion size of consumption for 25 food items or food groups during the past six months. Food frequency was categorized as follows: never, less than once per week, 1-3 times per week, 4-6 times per week, once per day, 2-3 times per day, and 4 or more times per day. They also reported their use of dietary supplements, including the brand, dose, and frequency of use. The daily food consumption and nutrient intakes in the FFQ were calculated according to the China Food Composition Tables (6th edition).[39]

Physical data (height, weight, blood pressure, waist circumference (WC) and hip circumference) were measured. We calculated and classified pre-pregnancy BMI using the World Health Organization (WHO) standard (BMI equals weight (kg) divided by height (m) squared):40 obesity (≥30.0 kg/m²), overweight (25.0–29.9 kg/m²), healthy weight (18.5–24.9 kg/m²) and underweight (<18.5 kg/m²).

SAT and VAT of the subjects were obtained by trained radiographer. Measurements were taken in millimeters by placing the probe 1cm above the navel as described by Andrea et al. (2009).41 SAT was measured from the subcutaneous fat layer to the outer border of the rectus abdominus muscle at the level of the linea alba, and VAT was measured from the inner border of the rectus abdominus muscle at the level of the linea alba to the anterior wall of the abdominal aorta.41 The optimal cut-off values for SAT and VAT have been calculated using the Youden Index. All study subjects were divided into two groups, the high group and the normal group.

Outcomes definition

Gestational hypertension is defined as hypertension that develops after 20 weeks' gestation (systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg) in the absence of proteinuria and without biochemical or hematological abnormalities.[42] Pre-eclampsia is defined as gestational hypertension accompanied by any of the following: urine protein quantification ≥0.3g/24h or urine protein/creatinine ratio ≥0.3 or random urine protein (+) (when protein quantification is not available), absence of proteinuria but involves major organs (e.g., heart, lungs, liver, kidneys), abnormal changes in the hematological system, digestive system or nervous system, placental involvement.[42]

Statistical analysis

Handling of missing data: Among the 2,536 enrolled participants, 188 (7.4%) were excluded due to missing VAT or SAT measurements, and 37 were excluded due to chronic

hypertension. Follow-up continued up to 12 weeks postpartum, during which time 86 had abortions or induced abortions and 313 (12.3%) were lost to follow-up or withdrew from the study. The remaining 1,912 participants with complete outcome data and key exposure variables were included in the final analysis. For other covariates, missing data were minimal (<5%). No imputation methods were applied. All analyses were performed using R version 4.3.1 (R Foundation for Statistical Computing, Vienna, Austria). Variables with a normal distribution are expressed as mean ± standard deviation (SD), and comparisons between groups are made using two independent samples t-tests. For variables with non-normal distribution, data are expressed as median and interquartile range, and comparisons between groups are made using the Wilcoxon rank-sum test. Categorical data are expressed as the number and percentage of cases in each category, and comparisons between groups are made using the chi-squared test or Fisher's exact test. The rates of PAH according to population characteristics were calculated.

Odds ratios (ORs) and 95% CIs for PAH were estimated using logistic regression. Maternal age (continuous, years), parity (multiparous or nulliparous), method of conception (spontaneous conception or in vitro fertilization), previous history of PAH, family history of hypertension, energy intake, and sodium intake were covariates adjusted for in our analyses. We also performed subgroup analyses to determine whether other factors altered the association between VAT, SAT and PAH. Two-sided *p* values less than 0.05 were considered significant.

After screening the predictor variables for the predictive model of PAH, we constructed the model in R Studio. And we visualized the model using the nomogram. The sum of the scores for each predictor predicts the probability of PAH occurrence. Discrimination performance of the nomogram was evaluated using receiver operating characteristic curves (ROC), and the area under the curve (AUC) was calculated to quantify predictive accuracy.[43] Based on the ROC curves, the optimal cut-off values for SAT and VAT were determined to be 28.85 mm and 17.05 mm, respectively. Furthermore, to comprehensively assess the performance of the model in different aspects, we also calculated the Brier scores.44 The Plotting calibration curve is used to assess the consistency between the predicted probability of occurrence and the actual probability of occurrence. The horizontal coordinate of the calibration curve is the probability of an event predicted by the model, and the vertical coordinate is the proportion of events that actually occur within the predicted probability range. We used Decision Curve Analysis (DCA) to evaluate model performance across thresholds and identify the optimal threshold for decision-making.45The net benefit curve evaluates predictor benefits, where the

x-axis represents the high-risk threshold and cost-benefit ratio, and the y-axis indicates the number of samples classified as high-risk per 1,000 at a given threshold.[46, 47]

RESULTS

Baseline characteristics of the study population

We enrolled 2536 pregnant women at 9-13^{+6} weeks of gestation who participated in the baseline survey between Feb 24, 2023, and Dec 30, 2023. After exclusions, a total of 1,912 pregnant women with complete data on outcomes, VAT, SAT, height, and weight were included in the final analysis (Figure 1). The overall incidence of PAH was 7.1% (136/1,912), of which 73 cases developed PE. Table 1 showed the population demographics for cases and non-cases of GH and PE. The median age was 31 (28, 34) years.

Association of PAH with abdominal fat thickness

Among the 1,173 participants with normal VAT, 33 (4.5%) had PAH, whereas among the 739 participants with high VAT, 83 (11.2%) had PAH. The proportion of pregnant women with high SAT in PAH was 7.5 % higher than in normal SAT (11.8% vs.4.3%). VAT and SAT were positively associated with PAH, and high VAT and SAT were significant risk factors for PAH (Table 1).

Dietary intake of nutrients

Daily nutrient intake was assessed across the normotensive (N = 1,776), GH (N = 63), and PE (N = 73) groups. The median daily intakes of carbohydrate, protein, total fat, sugars, energy, calcium, fiber, potassium, iron, sodium, and folate for each group are shown in Table 2.

Logistic regression analysis of association between individual obesity factors and PAH

We compared common maternal adiposity measurements as predictors of PAH (Table 3). Independent of BMI, both high SAT (≥17.05 mm) and high VAT (≥28.85 mm) were significantly associated with an increased risk of PAH, with adjusted odds ratios (aOR) of 2.74 (95% CI: 1.89-3.96) and 2.42 (95% CI: 1.67-3.50), respectively. For comparison, obesity (BMI ≥30 kg/m²) was associated with an aOR of 7.04 (95% CI: 3.94-12.6) for PAH, while the aOR for overweight was 3.16 (95% CI: 2.11-4.71). Higher values of WC, hip circumference, WHR, and WHtR were also significantly associated with PAH risk.

Subgroup analysis

In the subgroup analyses based on baseline characteristics, the associations of VAT and SAT with PAH risk were similar across most subgroups (p >0.05) (Figure 2).

Constructing and validating a predictive model of PAH

To construct the model, binary logistic regression analysis was used to screen a total of 10 variables, including method of conception, previous history of PAH, family history of hypertension, WC, hip circumference, WHR, WHtR, pre-pregnancy BMI, SAT and VAT. After determining the final variables for the PAH prediction model, we constructed the model in R Studio and generated a nomogram (Figure 3A). The area under the ROC curve of the model was 0.745 (95% CI: 0.703–0.788) (Figure 3B). The Brier score was 0.060, and the absolute error of the calibration curve was 0.005 (Figure 3C). The DCA curves revealed that across a wide range of thresholds, the model containing VAT and BMI had a higher net benefit than models using a single predictor (Figure 3D). The clinical impact curve indicated that using the predictor variables provided a higher net benefit when the threshold probability was below 0.2 (Figure 3E).

DISCUSSION

In this prospective cohort study, both early-pregnancy SAT and VAT were significantly associated with an increased risk of PAH, and the associations remained significant after adjustment for confounders. And the association remained significant after adjustment for confounders. We also found that SAT and VAT were positively correlated with PE and GH. We further developed a clinically predictive PAH model-based adiposity measurements in early pregnancy.

Previous evidence on the relationship between SAT and VAT with PAH is limited. Two studies examined the association between SAT or VAT and PAH and consistently reported a positive association.[27, 37] This is consistent with our findings. In the study by Nassr et al, most women developed gestational hypertension rather than pre-eclampsia, and the pathophysiology and clinical outcomes of the two conditions are partly different.[37] In our findings, the incidence of PE was similar to that of GH, which may be related to the geographical area where the study was conducted and the different diets. This observation underscores the potential role of modifiable factors like diet, as specific dietary patterns are known to influence systemic inflammation and metabolic health, which underlie both fat distribution and PAH risk.[48] Extending this perspective to specific nutrients, maternal iron

status has also been associated with the risk of pre-eclampsia in observational studies, further highlighting the potential link between nutritional profiles and PAH pathogenesis.[49] Another study recruited 1510 pregnant women, and only measured SAT.[27] A study of 463 pregnant women showed a negative correlation between VAT and PE in unadjusted models.[33] Our results were consistent with the previous report by Maack et al. that included 3777 pregnant women, showing that SAT thickness and VAT thickness are BMI-independent predictors of PE. Their findings are consistent with our study, but the study was conducted at 17-19 weeks of gestation.[36] In addition, Mendelian regression analysis suggested that VAT was positively associated with PE.[50] We provided the first comprehensive review of the association between SAT and VAT with PAH based on the results from the Chinese population, and developed a clinically predictive PAH model based on adiposity measurements in early pregnancy.

Obesity and inflammation are highly integrated processes in the pathogenesis of insulin resistance, diabetes mellitus, hypertension and nonalcoholic fatty liver disease (NAFLD).[51, 52] It has been extensively demonstrated that BMI⩾30kg/m2 is associated with an increased risk of developing PAH.[10] However, the prognostic value of BMI is limited by the fact that it does not distinguish excess fat, muscle or bone mass, thus SAT and VAT appear to be more sensitive than BMI to individual metabolic and cardiovascular comorbidities caused by obesity. SAT is characterized by a higher number of small adipocytes and higher insulin sensitivity. In contrast, VAT is characterized by a higher number of large adipocytes.18 The main differences between the two types of adipose tissue include their endocrine function and lipolytic responses to insulin and other hormones. Visceral adipocytes have a higher lipolytic activity and VAT releases free fatty acids (FFA) more rapidly than subcutaneous fat, which is observed in both obese and lean patients.[51] Excess visceral adipose tissue leads to the development of chronic inflammation, and the release of adipocytokines from visceral and subcutaneous adipose tissue into the bloodstream affects the metabolism involved in many metabolic processes.[53] Importantly, the pro-inflammatory state driven by visceral adiposity is modifiable. Nutritional intake is a key regulator, with Mediterranean or similar anti-inflammatory dietary patterns being associated with lower levels of systemic inflammation and a reduced risk of adverse pregnancy outcomes, including hypertensive disorders.54 However, this study suggested that PAH was better predicted using pre-pregnancy BMI than SAT or VAT measurements, and SAT was better than VAT, contrary to our expectations. This is consistent with the results of an earlier article.36 The exact reason for this needs to be investigated further.

The prediction and prevention of PAH are currently the focus of research and clinical attention. In particular, early detection of pregnant women at high risk of pre-eclampsia and early treatment with aspirin may be effective in reducing maternal and fetal complications. The American College of Obstetricians and Gynecologists (ACOG) recommends that women at high risk for pre-eclampsia start taking 60 to 80 mg of aspirin late in the first trimester.[42] The main challenge is to determine which women will benefit from aspirin therapy without overtreatment. It is therefore important to predict the diagnosis of pre-eclampsia in women at high risk before the end of the first trimester. Our study developed a clinical predictive model based on SAT and VAT, combined with other measurements of adiposity in early pregnancy, which helps identify high-risk pregnant women. The early risk stratification enabled by such a model creates a critical window of opportunity for implementing adjunctive preventive strategies. Among these, targeted nutritional counseling to promote diets that mitigate inflammation and improve metabolic health represents a promising and safe approach to potentially reduce PAH risk.[55]

The strengths of our study include the analysis of a large prospective cohort and the establishment of a clinical predictive model for PAH based on early-pregnancy adiposity measurements. However, several limitations should be considered. First, the relatively low incidence of PAH precluded further differentiation between early- and late-onset PE. Second, this was a single-center study conducted in China, which may limit the generalizability of our findings to other populations or clinical settings. Third, dietary intake was assessed using a food frequency questionnaire, which is subject to recall bias and measurement error, despite prior validation in pregnant populations. Therefore, while our model demonstrates good internal validity, its external applicability requires further validation. Future research should focus on multicenter prospective studies with diverse populations to confirm the robustness of our model and the observed associations.

Clinical implications of the predictive model

Our predictive model achieved an AUC of 0.745, indicating acceptable discriminative ability. This level of performance is comparable to other established obstetric prediction models, such as those for preeclampsia (AUC:0.75 [IQR 0.69-0.78]) and gestational diabetes (AUC: 0.71[IQR0.67-0.76]) reported in a recent systematic review,[56] as well as the externally validated full PIERS model (AUC: 0.742).[57] While not sufficiently accurate for standalone diagnostic use, the model may serve as a valuable risk stratification tool in clinical practice.

In terms of clinical integration, the model can be implemented during routine first-trimester ultrasound examination (9-13^{+6} weeks), without requiring additional equipment. Early

identification of high-risk women could facilitate timely implementation of established preventive interventions, such as low-dose aspirin for preeclampsia prevention as recommended by clinical guidelines.[56, 58] Cost-effectiveness analyses have shown that such screening strategies, combined with targeted aspirin prophylaxis, are cost-saving.[59, 60] The model is intended to complement, rather than replace, existing clinical assessment methods.[61] Future research should focus on external validation in diverse populations and explore incorporating additional predictors to improve performance.

Conclusion

In conclusion, this study demonstrated a significant association between SAT and VAT with PAH. Furthermore, we developed a clinical predictive PAH model. Our findings could allow clinicians to predict the occurrence of PAH in pregnant women as early as possible and implement timely interventions, among which personalized nutritional strategies aimed at optimizing metabolic health hold promise for reducing adverse perinatal outcomes.

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CONFLICT OF INTEREST AND FUNDING DISCLOSURE

The authors declare no conflict of interest.

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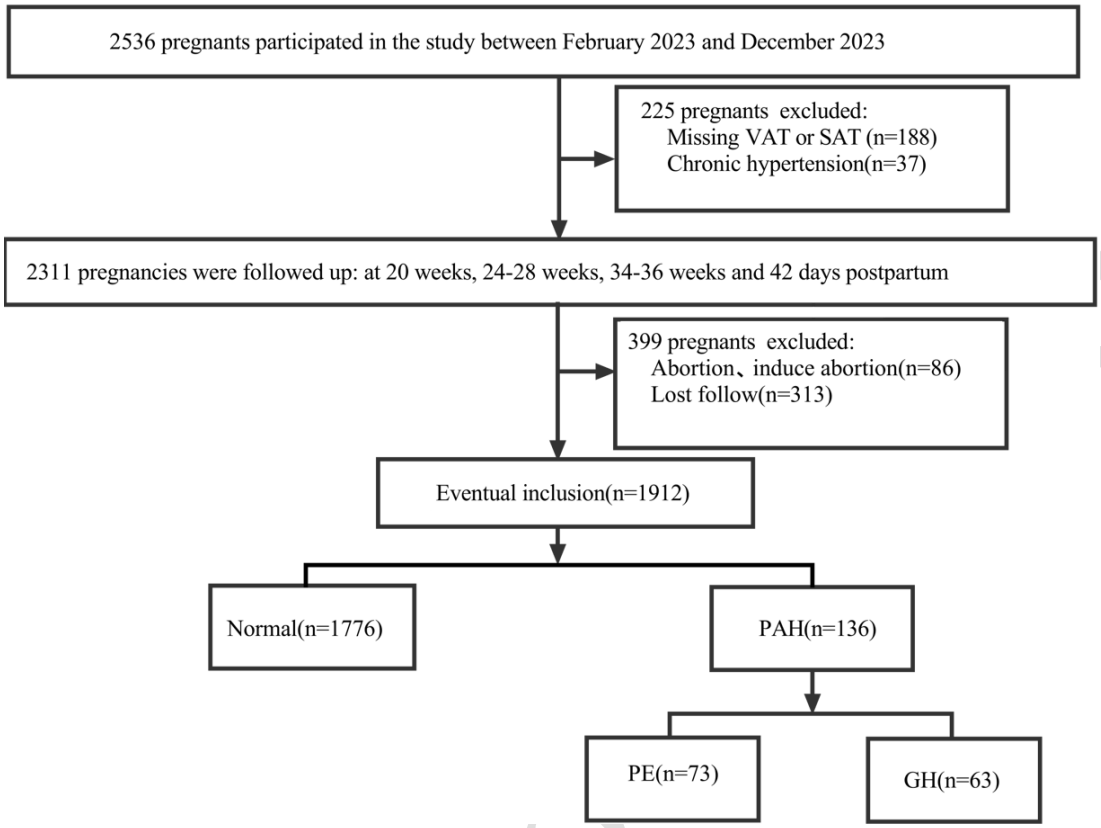

Figure 1. Study cohort. PAH: pregnancy-associated hypertension, PE: pre-eclampsia, GH: gestational hypertension.

A

Variable	Count	Percent(%)	OR(95%CI)	P value	P for interaction
parity					0.26
multipara	982	51.4	3.72 (2.15 , 6.42)	<0.001	
primipara	930	48.6	2.45 (1.51 , 3.96)	<0.001	
age					0.422
<35	1540	80.5	2.76 (1.85 , 4.12)	<0.001	
≥35	372	19.5	4.04 (1.74 , 9.39)	0.001	
Previous.history.of.PAH					0.952
No	1896	99.2	2.92 (2.02 , 4.23)	<0.001	
Yes	16	0.8	3.12 (0.38 , 25.57)	0.288	
Family.history.of.hypertension					0.357
No	1142	59.7	2.47 (1.49 , 4.11)	<0.001	
Yes	770	40.3	3.48 (2.07 , 5.86)	<0.001	
Conception					0.356
Natural	1647	86.1	3.04 (2.02 , 4.57)	<0.001	
Assisted	265	13.9	2.00 (0.91 , 4.40)	0.083	
WHR1					0.387
<0.85	1257	65.7	2.43 (1.51 , 3.92)	<0.001	
≥0.85	655	34.3	3.45 (1.84 , 6.49)	<0.001	
WHtR1					0.228
<0.5	1129	59	1.50 (0.76 , 2.95)	0.244	
≥0.5	783	41	2.55 (1.49 , 4.36)	0.001	
Overall	1912	100	2.98 (2.08 , 4.27)	<0.001	

B

Variable	Count	Percent(%)	OR(95%CI)	P value	P for interaction
parity					0.697
multipara	982	51.4	2.49 (1.47 , 4.24)	0.001	
primipara	930	48.6	2.87 (1.77 , 4.67)	<0.001	
age					0.588
<35	1540	80.5	2.81 (1.88 , 4.21)	<0.001	
≥35	372	19.5	2.21 (1.02 , 4.77)	0.044	
Previous.history.of.PAH					0.77
No	1896	99.2	2.55 (1.77 , 3.67)	<0.001	
Yes	16	0.8	1.71 (0.12 , 23.94)	0.689	
Family.history.of.hypertension					0.071
No	1142	59.7	1.90 (1.15 , 3.14)	0.012	
Yes	770	40.3	3.71 (2.20 , 6.28)	<0.001	
Conception					0.074
Natural	1647	86.1	3.03 (2.01 , 4.58)	<0.001	
Assisted	265	13.9	1.40 (0.67 , 2.93)	0.366	
WHR1					0.034
<0.85	1257	65.7	1.78 (1.10 , 2.88)	0.02	
≥0.85	655	34.3	4.44 (2.22 , 8.90)	<0.001	
WHtR1					0.796
<0.5	1129	59	1.71 (0.90 , 3.28)	0.103	
≥0.5	783	41	1.91 (1.15 , 3.16)	0.012	
Overall	1912	100	2.67 (1.87 , 3.82)	<0.001	

Figure 2. (A) Effect of SAT on PAH, by other subgroups. (B) Effect of VAT on PAH, by other subgroups

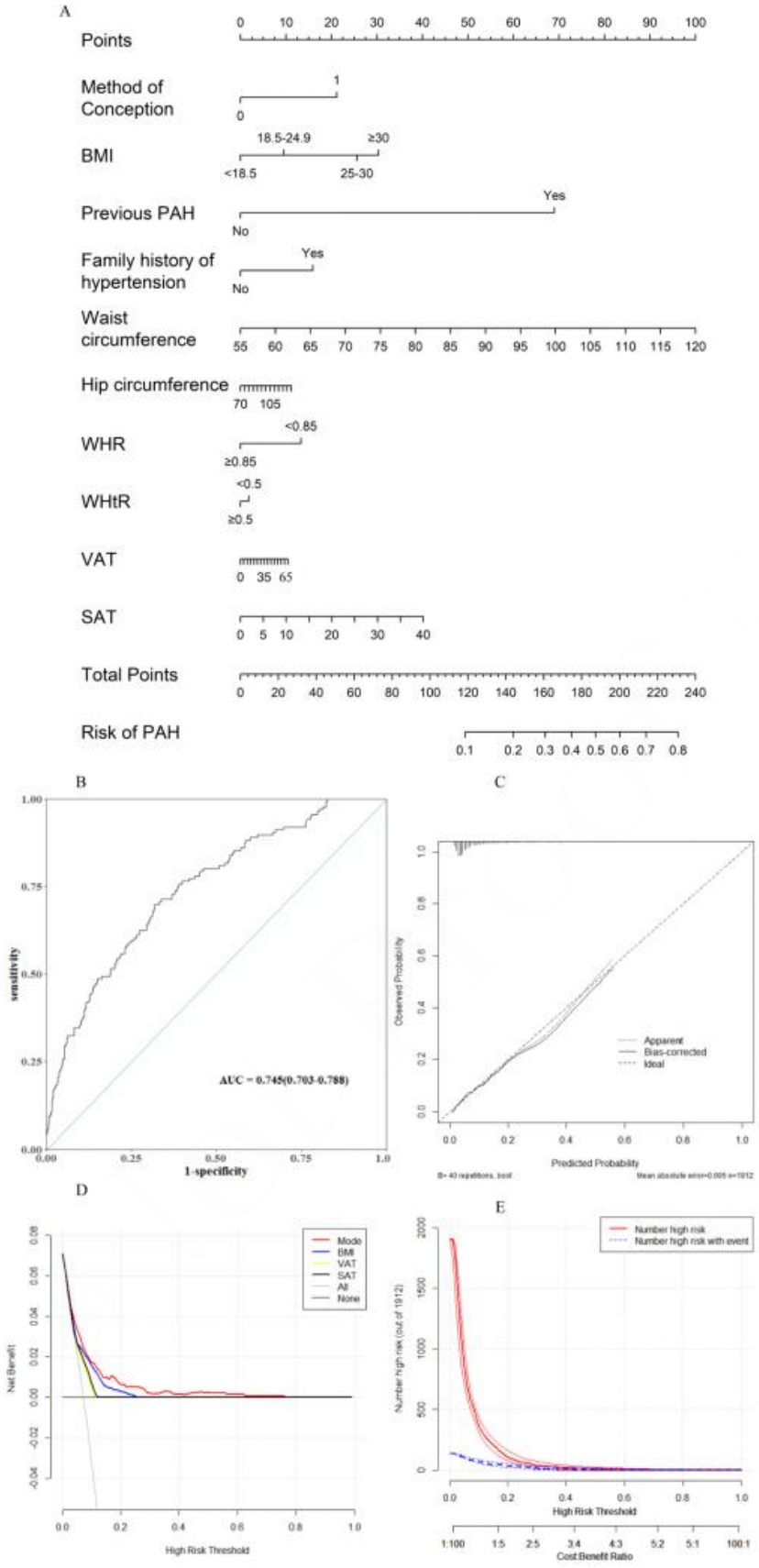

Figure 3. The PAH predictive model for pregnant women in early pregnancy. (A) nomogram: the sum of the scores for each predictor, which predicts the probability of PAH (B) ROC plot of the model (C) Calibration curves (D) DCA char (E) Net Benefit Curve

Table 1. Baseline characteristics of the study population by PAH status

Variable	Overall (N = 1,912)	Normal (N = 1,776)	GH (N = 63)	PE (N = 73)	*p*-value
Age, years, median (IQR)	31.00 (28.00-34.00)	31.00(28.00-34.00)	32.00 (27.00-36.00)	32.00 (28.00-34.00)	0.537
Age ≥35, years, n (%)	372 (19.5)	343 (19.3)	13 (20.6)	16 (21.9)	0.835
Rural, n (%)	84 (4.4)	72 (4.1)	6 (9.5)	6 (8.2)	0.028
In vitro fertilization, n (%)	265 (13.9)	232 (13.1)	10 (15.9)	23 (31.5)	<0.001
Nullipara, n (%)	930 (48.6)	855 (48.1)	34 (54.0)	41 (56.2)	0.280
College /above, n (%)	1,696 (88.7)	1,558 (87.7)	52 (82.5)	56 (76.7)	0.324
Previous of PAH, n (%)	16 (0.8)	9 (0.5)	2 (3.2)	5 (6.8)	<0.001
Family history of hypertension, n (%)	770 (40.3)	699 (39.4)	30 (47.6)	41 (56.2)	0.008
Smoke exposure, n (%)	118 (6.2)	111 (6.3)	3 (4.8)	4 (5.5)	0.999
PCOS, n (%)	213 (11.1)	191 (10.8)	11 (17.5)	11 (15.1)	0.139
Waist circumference, cm, median (IQR)	79.00(74.00-86.00)	79.00(73.20-85.00)	88.00 (79.00-94.25)	85.00 (78.20-93.00)	<0.001
Hip circumference, cm, median (IQR)	96.00(91.50-101.00)	95.90(91.20-100.13)	102.00 (96.75-107.30)	100.00 (95.30-105.00)	<0.001
WHR≥0.85, n (%)	655 (34.3)	592 (33.3)	30 (47.6)	33 (45.2)	0.008
WHTR≥0.5, n (%)	783 (41.0)	692 (39.0)	40 (63.5)	51 (69.9)	<0.001
BMI, kg/m^2, median (IQR)	21.98 (20.08-24.47)	21.79 (20.03-24.17)	25.64 (22.33-28.97)	24.84 (21.48-28.04)	<0.001
BMI, n (%)					<0.001
18.5-24.9 kg/m^2	1,326 (69.4)	1,263 (71.1)	26 (41.3)	37 (50.7)	
<18.5 kg/m^2	163 (8.5)	160 (9.0)	1 (1.6)	2 (2.7)	
25-29.9 kg/m^2	346 (18.1)	296 (16.7)	28 (44.4)	22 (30.1)	
≥30 kg/m^2	77 (4.0)	57 (3.2)	8 (12.7)	12 (16.4)	
SAT ≥17.05mm, n (%)	722 (37.8)	637 (35.9)	36 (57.1)	49 (67.1)	<0.001
VAT ≥28.85mm, n (%)	739 (38.7)	656 (36.9)	41 (65.1)	42 (57.5)	<0.001
VAT/SAT Radios, median (IQR)	1.67 (1.32-2.05)	1.67 (1.31-2.04)	1.71 (1.38-2.07)	1.71 (1.50-2.05)	0.629

PAH: pregnancy-associated hypertension, PE: pre-eclampsia, GH: gestational hypertension. GDM: gestational diabetes mellitus. PCOS: polycystic ovary syndrome.UA: uric acid. CREA: creatinine. MPV: mean platelet volume. PCT: thrombocytocrit. PDW: platelet volume distribution width. NEU: neutrophils. LYM: lymphocyte.
Data are presented as median (IQR) for continuous variables, or n (column percentage) for categorical variables. p-values were calculated using Kruskal-Wallis test (continuous) or chi-squared test (categorical).

Table 2. Daily nutrient intake by PAH status

Variable	Overall (N =1,912)	Normal (N =1,776)	GH (N=63)	PE (N = 73)
Carbohydrate, g/d, Median (IQR)	125.10 (89.99- 162.66)	124.90 (89.63-163.62)	127.23 (92.60-158.18)	132.80 (99.08-160.23)
Protein, g/d, Median (IQR)	43.85 (35.01-54.39)	43.85 (35.01- 54.07)	42.40 (32.89- 56.01)	48.71 (38.01- 66.52)
Total Fat, g/d	25.84 (19.63-34.18)	25.83 (19.65-33.96)	24.48 (18.59-36.55)	28.74 (20.14-39.08)
Sugars,g/d, Median (IQR)	114.37 (81.45-150.48)	113.81 (81.08-150.75)	116.73 (79.76-148.96)	117.52 (89.40-147.47)
Energy, kcal/d, Median (IQR)	1,105.07 (839.65- 1,417.46)	1,103.85 (839.35– 1,414.70)	1,105.01 (806.77-1,448.18)	1,167.04 (875.69-1,562.27)
Calcium, mg/d, Median (IQR)	455.26 (315.29 -572.52)	454.86 (315.29- 570.66)	450.53 (307.07-581.37)	464.02 (339.55-579.96)
Dietary Fiber, g/d, Median (IQR)	8.86 (6.48-11.46)	8.90 (6.44-11.46)	8.47 (6.68- 11.74)	8.73 (7.03-11.79)
Iron, mg/d, Median (IQR)	22.78 (15.61-29.49)	22.90 (15.67-29.52)	21.25 (14.75-28.23)	24.14 (15.52- 36.03)
Potassium, mg/d, Median (IQR)	1,480.97 (1,147.58- 1,877.01)	1,478.18 (1,137.73- 1,876.91)	1,359.04 (1,138.69-1,843.65)	1,550.75 (1,239.64 -1,931.88)
Sodium, mg/d, Median (IQR)	664.86 (486.80 - 875.68)	667.32 (486.16- 876.75)	638.67 (459.57- 819.44)	637.11 (520.66- 893.43)
Dietary Folate, µg/d, Median (IQR)	228.21 (173.74- 296.44)	227.27 (173.74-293.39)	243.72 (164.35- 304.36)	255.70 (191.41-320.20)

PAH: pregnancy-associated hypertension, PE: pre-eclampsia, GH: gestational hypertension
Data are presented as median (IQR).

Table 3. Binary logistic regression analysis of factors associated with PAH

Variable	Crude OR (95% CI)	*p*-value	Adjusted† OR (95% CI)	*p*-value
WHR				
<0.85	ref		ref	
≥0.85	1.73 (1.21-2.45)	0.002	1.66 (1.16-2.38)	0.006
WHtR				
<0.5	ref		ref	
≥0.5	3.17 (2.19-4.58)	<0.001	2.98(2.03-4.36)	<0.001
WC, cm	1.08 (1.06-1.10)	<0.001	1.08 (1.06-1.10)	<0.001
Hip, cm	1.09 (1.07-1.12)	<0.001	1.09 (1.07-1.12)	<0.001
BMI, kg/m^2				
18.5-25	ref		ref	
<18.5	0.38 (0.12-1.21)	0.101	0.42 (0.13-1.38)	0.153
25-30	3.39 (2.29-5.01)	<0.001	3.16 (2.11-4.71)	<0.001
≥30	7.03(3.98-12.42)	<0.001	7.04 (3.94-12.60)	<0.001
SAT, mm				
<17.05	ref		ref	
≥17.05	2.98(2.08-4.27)	<0.001	2.74 (1.89-3.96)	<0.001
VAT, mm				
<28.85	ref		ref	
≥28.85	2.67 (1.87-3.82)	<0.001	2.42 (1.67-3.50)	<0.001
VAT/SAT ratio	1.02 (0.79-1.32)	0.859		
Dietary folate, μg/d				
<228.20	ref		ref	
≥228.20	1.59(1.12-2.26)	0.009	1.32(0.89-1.95)	0.164

PAH: pregnancy-associated hypertension; VAT, visceral adipose tissue; SAT, subcutaneous adipose tissue; sodium and energy
†Models adjusted for age, parity, conception, previous history of PAH, family history of hypertension.

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