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ORIGINAL RESEARCH

Development and Validation of a Nomogram for Predicting Hyperuricemia in Perimenopausal Women

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Objective: To develop and validate a nomogram model for predicting the risk of hyperuricemia (HUA) in perimenopausal women.
Methods: In this study, physical examination information of perimenopausal women was collected at the First Affiliated Hospital of University of Science and Technology of China. We utilized the Least Absolute Shrinkage and Selection Operator (Lasso) and binary logistic regression to investigate the risk factors of HUA among perimenopausal women.
Results: We finally collected 5637 patients in this study. Based on the results of Lasso-logistic regression analysis, we incorporated ten different independent variables into the risk prediction model for HUA. The risk prediction model showed good discrimination ability in both the training set (AUC=0.819; 95% CI=0.801~0.838) and validation set (AUC=0.787; 95% CI=0.756~0.818), the calibration curve demonstrates that the model was well-calibrated. In addition, we constructed HUA risk prediction models for perimenopausal women with BMI < 25.0 and BMI ≥ 25.0, respectively. The AUC of the prediction model in the population with BMI < 25.0 was 0.793, and the AUC of the prediction model in the population with BMI ≥ 25.0 was 0.765.
Conclusion: Our study identified several independent risk factors for HUA in perimenopausal women and developed a prediction mode, which might be used to detect the individual conditions and implement the preventive interventions.
Keywords: perimenopausal women, hyperuricemia, prediction

Background

Serum uric acid (SUA), which results from purine metabolism, is normally filtered by the kidneys and excreted in urine.[1] However, hyperuricemia (HUA) occurs when SUA production exceeds renal excretion capacity or when impaired kidney clearance reduces uric acid output. These mechanisms elevate serum SUA concentrations.[2] HUA is closely related to gout, and asymptomatic HUA can lead to the deposition of urate in the joints of patients and even bone erosion. Studies had found that asymptomatic HUA leading to gout was a continuous pathological process.[3] Epidemiological evidence has demonstrated that HUA is both a critical contributor to gout pathogenesis and significantly associated with malignancies. According to a survey conducted in Chinese population, the overall prevalence of HUA among adults was 11.1% during 2015–2019, while the prevalence rate had risen to 14% by 2019.[4] Additionally, a national health and nutrition survey conducted in the United States showed that the risk of death from HUA was similar to that of diabetes.[5] HUA had become a serious risk factor for public health that could not be ignored.

The Framingham Heart Study indicated that SUA levels in men remain stable after puberty, whereas the SUA levels gradually increase after middle age in women.[6] During perimenopause, declining ovarian function reduces estrogen levels. This impairs uric acid excretion, consequently elevating SUA concentrations.[7] According to epidemiological studies, the prevalence of HUA increases with age. One study found that the prevalence of HUA among women was around 11%.[8] A Meta-analysis indicated that the prevalence of HUA among Chinese perimenopausal

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women reached 13%, and it continued to rise as women enter menopause.[9] This could potentially lead to various health issues in this demographic. Additionally, the hormonal changes make women more prone to obesity and metabolic syndrome during perimenopause, which could further exacerbate elevated SUA levels and consequently contribute to HUA.[10]

Previous studies showed that HUA was closely linked to the development and progression of cardiovascular disease, diabetes, and other diseases.[11] Therefore, perimenopausal women should pay more attention to the changes in serum uric acid levels and take some measures to prevent HUA. According to one study, 88% of women experience menopause at a mean age of 51.4 years, with over 800 million women worldwide currently in this life stage.[12] Therefore, predicting the prevalence of HUA in this group is crucial for their sustainable health management.

Currently, there were relatively few reports on the risk factors and predictive models for HUA in perimenopausal women. Eljaaly et al found that HUA was linked to SCR, high-density lipoprotein, triglycerides, hip circumference, total cholesterol.[13] Zhang et al explored the risk factors for elevated serum uric acid (SUA) levels in elderly individuals and constructed a predictive model, which identified a SUA level ≥ 360 μmol/L as a common risk factor for both men and women.[14] Therefore to advance clinical practice for perimenopausal women's health promotion, we aimed to identify risk factors for HUA in perimenopausal women and to construct and validate a nomogram model for clinical risk prediction.

Methods

Study Population

The study was conducted in accordance with the provisions of the Declaration of Helsinki, and approval was granted by the First Affiliated Hospital of the University of Science and Technology of China (2025-RE-191). Informed consent was also obtained from all patients prior to the study. We collected clinical data from perimenopausal women who had completed standardized health evaluations at USTC First Affiliated Hospital's Health Management Center.

The determination of HUA was based on a reasonable standard: the SUA level was equal to more than 360 mmol/L.[15] The exclusion criteria included: (1) younger than 45 years old or older than 55 years old; (2) duplicated clinical data; (3) Severe hepatic and renal dysfunction; (4) currently suffering from malignant tumors.

We collected information from 6225 physical examinees. After excluding 512 records with missing medical data and 28 duplicate records, we retained records for 5685 examinees. Further excluding 46 individuals with abnormal liver and kidney function and 2 patients with malignant tumors, a total of 5637 examinees met the inclusion criteria. In this study, we collected a total of 28 different variables, according to the 10 EPV (Events Per Variable) rule,[16] the number of positive samples should exceed 270. With a total of 733 positive samples among all study subjects, the sample size meets the requirement for model development. These were randomly divided into a model training set of 3945 individuals and a validation set of 1692 individuals. The flowchart is shown in Figure 1.

Data Collection

The data for this study were sourced from the participants' medical records, including the following information: (1) Demographic characteristics – Age; Gender; Body Mass Index (BMI), (2) Relevant medical history - HUA, fatty liver, (3) Renal function tests: Blood Urea Nitrogen (BUN); Serum Creatinine (SCR), (4) Liver function tests: Alanine Aminotransferase (ALT); Aspartate Aminotransferase (AST); Alkaline Phosphatase (ALP); Total Protein (TP); Albumin (ALB); Globulin (GLB), (5) Random blood glucose (GLU), (6) Blood lipid tests: High-density lipoprotein (HDL); Low-density lipoprotein (LDL); Very Low-Density Lipoprotein (VLDL); Total Cholesterol (TC); Triglyceride (TG), (7) Blood cell tests: White Blood Cell Count (WBC); Red Blood Cell Count (RBC); Hemoglobin (HGB); Platelet count (PLT); Eosinophils percentage (Eos%); Basophils percentage (Baso%); Lymphocyte Percentage (LY%); Neutrophil percentage (NEUT%), (8) Routine Urine index: Urine pH (UPH); Urine Specific Gravity (SG).

Statistical Analysis

Statistical analysis was performed via R 4.4.2 and SPSS 26.0. For Gaussian distribution was used to represent the data, for non-normally distributed data, the median and interquartile range (IQR) were used for representation; For categorical variables, we

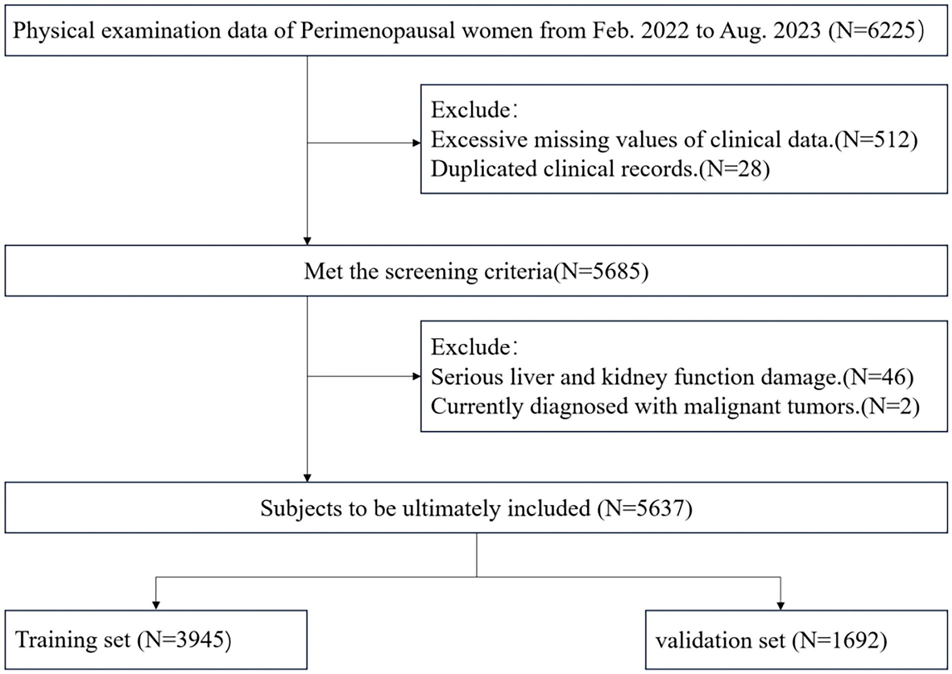

Figure 1 The process of determining research subjects.

reported frequencies and proportions. The entire study population was random divided into a model training set and a model validation set at a ratio of 7:3. Continuous variables were analyzed using Student's *t*-test (for normally distributed data) or the Mann–Whitney *U*-test (for nonparametric data), while categorical variables were assessed with the chi-square test. Since the study mainly selected hematological examination results as independent variables, which had strong collinearity, we used the least absolute shrinkage and selection operator (Lasso)[17] and binary logistic regression were employed for feature variable screening, and employed Decision Curve Analysis (DCA) to evaluate the maximum net benefit of the prediction model. To visualize the prediction model, we presented it using a nomogram and evaluated its performance through Receiver Operating Characteristic (ROC) analysis and the Area Under the Curve (AUC). All analyses used two-tailed tests, with statistical significance set at $P < 0.05$. We utilized R packages "glmnet" and "rms" to perform Lasso regression and construct a nomogram model.

Result

Baseline Characteristics

In the group of 5637 perimenopausal women who met the research criteria, a total of 733 individuals suffered from HUA, the prevalence rate of HUA among all participants was 13%. There were statistically significant differences between non-HUA group and HUA group in terms of AGE, BMI, renal function (BUN, SCR), liver function (ALT, AST, ALP, TP, ALB, GLB), blood lipid (HDL, TC, VLDL, LDL, TG), blood cell (WBC, RBC, HGB, PLT, Eos), fasting blood glucose, and medical history (hypertension, fatty liver) ($P< 0.05$) (Table 1).

The training set comprised 3945 participants, while the validation set included 1692 participants. No statistically significant differences were observed in baseline characteristics or clinical features between the two groups (Table 2).

The Related Risk Factors of HUA

Variable selection was performed on the training data through Lasso regression coupled with tenfold cross-validation (Figure 2), where the final model was chosen based on the one standard error criterion, the λ was taken. Finally, thirteen indicators were screened, including BMI, UPH, ALP, WBC, SCR, HDL, LDL, HGB, ALT, TP, TG, hypertension, fatty liver. Subsequently, the above indicators were included in the binary logistic regression analysis, and the results were

Table 1 Baseline Characteristics of HUA Group and Non-HUA Group

Variables	Non-HUA Group (4904)	HUA Group (733)	*P*
AGE	49.92±3.09	50.62±2.99	<0.001
BMI	22.80 (21.20, 24.60)	24.65 (22.77, 27.10)	<0.001
Blood glucose index			
GLU	5.04 (4.77, 5.371)	5.23 (4.89, 5.621)	<0.001
Blood lipid index			
HDL	1.31 (1.12, 1.54)	1.14 (0.98, 1.341)	<0.001
LDL	2.82 (2.36, 3.31)	3 13 (2.61, 3.66)	<0.001
VLDL	0.80 (0.64, 1.01)	0.87 (0.67, 1.12)	<0.001
TC	5.01 (4.48, 5.57)	5.19 (4.65, 5.83)	<0.001
TG	1.09 (0.82, 1.50)	1.56 (1.14, 2.13)	<0.001
Liver function index			
ALT	17.00 (14.00, 23.00)	22.00 (17.00, 32.00)	<0.001
AST	22.00 (19.00, 26.00)	24.00 (21.00, 28.00)	<0.001
ALP	70.10±19.19	77.79±20.00	<0.001
TP	74.30 (71.80, 76.90)	75.50 (73.00, 78.20)	<0.001
ALB	45.40 (43.80, 46.90)	46.00 (44.50, 47.50)	<0.001
GLB	28.80 (26.90, 30.90)	29.50 (27.50, 31.60)	<0.001
Renal function index			
BUN	4.95 (4.21, 5.78)	5.29 (4.56, 6.12)	<0.001
SCR	53.00 (48.00, 58.00)	57.00 (52.00, 63.00)	<0.001
Blood cell content			
WBC	5.25 (4.49, 6.17)	5.85 (4.94, 6.88)	<0.001
RBC	4.39 (4.17, 4.62)	4.51 (4.29, 4.74)	<0.001
HGB	130.00 (124.00, 137.00)	134.00 (127.00, 140.00)	<0.001
PLT	233.00 (198.00, 274.00)	244.00 (208.00, 285.00)	<0.001
Eos	1.70 (1.00, 2.60)	1.80 (1.20, 2.80)	<0.001
Baso	0.50 (0.40, 0.70)	0.50 (0.40, 0.70)	0.822
LY	33.75±7.67	33.33±7.55	0.167
NEUT	56.66±8.25	57.19±7.97	0.101
Routine Urine index			
UPH	6.14±0.42	6.03±0.41	<0.001
SG	1.02±0.01	1.02±0.01	0.001
Disease history			
Hypertension (Yes)	152 (3.1)	64 (8.7)	<0.001
Fatty liver (Yes)	1035 (21.1)	430 (58.7)	<0.001

Notes: Data are shown as mean ± standard deviation, median (interquartile range), or frequency (percentage).

Table 2 Baseline Characteristics of Training Set and Validation Set

Variables	Training Set (3945)	Validation Set (1692)	*P*
AGE	50.03±3.08	49.96±3.10	0.479
BMI	23.00 (21.36, 24.90)	23.00 (21.32, 24.80)	0.927
Blood glucose index			
GLU	5.07 (4.78, 5.42)	5.05 (4.79, 5.371)	0.421
Blood lipid index			
HDL	1.28 (1.09, 1.51)	1.29 (1.10, 1.52)	0.514
LDL	2.84 (2.38, 3.36)	2.87 (2.39, 3.35)	0.341
VLDL	0.81 (0.65, 1.02)	0.80 (0.64, 1.02)	0.630
TC	5.02 (4.48, 5.60)	5.03 (4.52, 5.61)	0.538
TG	1.14 (0.84, 1.59)	1.13 (0.83, 1.58)	0.876

(*Continued*)

Table 2 (Continued).

Variables	Training Set (3945)	Validation Set (1692)	*P*
Liver function index			
ALT	18.00 (14.00, 24.00)	18.00 (14.00, 25.00)	0.821
AST	22.00 (19.00, 26.00)	22.00 (19.00, 26.00)	0.635
ALP	71.33±19.76	70.56±18.76	0.172
TP	74.40 (71.80, 77.10)	74.60 (72.10, 77.20)	0.135
ALB	45.40 (43.90, 46.90)	45.50 (44.00, 47.10)	0.156
GLB	28.90 (27.00, 30.90)	29.00 (26.90, 31.10)	0.363
Renal function index			
BUN	5.00 (4.25, 5.83)	5.00 (4.24, 5.81)	0.536
SCR	54.00 (49.00, 59.00)	54.00 (48.00, 59.00)	0.218
Blood cell content			
WBC	5.34 (4.56, 6.24)	5.30 (4.49, 6.25)	0.484
RBC	4.40 (4.18, 4.63)	4.41 (4.20, 4.65)	0.233
HGB	131.00 (124.00, 137.00)	131.00 (124.00, 137.00)	0.455
PLT	233.00 (198.00, 275.00)	236.00 (202.00, 276.00)	0.056
Eos	1.70 (1.10, 2.60)	1.80 (1.10, 2.60)	0.358
Baso	0.50 (0.40, 0.70)	0.50 (0.40, 0.70)	0.762
LY	33.62±7.64	33.89±7.69	0.217
NEUT	56.84±8.19	56.47±8.27	0.118
Routine Urine index			
UPH	6.12±0.42	6.14±0.43	0.174
SG	1.02±0.01	1.02±0.01	0.704
Disease history			
Hypertension (Yes)	159 (4.0)	57 (3.4)	0.267
Fatty liver (Yes)	1026 (26.0)	439 (25.9)	0.988

shown in Table 3. Finally, we found BMI ($P<0.001$), UPH ($P=0.002$), ALP ($P<0.001$), WBC ($P=0.011$), HDL ($P<0.001$), LDL ($P<0.001$), SCR ($P<0.001$), ALT ($P=0.003$), TP ($P=0.005$) and fatty liver ($P<0.001$) were recognized as independent risk factors for HUA in perimenopausal women.

Constructing and Evaluating a Prediction Model for HUA in Perimenopausal Women

From the analysis of Lasso-logistic regression, we created a nomogram prediction model (Figure 3). The risk level of the results could be calibrated, and we could obtain the total score for the probability of a certain outcome event by

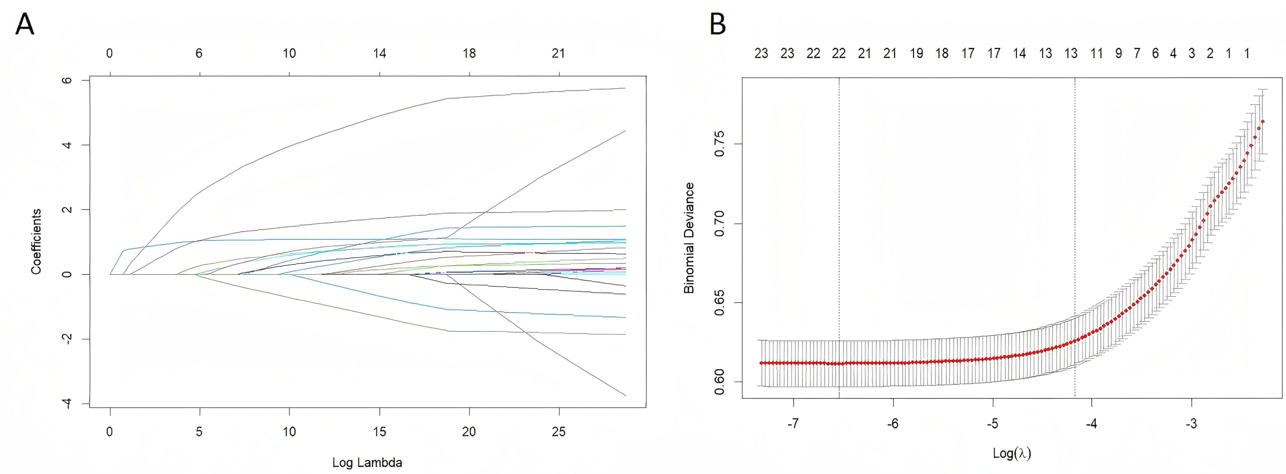

Figure 2 Lasso regression results. (**A**) Lasso coefficient profiles of the variables. (**B**) Demonstrates the process of selecting the optimal parameter (lambda) in Lasso.

Table 3 Binary Logistic Regression Analysis of the Risk Factors for HUA in Perimenopausal Women

Variables	*B*	S.E	OR (95% CI)	*P*
BMI	0.087	0.020	1.091 (1.050–1.134)	<0.001
UPH	−0.430	0.138	0.651 (0.497–0.852)	0.002
ALP	0.010	0.003	1.010 (1.004, 1.015)	<0.001
WBC	0.103	0.041	1.109 (1.024–1.201)	0.011
HDL	−0.950	0.205	0.387 (0.259–0.578)	<0.001
SCR	0.072	0.006	1.072 (1.062–1.088)	<0.001
LDL	0.263	0.069	1.301 (1.136–1.489)	<0.001
ALT	0.011	0.004	1.011 (1.004–1.018)	0.003
TP	0.039	0.014	1.040 (1.012–1.068)	0.005
Fatty liver (Yes)	1.152	0.128	3.164 (2.464–4.063)	<0.001

combining them. An increased total score was associated with a higher risk of HUA. The Bootstrap method was used to validate the nomogram by resampling 1000 times for internal validation of the model, and calibration curves were plotted for both the training and validation sets (Figure 4). The Lasso-logistic method had good performance in predicting HUA in perimenopausal women.

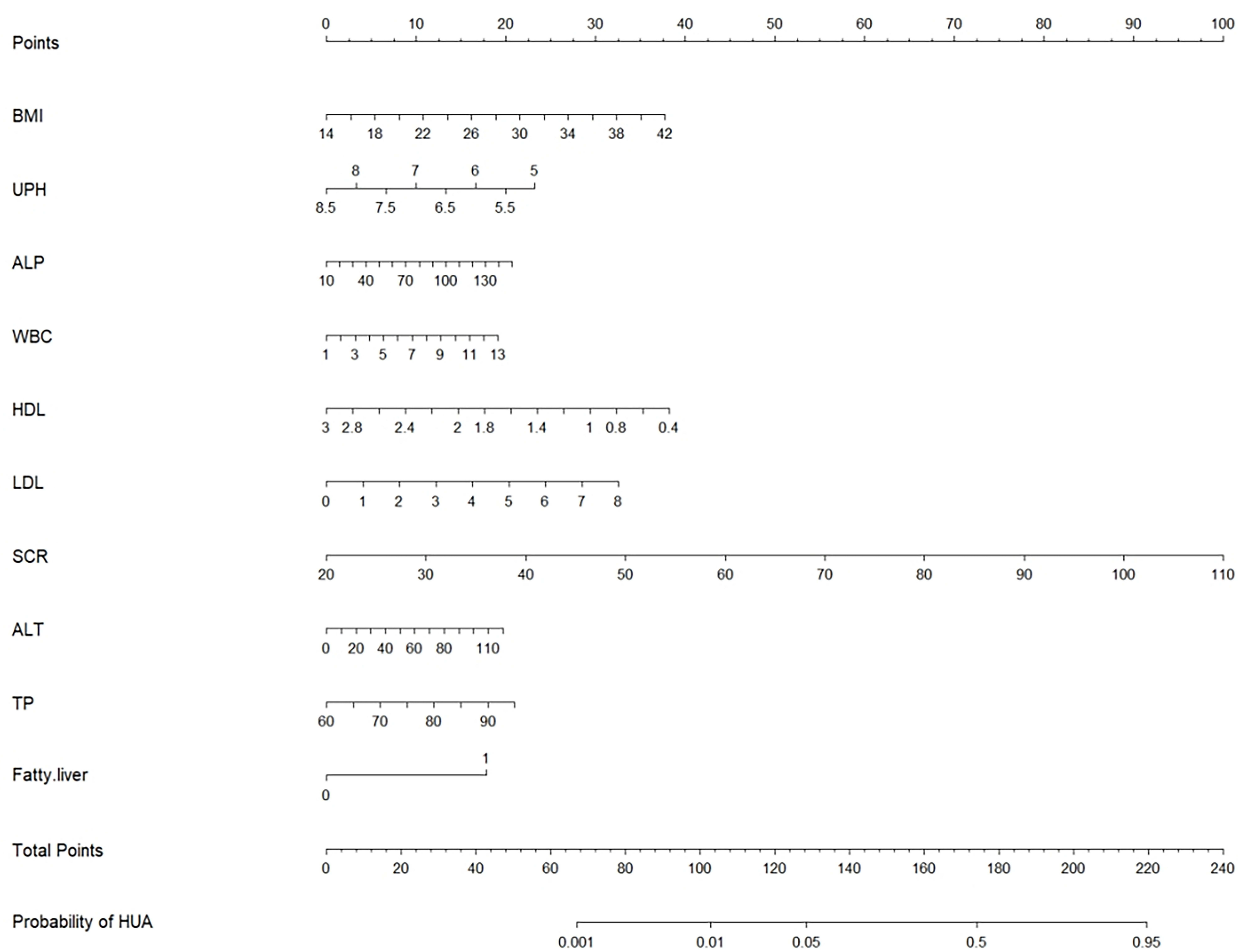

Figure 3 Nomogram for predicting the risk of HUA in perimenopausal women.

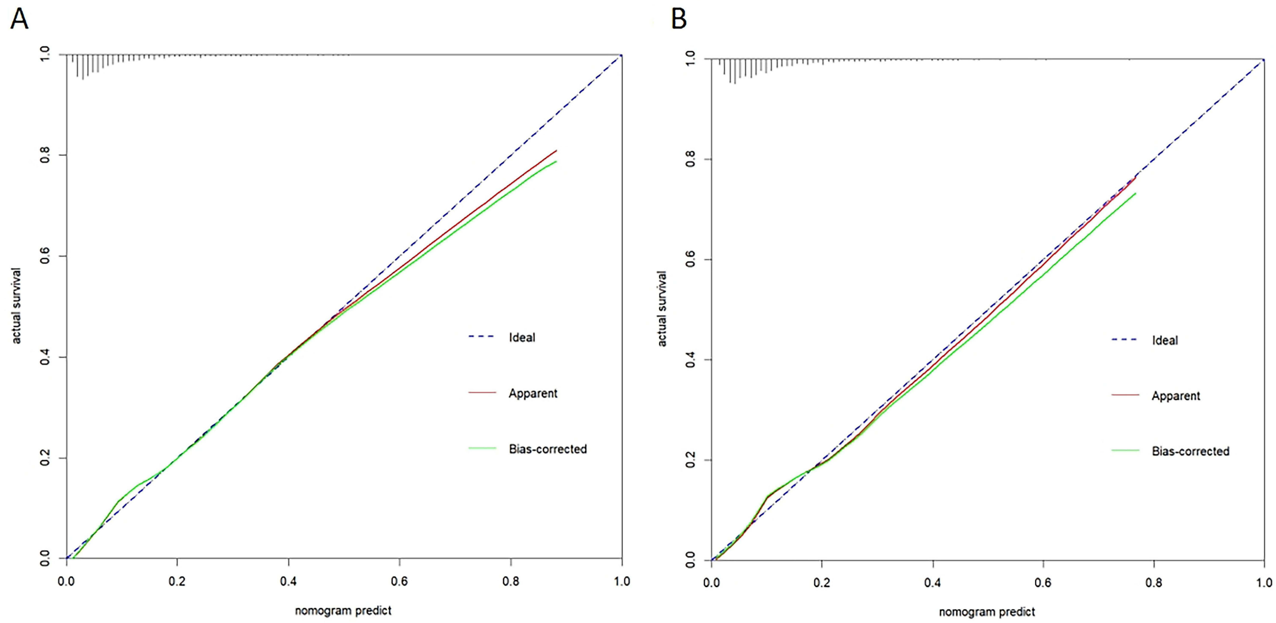

Figure 4 The calibration plot for nomogram. (**A**) Calibration plot for the accuracy of the training set model. (**B**) Calibration plot for the accuracy of the validation set model.

The AUC of the prediction model in training set was 0.819 (95% CI: 0.801–0.838), and the AUC of the prediction model in validation set was 0.787 (95% CI: 0.756–0.818) (Figure 5A). As shown in Figure 5B, the DCA depicted was utilized to ascertain the maximum net benefit of the predictive model.

Stratified Analysis

In this study, we further performed a stratified analysis by BMI. The perimenopausal women were categorized into two groups according to their BMI: the women with BMI ≥ 25 and the women with BMI < 25.[18] Then Lasso regression was used to screen the significant variables, and binary logistic regression analysis was performed in the two groups, and nomogram models were respectively established.

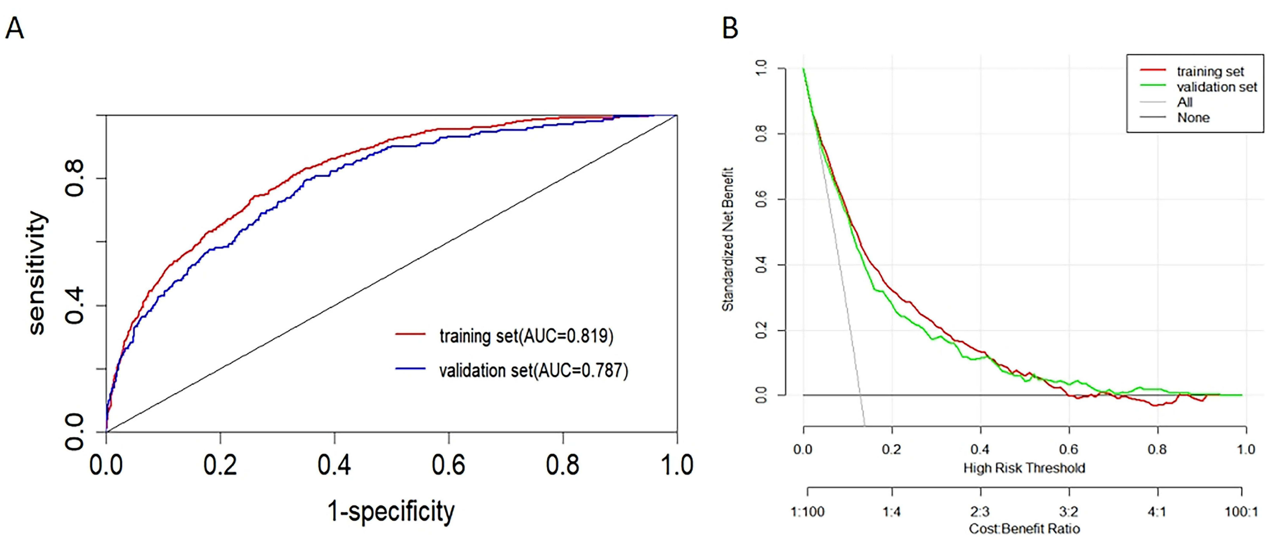

Figure 5 (**A**) ROC curve of the risk factor for predicting HUA in perimenopausal women. (**B**) DCA curve of the predictive model.

Table 4 Binary Logistic Regression Analysis of the Risk Factors for HUA in Perimenopausal Women with BMI < 25.0

Variables	B	S.E	OR (95% CI)	P
BMI	0.134	0.038	1.143 (1.061–1.232)	<0.001
SCR	0.070	0.006	1.073 (1.059–1.086)	<0.001
HDL	−0.710	0.222	0.492 (0.318–0.760)	0.001
LDL	0.339	0.073	1.403 (1.216–1.618)	<0.001
ALT	0.017	0.004	1.017 (1.009–1.026)	<0.001
TP	0.065	0.014	1.067 (1.037–1.098)	0.006
TG	0.178	0.065	1.195 (1.051–1.359)	<0.001
Fatty liver (Yes)	1.036	0.133	2.819 (2.172–3.657)	<0.001

In the population with BMI < 25.0, the prevalence of HUA was 9.4%, while in the population with BMI ≥ 25.0, the prevalence of HUA was 24.3%. In the population with BMI < 25.0, the occurrence of HUA was associated with the following factors: BMI (*P*<0.001), SCR (*P*<0.001), HDL (*P*=0.001), LDL (*P*<0.001), ALT (*P*<0.001), TP (*P*=0.006), TG (*P*<0.001), Fatty liver (*P*<0.001) (Table 4 and Figure 6A). In the population with BMI ≥ 25.0, the development of HUA was influenced by the following factors: BMI (*P*=0.004), ALP (*P*=0.004), WBC (*P*=0.006), SCR (*P*<0.001), HDL (*P*=0.001), ALT (*P*=0.023), Hypertension (*P*=0.034), Fatty liver (*P*<0.001) (Table 5 and Figure 6B). As presented in Figure 7, the predictive model had an AUC of 0.765 in the population with BMI ≥ 25.0 and an AUC of 0.793 in the population with BMI < 25.0.

Discussion

At present, HUA has become a major threat to public health, so it is of great significance to investigate the influencing factors of HUA and then construct a prediction model. In this study, we found that the independent risk factors for HUA were BMI, UPH, ALP, WBC, HDL, LDL, SCR, ALT, TP, and history of fatty liver disease in perimenopausal women through Lasso-logistic regression analysis. We further developed HUA prediction model using these factors in perimenopausal women, achieving an AUC of 0.787. Subsequently, we constructed BMI-stratified prediction models: for individuals with BMI < 25, the model AUC was 0.793; for those with BMI ≥ 25, AUC reached 0.765.

Previous studies have found that estrogen contributes to promote the excretion of SUA in the body.[19] For perimenopausal women, the decline in ovarian function leads to a reduction in estrogen secretion, affecting their SUA levels, thereby increasing the prevalence of HUA in this population. In additional, epidemiological research indicates that the abnormal changes of blood lipid levels, especially triglyceride and HDL, are independent risk factors for HUA. Moreover, insulin resistance caused by obesity and the inhibition of uric acid excretion mediated by adipokines can

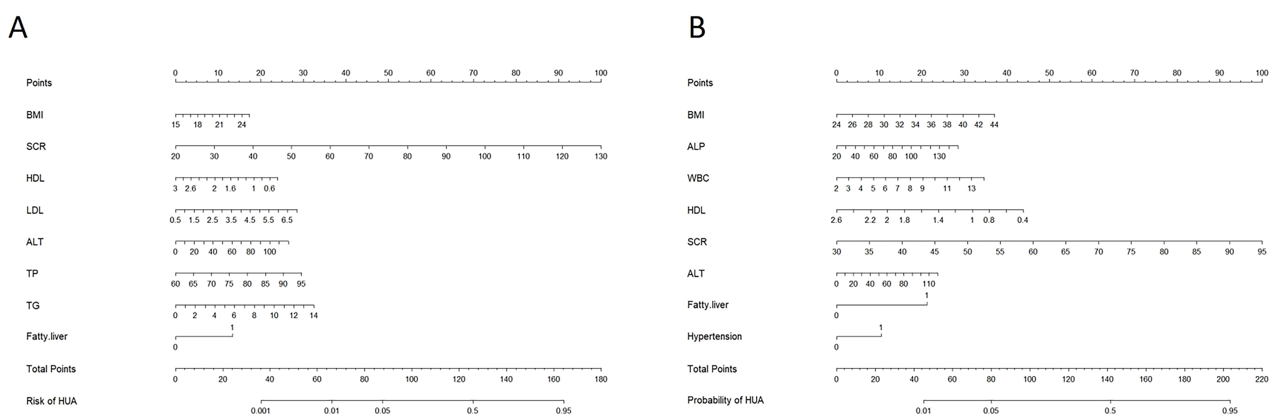

Figure 6 (**A**) Nomogram model for predicting the risk of HUA with BMI < 25. (**B**) Nomogram model for predicting the risk of HUA with BMI ≥ 25.

Table 5 Binary Logistic Regression Analysis of the Risk Factors for HUA in Perimenopausal Women with BMI ≥ 25.0

Variables	*B*	S.E	OR (95% CI)	*P*
BMI	0.088	0.031	1.092 (1.028–1.161)	0.004
ALP	0.010	0.004	1.011 (1.003–1.018)	0.004
WBC	0.137	0.050	1.147 (1.041–1.265)	0.006
SCR	0.073	0.009	1.076 (1.058–1.094)	<0.001
HDL	−0.950	0.279	0.387 (0.224–0.669)	0.001
ALT	0.009	0.004	1.009 (1.001–1.018)	0.023
Hypertension (Yes)	0.499	0.235	1.647 (1.039–2.612)	0.034
Fatty liver (Yes)	1.014	0.168	2.755 (1.983–3.828)	<0.001

increase serum uric acid levels.[20] Hence, HUA is more prevalent in overweight or obese individuals, which is consistent with the findings of our study.[21,22] In our study, the increased levels of ALP, ALT, and HDL were related to the increased risk of HUA, which is similar to the previous findings.[23]

Based on the results of binary logistic regression, HDL and UPH were identified as protective factors against HUA in perimenopausal women. Previous studies suggest that fluctuations in HDL levels can affect the kidneys, thereby influencing the excretion of uric acid.[24,25] Acidic urine might aggravate insulin resistance, leading to increased serum uric acid levels, and increased insulin resistance could further reduce UPH. In the preset study, fatty liver was a significant risk factor for HUA in perimenopausal women. However, the exact mechanism is not yet clear. It is speculated that insulin resistance increases hepatic fat synthesis, promoting the development of fatty liver, which finally leads to disordered purine metabolism and

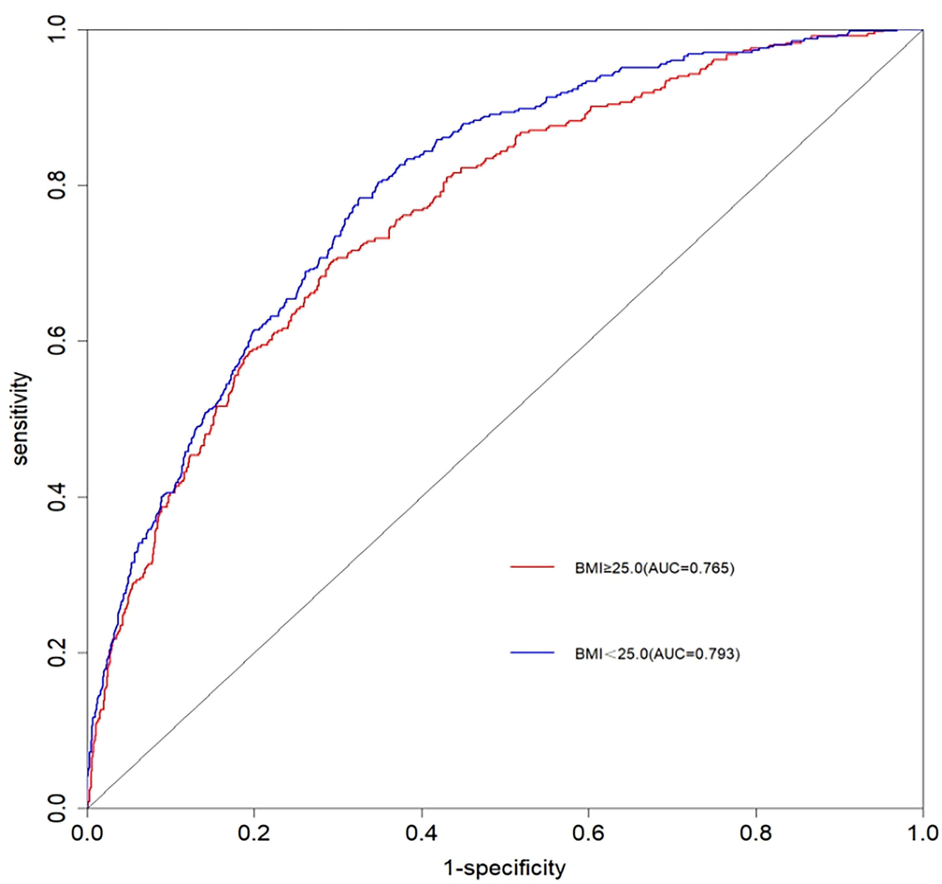

Figure 7 ROC curve to evaluate two models.

elevated serum uric acid levels.[26] We also found that SCR was closely related to HUA, which is contrary to a previous study conducted on the general population.[27] This discrepancy might be attributed to the specific physiological stage of the study subjects, and the all subjects were perimenopausal women. Specifically, the estrogen levels in women are relatively stable, therefore having a smaller impact on uric acid or SCR levels, before entering perimenopause. However, a significant increase in SCR levels typically requires a longer period of estrogen deficiency, a pathophysiological process that often occurs after women have fully entered perimenopause. Notably, all participants were already in perimenopause in this study, which might influence the interpretation of these results.

After stratifying by different BMI, we also found significant differences in the risk factors for HUA among perimenopausal women with different BMI. Compared to the group with BMI ≥ 25.0, LDL, TP, and TG were identified as different predictors in the group with BMI < 25.0. The reason for this situation might be that visceral fat accumulation leads to increased release of free fatty acids and disordered secretion of adipokines, resulting in increased hepatic lipoprotein synthesis and reduced uric acid excretion. High LDL levels might reflect a disturbance in reverse cholesterol transport, elevated TP suggests hepatic protein metabolism disorder, and abnormal TG directly participates in the formation of insulin resistance. Together, these three factors constitute the core characteristics of "metabolic obesity".[28] In the group with BMI ≥ 25.0, ALP, WBC, and Hypertension were identified as different predictors, and the combination of these risk factors exhibits a stronger characteristic of systemic inflammation. An elevated ALP level might reflect the progression of non-alcoholic fatty liver disease, an increased WBC count suggests a state of chronic low-grade inflammation, and the synergistic effect between hypertension and HUA might originate from reduced renal blood flow and inhibited uric acid excretion caused by the activation of the renin-angiotensin system.[29]

Previous studies have identified PPARγ gene, BMI, and gender as significant predictors of hyperuricemia (HUA) when developing prediction models.[30] However, applying this model requires individuals to possess a high level of professional knowledge and necessitates consideration of other factors, which presents significant challenges for its promotion and practical use. Furthermore, a Japanese study utilized gut microbiota to predict hyperuricemia.[31] Although the model demonstrated strong predictive ability, its requirement for stool sample collection makes it difficult to promote clinically.

However, this study still has several limitations. First, as a single-center study, we lacked external data to validate our model, which might affect the generalizability of our findings. Second, the definition of perimenopausal women did not include hormone level testing for all participants. Finally, the collected information lacked data on subjects' daily lifestyle factors.

In Summary, our study developed a nomogram model for predicting HUA risk in perimenopausal women using ten distinct clinical indicators (BMI, UPH, ALP, WBC, HDL, LDL, SCR, ALT, TP, and history of fatty liver disease). Furthermore, the performance of this model were proven to be quite effective. These findings provide important clues for enhancing the health management of perimenopausal women.

Data Sharing Statement

The data of this study are available from Dr. Tian-Ping Zhang upon reasonable request.

Ethics Statement

This study was approved by the Ethical Committee of the First Affiliated Hospital of USTC (2025-RE-191).

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

Yu-Fei Liu and Xiao-Jing Li are co-first authors for this study. The authors have no conflicts of interest in this work.

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