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A predictive model of macrosomic birth based upon real-world clinical data from pregnant women

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Abstract

Background: Fetal macrosomia is associated with an increased risk of several maternal and newborn complications. Antenatal predication of fetal macrosomia remains challenging. We aimed to develop a nomogram model for the prediction of macrosomia using real-world clinical data to improve the sensitivity and specificity of macrosomia prediction.

Methods: In the present study, we performed a retrospective, observational study based on 13,403 medical records of pregnant women who delivered singleton infants at a tertiary hospital in Shanghai from 1 January 2018 through 31 December 2019. We split the original dataset into a training set ($n = 9382$) and a validation set ($n = 4021$) at a 7:3 ratio to generate and validate our model. The candidate variables, including maternal characteristics, laboratory tests, and sonographic parameters were compared between the two groups. A univariate and multivariate logistic regression was carried out to explore the independent risk factors for macrosomia in pregnant women. Thus, the regression model was adopted to establish a nomogram to predict the risk of macrosomia. Nomogram performance was determined by discrimination and calibration metrics. All the statistical analysis was analyzed using R software.

Results: We compared the differences between the macrosomic and non-macrosomic groups within the training set and found 16 independent risk factors for macrosomia ($P < 0.05$), including biparietal diameter (BPD), head circumference (HC), femur length (FL), amniotic fluid index (AFI) at the last prenatal examination, pre-pregnancy body mass index (BMI), and triglycerides (TG). Values for the areas under the curve (AUC) for the nomogram model were 0.917 (95% CI, 0.908–0.927) and 0.910 (95% CI, 0.894–0.927) in the training set and validation set, respectively. The internal and external validation of the nomogram demonstrated favorable calibration as well as discriminatory capability of the model.

Conclusions: Our model has precise discrimination and calibration capabilities, which can help clinical healthcare staff accurately predict macrosomia in pregnant women.

Keywords: Macrosomia, Prediction model, Nomogram, Clinical data

Background

Macrosomia refers to a birth weight that reaches 4000 g. It affects approximately 3–15% of pregnancies and frequently leads to adverse pregnancy outcomes such as shoulder dystocia, postpartum hemorrhage, and birth fractures [1]. A reliable prenatal predictor of

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macrosomia would therefore be of great significance for the optimization of clinical management and for improving maternal and infant outcomes.

Fetal weight is traditionally estimated by abdominal examination or ultrasonographic evaluation in obstetric practice [2]. The accuracy of abdominal examination is influenced by obesity, uterine fibroids, the amount of amniotic fluid, and clinical experience, and as ultrasonography is a practical method used to estimate the fetal weight (EFW), the post-test probability of identifying macrosomia varies from 15 to 79% with sonographic EFW [3]. This accuracy is significantly diminished with macrosomic infants, and low accuracy limits the predictive value of ultrasonograms.

Various predictive models based upon traditional statistical formulas or machine learning algorithms have been applied to the prediction of macrosomia in recent years. Daisuke et al. [4] developed an integer scoring system for excluding macrosomia using only maternal physical examination without sonographic information, and Wang et al. [5] constructed a random forest model that involved extra-pelvic measurement information and achieved improved sensitivity, specificity, and area under the receiver operating characteristic (ROC) curve (at 91.7, 91.7, and 95.3%, respectively). While the current predictive models obtained a higher degree of accuracy than the ultrasonographic and maternal abdominal evaluative methods, the models did not address comprehensive variables relevant to risk factors of macrosomia, and this might have weakened the overall accuracy of these models. Unusual indicators (e.g., carnitine metabolism or fetal soft tissue) were selected in some of the previous models, but these indicators are not always available, which is troubling to clinicians. Some models are exclusive in their predictions within a population of women with gestational diabetes mellitus (GDM) but neglect predictions in individuals without GDM [6]. Based on the aforementioned data, the use of comprehensive and readily available predictors—as well as the expansion of the study population—may augment the application and precision of the model.

In this study, we aimed to develop a more accurate, applicable, and stable model to predict the risk of macrosomia and employed a retrospective analysis of common clinical data that encompassed maternal characteristics, laboratory tests, and sonographic parameters in a large cohort of pregnant women. We believe that our study will provide a reference for the development of macrosomia prevention and appropriate intervention strategies.

Methods

Study population

In this retrospective study, we extracted data from the digital medical records system of the International Peace Maternity and Child Health Hospital between 1 January 2018 and 31 December 2019. The inclusion criteria were as follows: (1) singleton pregnancy; (2) gestational weeks $\geq$28; (3) a normal pregnancy outcome (no stillbirths, neonatal deaths, or severe fetal malformations). After data screening, a total of 13,403 subjects were included in our study (Fig. 1).

Data collection and variables included for analysis

We searched for variables of macrosomia that were reported in studies or systematic reviews, can be easily ascertained in different setting with various clinical experience, and are part of the routine examination during pregnancy. In this retrospective study, we collected maternal data that included demographics, clinical characteristics, laboratory tests, and fetal B-ultrasonographic examination. The extreme and error values of the measurement data were cleaned and the categorical data were normalized and coded.

At the first antenatal visit between 9 and 13 weeks of gestation, we collected data on the mother's and husband's demographic characteristics, medical history, and reproductive history. Maternal height, weight, gravity, parity, educational level, and basal blood pressure (systolic blood pressure, SBP; diastolic blood pressure, DBP) were recorded via face-to-face interviews. The pre-pregnancy body mass index (pre-pregnancy BMI) was calculated by dividing the pre-pregnancy weight (kg) by the pre-pregnancy height (m^2), and numbers were divided into four levels: $<18.5\,kg/m^2$ for underweight, 18.5–$24.9\,kg/m^2$ for normal weight, 25.0–$29.9\,kg/m^2$ for overweight, and $30\,kg/m^2$ for obesity. Gestational weight gain (GWG) during pregnancy was determined by subtracting pre-pregnancy weight from the woman's weight at her last prenatal examination. Appropriate gestational weight gain was stated as 12.5 kg to 18 kg for underweight, 11.5 kg to 16.0 kg for normal weight, 7 kg to 11.5 kg for overweight, and 5 kg to 9 kg for obesity according to the recommendations of the 2009 Institute of Medicine (IOM) guidelines categorized by pre-pregnancy BMI for each woman, and below or above the interval range was defined as insufficient or excessive weight gain [7].

We computed gestational age from the first day of the last menstrual period or the dating ultrasonographic scan performed prior to 20 weeks of pregnancy. Maternal fasting lipid serum samples were obtained in the first trimester (between nine and 14 weeks), collected in 10-mL vacutainer tubes, and centrifuged. Laboratory indices included triglycerides (TG), total cholesterol (TC), high-density lipoprotein (HDL), and low-density lipoprotein (LDL). The glucose index was obtained from a 75-g oral glucose tolerance test (OGTT) between

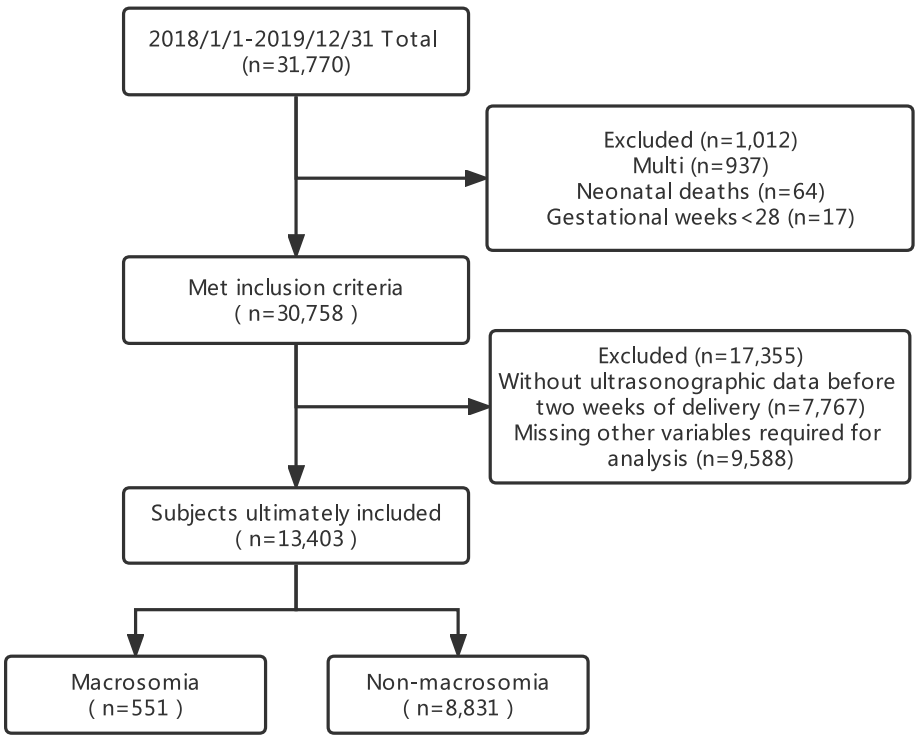

Fig. 1 Chart illustrating patient flow in the present study

gestational weeks 24 and 28—including fasting plasma glucose (FPG), one-hour glucose (GLU-1H), and two-hour glucose (GLU-2H), and hemoglobin (HbA1c). Routine sonographic evaluations performed by experienced doctors included fetal abdominal circumference (AC), biparietal diameter (BPD), head circumference (HC), humerus length (HL), femur length (FL), transverse trunk diameter (TTD), anteroposterior trunk diameter (APTD), and amniotic fluid index (AFI) at the last prenatal examination. The occurrence of macrosomia was the primary outcome used in this study. Shortly after birth, the newborns were weighed, their weights were recorded by medical staff, and those neonates with birth weights ≥4000 g were defined as manifesting macrosomia.

Statistical analysis

We performed data analysis using R software version 4.1.2 (2021-11-01). Preliminary statistical analyzes including the Kolmogorov-Smirnov test [8] and Q-Q plots were performed to assess whether the data followed a normal distribution. Medians (and interquartile ranges, IQR) were used for continuous variables and counts and percentages for categorical variables. The Wilcoxon rank-sum test was employed for comparisons of continuous variables between groups, and the Chi-squared and Fisher's exact probability tests were used for categorical variables, as appropriate. Differences were considered significant when they showed a p-value < 0.05.

The original dataset was randomly allocated to training and validation sets at a 7:3 ratio. A univariate logistic regression analysis was first performed to assess each variable's significance separately. Any variables having a significant univariate test at a 0.05 level were selected as candidates for the following multivariate analysis. After the initial variable selection in the univariate analysis, multivariate logistic regression with a backward stepwise method was exploited within the training set to determine the risk factors associated with macrosomia. All variables screened by the backward stepwise algorithm would be included in the final model. Odds ratios and their corresponding 95% confidence intervals were then calculated for each independent variable. In addition, we employed ROC_AUC, sensitivity and specificity as our model evaluation metrics. To appraise the prediction capability of the logistic model and its fitness, we used the Hosmer and Lemeshow test and calculated the areas under the receiver operating characteristic curve (AUC). Multicollinearity was also tested on the final model by accessing the value of variance inflation factor (VIF). A nomogram model was then created based upon the final logistic regression model, and the nomogram model

was validated by measuring discrimination and calibration curves both internally (training set) and externally (validation set). We assessed discrimination between observed and predicted outcomes using the metrics of ROC_AUC.

Results

Demographic and medical characteristics

The data from a total of 13,403 pregnant women were entered into our analysis. The original dataset was split into a training set ($n=9382$) and a validation set ($n=4021$), and we then compared the differences between the macrosomic and non-macrosomic groups within the training set. The mean birth weight of newborns in this study was 3345.9 g; 6893 (51%) of the neonates were male, and 6510 (49%) were female, with a macrosomia prevalence of 5.7%.

The demographic information is summarized in Table 1 and indicates that the BMI, gestational age (GA), SBP, and DBP were significantly higher in the macrosomic group than in the non-macrosomic group. Compared with the non-macrosomic group, the macrosomic group exhibited a significantly elevated percentage of excessive gestational weight gain (62% vs. 34%, $p<0.05$) and showed a significantly reduced proportion of individuals with an educational level above a bachelor's degree for both women (17% vs. 23%, $p<0.05$) and their partners (22% vs. 25%, $p<0.05$).

Table 2 depicts the medical characteristics, including ultrasonographic and clinical laboratory test results. BPD, HC, FL, HL, TTD, APTD, AC, and AFI showed significantly higher median values in the macrosomic group compared to the non-macrosomic group. In terms of clinical laboratory findings, FPG, GLU-1H, GLU-2H, and TG in the macrosomic group were significantly augmented relative to the non-macrosomic group, while HDL was lower than in the non-macrosomic group. TC ($p=0.6$) and LDL ($p=0.4$) did not differ between groups.

Table 1 Baseline characteristics of the study groups

Characteristic	Macrosomia[a] $n=551$	Non-macrosomia[a] $n=8831$	p-value[*]
BMI	22.2 (20.2, 24.2)	20.7 (19.2, 22.6)	< 0.001
Gestational age (GA)	39.5 (39.0, 40.3)	39.1 (38.4, 39.6)	< 0.001
Gravidity	2.0 (1.0, 3.0)	2.0 (1.0, 2.0)	< 0.001
Parity	1.0 (1.0, 2.0)	1.0 (1.0, 2.0)	0.074
SBP	112 (104, 121)	110 (102, 119)	< 0.001
DBP	69 (63, 76)	68 (62, 75)	0.029
Age			0.087
≥ 35	418 (76%)	6971 (79%)	
< 35	133 (24%)	1860 (21%)	
Husband age			0.021
≥ 35	354 (64%)	6089 (69%)	
< 35	197 (36%)	2742 (31%)	
Educational level			< 0.001
Bachelor's degree	293 (53%)	4728 (54%)	
Above bachelor's	93 (17%)	,2020 (23%)	
Below bachelor's	165 (30%)	2083 (24%)	
Husband's educational level			0.017
Bachelor's degree	286 (52%)	4762 (54%)	
Above bachelor's	121 (22%)	2198 (25%)	
Below bachelor's	144 (26%)	1871 (21%)	
Conception			0.12
Natural conception	498 (90%)	8144 (92%)	
Assisted reproduction	53 (9.6%)	687 (7.8%)	
GWG			< 0.001
Optimal	165 (30%)	3754 (43%)	
Inadequate	44 (8.0%)	2064 (23%)	
Excessive	342 (62%)	3013 (34%)	
Smoking-tobacco use			0.8
No	547 (99%)	8771 (99%)	
Yes	4 (0.7%)	60 (0.7%)	
Alcohol use			0.2
No	530 (96%)	8571 (97%)	
Yes	21 (3.8%)	260 (2.9%)	
Family history of diabetes or hypertension			0.6
No	422 (77%)	6693 (76%)	
Yes	123 (22%)	1996 (23%)	
Unknown	(1.1%)	142 (1.6%)	

[a] Median (IQR); n (%)

[*] Wilcoxon rank-sum test; Pearson's Chi-squared test; Fisher's exact probability test

Regression analysis and risk factors for macrosomia

Our multivariate regression analysis of factors associated with macrosomia is shown in Table 3. The model was established with macrosomia as the outcome variable and twenty-four significant indices in the univariate analysis as independent variables using backward stepwise regression. Sixteen predictors were included in the final model: E educational level, GWG, fetal sex, gravidity (GNUM), BMI, GA, AC, BPD, HC, FL, HL, TTD, AFI, FPG, GLU-1H, and TG. The results of the Hosmer and Lemeshow test provide a p-value of 0.15, that is greater than 0.05, indicating no evidence of poor fit and our model is correctly specified. We also analyzed multicollinearity, with all indices showing a VIF of less than 3, and thus, we had no issue with collinearity.

Table 2 Medical characteristics of the study groups

Characteristic	Macrosomia[a] $n=551$	Non-macrosomia[a] $n=8831$	p-value[*]
BPD	97.0 (95.0, 99.0)	94.0 (92.0, 96.0)	< 0.001
HC	332 (324, 338)	320 (313, 328)	< 0.001
FL	72.0 (70.0, 73.0)	69.0 (67.0, 71.0)	< 0.001
HL	63.0 (62.0, 64.0)	60.0 (59.0, 62.0)	< 0.001
TTD	108 (105, 112)	101 (97, 105)	< 0.001
APTD	110 (106, 114)	103 (99, 107)	< 0.001
AC	342 (334, 351)	320 (309, 331)	< 0.001
AFI	131 (108, 157)	120 (102, 142)	< 0.001
FPG	4.30 (4.04, 4.60)	4.20 (3.96, 4.46)	< 0.001
GLU-1H	7.92 (6.93, 9.01)	7.61 (6.67, 8.74)	< 0.001
GLU-2H	6.69 (5.86, 7.61)	6.41 (5.62, 7.35)	< 0.001
HbA1c	5.00 (4.80, 5.20)	5.00 (4.80, 5.10)	< 0.001
TG	1.39 (1.09, 1.74)	1.28 (1.02, 1.62)	< 0.001
TC	4.44 (3.97, 4.89)	4.44 (4.00, 4.92)	0.6
HDL	1.85 (1.59, 2.15)	1.94 (1.68, 2.21)	< 0.001
LDL	2.51 (2.12, 2.97)	2.50 (2.12, 2.94)	0.4

[a] Median (IQR)

[*] Wilcoxon rank-sum test

Table 3 Factors associated with macrosomia among women at the international peace maternity and child health hospital ($n=9382$)

Characteristic	OR[a]	95% CI[b]	p-value
Educational level			
Bachelor's degree	–	–	
Above bachelor's	0.75	(0.56, 0.98)	0.037
Below bachelor's	1.19	(0.93, 1.51)	0.2
GWG			
Optimal	–	–	
Inadequate	0.51	(0.34, 0.74)	< 0.001
Excessive	1.59	(1.27, 2.00)	< 0.001
Fetal Sex			
Female	–	–	
Male	1.67	(1.34, 2.08)	< 0.001
GNUM	1.14	(1.04, 1.24)	0.005
BMI	1.07	(1.03, 1.11)	< 0.001
GA	1.22	(1.08, 1.37)	0.001
AC	1.08	(1.06, 1.09)	< 0.001
BPD	1.08	(1.03, 1.14)	0.003
HC	1.03	(1.01, 1.04)	< 0.001
FL	1.08	(1.01, 1.15)	0.023
HL	1.19	(1.12, 1.26)	< 0.001
TTD	1.02	(0.99, 1.05)	0.12
AFI	1.01	(1.00, 1.01)	< 0.001
FPG	1.44	(1.11, 1.87)	0.006
GLU-1H	1.09	(1.01, 1.18)	0.030
TG	1.17	(0.97, 1.40)	0.093

[a] *OR* Odds ratio

[b] *CI* Confidence interval

Nomogram construction and validation

We constructed a nomogram model based upon the 16 predictors noted above to predict the risk of macrosomia (Fig. 2), with each predictor given a point according to the characteristics of each woman; the total number of points was then calculated to obtain the risk of macrosomia. The model achieved satisfactory performance, obtaining a sensitivity of 0.898 and a specificity of 0.781 with the optimal probability threshold chosen. Additionally, the values for the AUCs of the nomogram model were 0.917 (95% CI, 0.908–0.927) and 0.910 (95% CI, 0.894–0.927) in the training set and validation set, respectively, indicating that the model was robust in its discriminative ability (Fig. 3). Both internal and external calibration curves also confirmed that there was a favorable concordance between the observed and predicted probabilities (Fig. 4).

Discussion

In this single-center retrospective study, we developed and validated a nomogram model for the prediction of macrosomia among newborns and achieved satisfactory predictive effects based upon the clinical data from a large cohort of pregnant women.

Pregnancy is a complex process accompanied by substantial changes in sugar and lipid metabolism [9]. Considering that these indicators may be key factors that contribute to fetal weight changes, we combined the key indicators (including blood glucose and lipid parameters) with the general indicators (for example, demographic characteristics and fetal intuitive sonography image measurements) as variables in our model.

It has been proposed that maternal hyperglycemia leads to fetal hyperglycemia, stimulating maturation and hypertrophy of the fetal pancreas [9], and various studies have indicated that GDM constitutes one of the important factors affecting the onset and development of macrosomia [8, 10]. The results of our study revealed that the incidence of macrosomia was 6.38% (115/1802) for women with GDM and 5.65% (649/11,482) for women with non-GDM, numbers consistent with the previous studies [11]. Several authors have supported pregnant women's blood glucose levels as strongly associated with the incidence of macrosomia, regardless of a diagnosis of GDM [12, 13]. We usually perform an OGTT on pregnant women between their 24th and 28th gestational weeks to diagnose GDM, as OGTT, FPG, GLU-1H, GLU-2H, and HBA1C are important indicators in the assessment of maternal blood glucose levels. Through multivariate analysis, fasting glucose

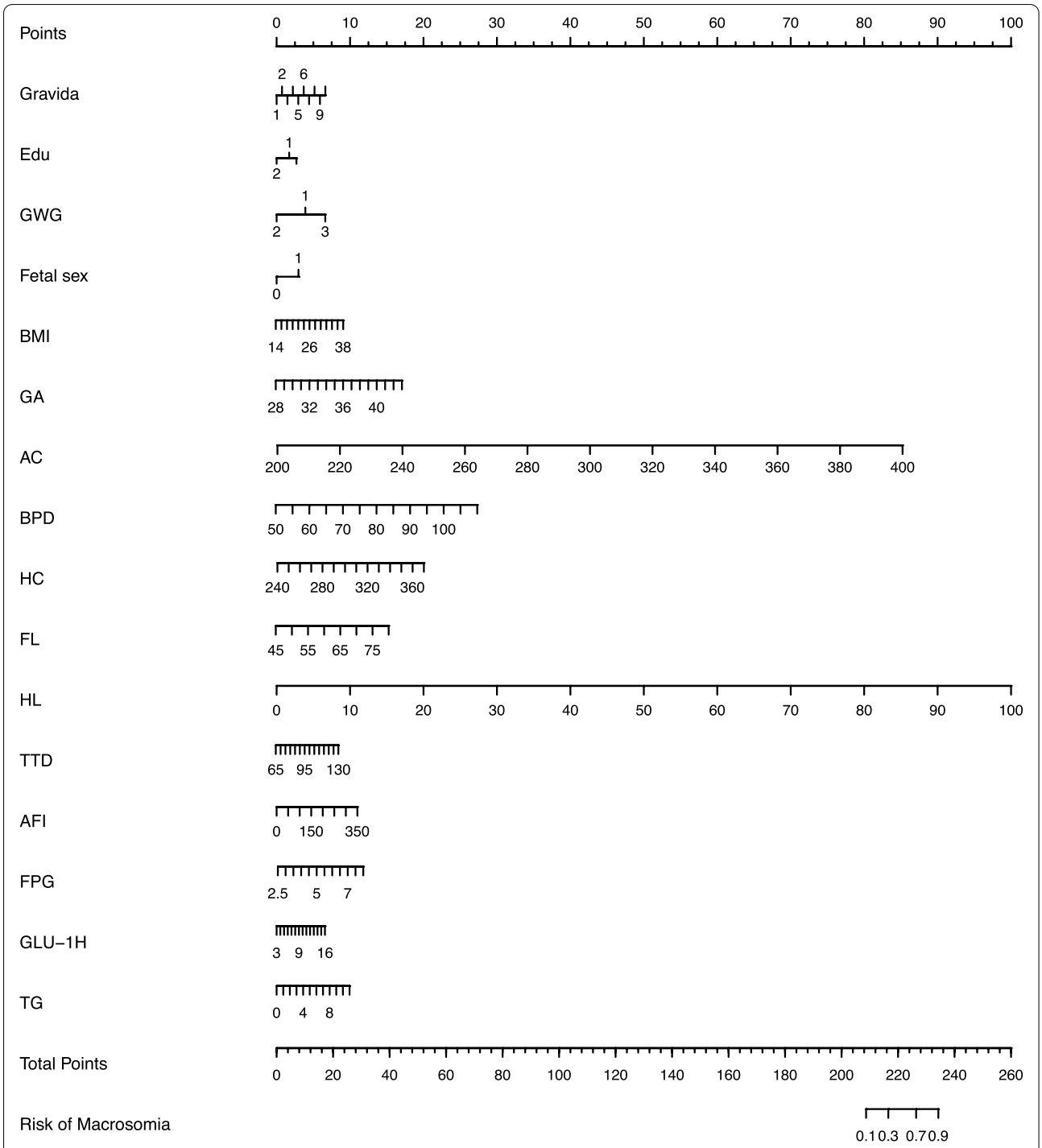

Fig. 2 Nomogram model for predicting the risk of macrosomia. Nomogram model for predicting the risk of macrosomia using 16 predictors: Gravida, gravidity; Edu, educational level; GWG, gestational weight gain; fetal sex; BMI, body mass index; GA, number of gestational weeks; AC, abdominal circumference; BPD, biparietal diameter; HC, head circumference; FL, femur length; HL, humerus length; TTD, transverse trunk diameter; AFI, amniotic fluid index; FPG, fasting plasma glucose; GLU-1H, glucose at one-hour post-OGTT; TG, triglycerides

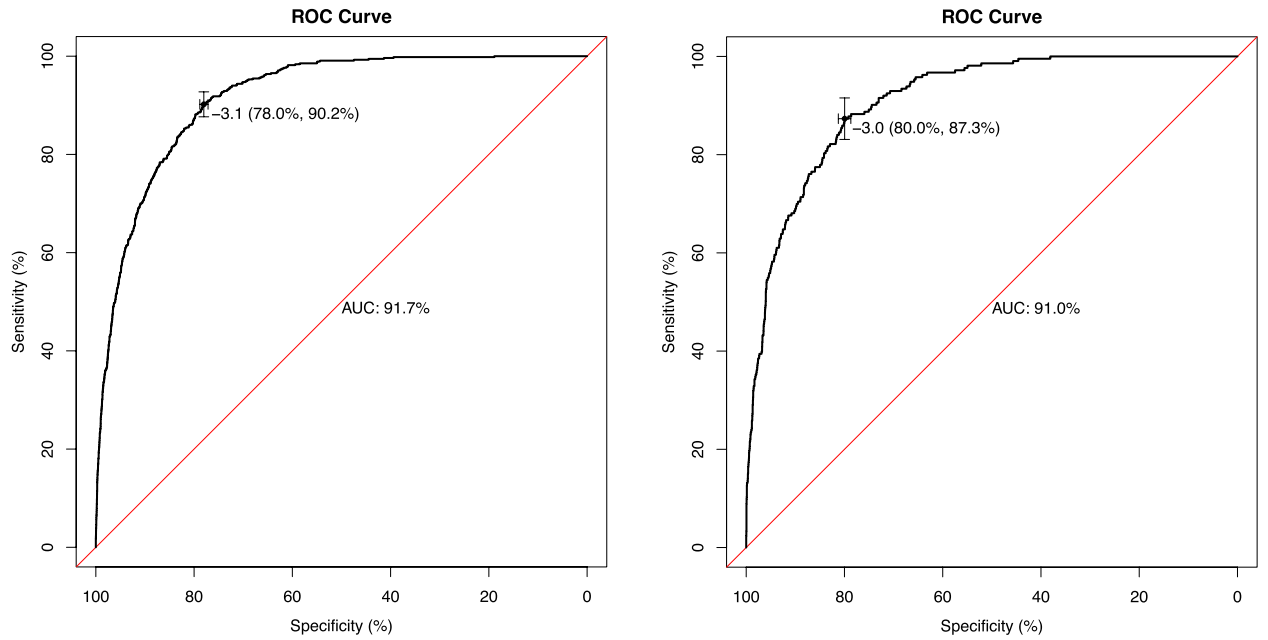

Fig. 3 ROC curve of macrosomia. The ROC curve of macrosomia concerning its internal validation is shown in the left panel, and that for external validation is shown in the right panel

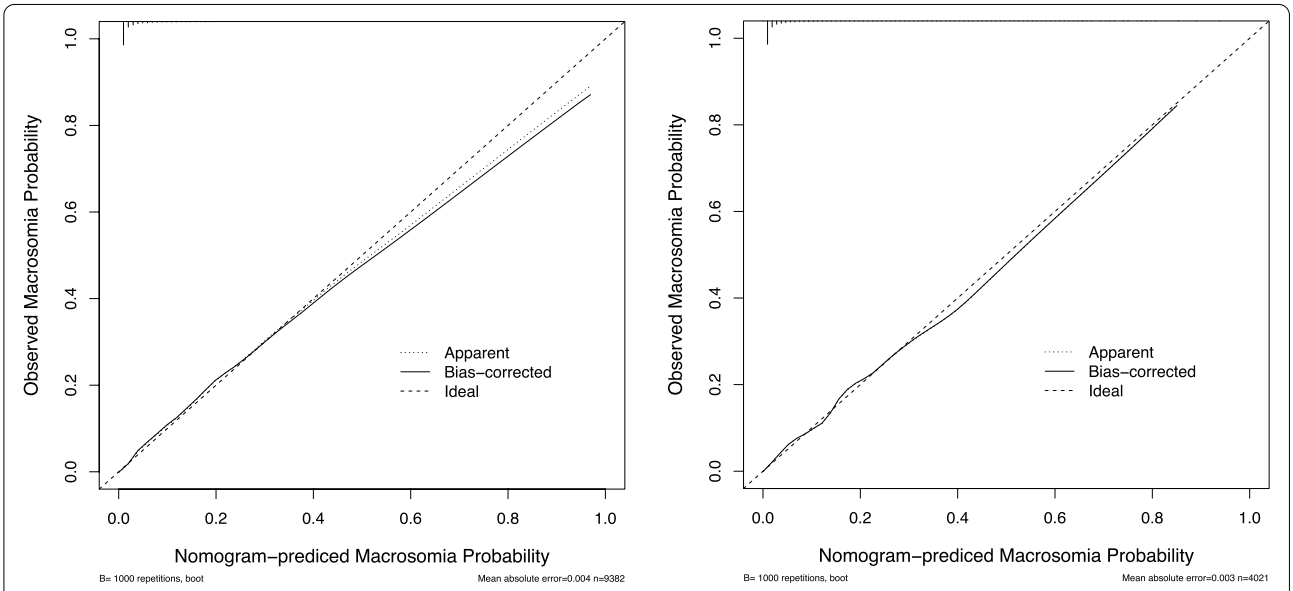

Fig. 4 Calibration curve. The calibration curve for the internal validation of the nomogram model is shown in the left panel, and the calibration curve for external validation is shown in the right panel

and OGTT-1H were then utilized as indicators in our model.

Maternal serum lipids may comprise an important fuel in fetal overgrowth during the entire pregnancy [14]. Since Xue et al. hypothesized that elevated TG levels in early pregnancy but not in late pregnancy were crucial risk factors associated with the incidence of fetal macrosomia [15], we then chose the lipid index in early pregnancy as the variable we used for the prediction of macrosomia. When we herein collected four maternal

lipid parameters (TC, TG, HDL, and LDL) in the first trimester (9th and 13th gestational weeks) of pregnancy, our results revealed that TGs showed high specificity in the prediction of macrosomia, and we, therefore, chose TGs as the predictive indicator in our model.

The most objective method currently employed to estimate fetal body weight is ultrasonographic (US) measurement, which encompasses over 30 different formulas for the US estimates to predict newborn birth weight [16–18], with the most widely used being the Hadlock formula [19]. To generate sonographic fetal weight estimations with a lower error margin, many formulas have reflected disparate parameters of the fetus (fetal abdominal fat layer [20], shoulder soft-tissue thickness [21], biacromial diameter [22]), and some have even entailed 3D sonographic measurements [23]. Although such formulas and novel predictors may improve the accuracy of US evaluation, they nevertheless increase technical difficulty and sonication time. Considering the availability of predictors, we used B-ultrasonography 2 weeks prior to delivery to assess the predictive capability of our model.

The nomogram model can transform the cumbersome regression equation into a visually legible graph that is both convenient and rapid in its practical application [24]. Each predictor is given a point according to the woman's characteristics, and the point total is then calculated to obtain the risk of macrosomia. Mazouni et al. developed a nomogram to predict macrosomia based on maternal demographic characteristics and US variables, with the model achieving moderate predictive ability at an AUC of 0.850 [25]. These authors' sample size was, however, quite small and dismissed the influence of maternal metabolism. Although Sun et al. established a nomogram model combined with carnitine-related metabolic variables for predicting macrosomia in pregnant women with GDM [26], carnitine metabolism is not routinely used in the clinical setting, restricting its application. Shigemi et al. created a scoring system based upon the significant predictors of macrosomia without sonographic information [4], and their system exhibited a high negative predictive value of 0.996–1.000, while the positive predictive value for screening macrosomia was extremely low (0.003). Zou et al. [27] and Kang et al. [28] published models that could only be applied to women with GDM rather than to all pregnant women, and Ye et al. used ensemble methods (one comprising a machine-learning algorithm) to improve the prediction of fetal macrosomia [18]. Unfortunately, ensemble methods are cumbersome and limited in their practicability. In contrast, our model was applied to the entire population of pregnant women and displayed many advantages. First, the precision of our model met or exceeded the optimal predictive levels recorded in the literature [25, 27]. The areas under the ROC curves (AUCs) for the internal and external validation of our model were 91.7 and 91.0, respectively. In addition, using our model, we selected alternative but still routine clinical data that were easily accessible and relatively comprehensive. Macrosomia risk factors can be classified into three components: maternal characteristics, metabolic parameters, and US measurements. However, as most models only incorporate some of these three risk factors to predict the incidence of macrosomia, we posit that our predictive model is more generalizable, precise, and clinically suitable.

Our nomogram model could be a practical tool for clinical work. Once the model shows the possibility of macrosomia, suggesting that pregnant women might be in an over-nutrition condition and need strictly controlled weight gain by lifestyle, diet, exercise. Meanwhile, doctors enhance close monitoring and supervision on them. Nowadays, over-estimated fetal weight could result in over-classification of fetuses as macrosomic with unnecessary cesarean deliveries, under-estimated fetal weight could also pose a risk of dystocia or even stillbirth. Our model fits with the current strategy for precision medicine can guide the mode of delivery and provide assistance at birth. For example, if the model shows a high probability of macrosomia, we can arrange medical personnel, drugs and medical supplies ahead of time in order to prevent postpartum hemorrhage, shoulder dystocia, severe perineal lacerations etal actively. All in all, our model judges the macrosomia accurately and covers the clinical pregnancy management during the antenatal, intrapartum and postpartum periods. Significantly, our model will be a strong aidarm to enhance doctors and midwives' decision confidence, as well as bring lower anxiety of pregnant women due to the uncertainty of their fetal weight. There were some limitations to the present study. First, we are a single obstetric hospital that principally covers low-to-moderate-risk pregnant women, and this cohort may not fully represent all obstetric practices in the community. Second, this was a retrospective study that lacked the validation of relevant variables, thus slightly reducing its overall credibility; in the next phase, we will include the relevant variables and appropriate influencing factors and initiate a prospective study. Finally, the occurrence of macrosomia is affected by many factors, including the environment. For example, some evidence suggests that exposure of pregnant women to air pollutants [29], such as $PM_{2.5}$, NO_2, and O_3, and passive smoking may also increase the fetal risk for macrosomia. We herein ignored the influences of environmental effects, lifestyle, work stress, and social relationships.

Conclusions

In summary, our proposed predictive nomogram model can be used effectively to prognosticate the incidence of macrosomia. The highly predictive sensitivity and specificity of our model can thus aid clinicians in reducing adverse pregnancy outcomes. In the future, we will convert the nomogram model into an electronic medical records system or mobile application for every pregnant woman to expand the potential value of this predictive model.

Abbreviations

AC: Abdominal circumference; AFI: Amniotic fluid index; APTD: Anteroposterior trunk diameter; AUC: Area under the curve; BMI: Body mass index; BPD: Biparietal diameter; DBP: Diastolic Blood Pressure; EFW: Estimating fetal weight; FL: Femur length; FPG: Fasting plasma glucose; GA: Gestational age; GDM: Gestational diabetes mellitus; GLU-1H: 1-hour glucose; GLU-2H: 2-hour glucose; GNUM: Gravidity; GWG: Gestational weight gain; HbA1c: Hemoglobin; HC: Head circumference; HDL: High-density lipoprotein; HL: Humerus length; IOM: Institute of Medicine; IQR: Interquartile ranges; LDL: Low-density lipoprotein; OGTT: Oral glucose tolerance test; ROC: Receiver operating characteristic; SBP: Systolic Blood Pressure; TC: Total cholesterol; TG: Triglycerides; TTD: Transverse trunk diameter; VIF: Variance inflation factor.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12884-022-04981-9.

Additional file 1: Supplemental Table. Meaning of all the 24 variables.

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Not applicable.

Authors' contributions

The study was designed by Cheng Weiwei, Xu Jie, Gao Jing, and Shi Huwei. Gao Jing, Chen Ruiyao, Lu Lu , Chen Chao , Luo Shuqing, and Yang Kaixiang were responsible for writing the manuscript. Chen Lei, Wang Ping, Chen Chao, Yang Sen, and Chen Jiayuan collected and sorted out the data. Xiao Zhongzhou and Shi Huwei performed the statistical analysis. Cheng Weiwei and Xu Jie reviewed and edited manuscripts. All authors read and approved the final manuscript.

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Availability of data and materials

The datasets analyzed during the current study are not publicly available due to the metadata containing information that could compromise the patients but are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

Our research strictly adheres to the Declaration of Helsinki. The life, health, privacy and dignity of the subjects are protected from the procedures of trial design, subject inclusion, data collection, data analysis, and result presentation in our research.

Ethical approval for the use of patient information was obtained from the Research Ethics Committee of International Peace Maternity and Child Health Hospital (GKLW2021–20). We confirmed that informed consent was waived by the Ethics Committee of International Peace Maternity and Child Health Hospital due to the review of the study.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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