Risk Prediction of Gestational Diabetes Mellitus In Women With Polycystic Ovary Syndrome Based On A Nomogram Model

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Research Article

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Abstract

Women with polycystic ovary syndrome are prone to develop gestational diabetes mellitus, a disease which may have significant impact on the postpartum health of both mother and infant. We performed a retrospective cohort study to develop and test a model that could predict gestational diabetes mellitus in the first trimester in women with polycystic ovary syndrome. Our study included 520 pregnant women who were referred to the obstetrics department between December 2017 and March 2020 with a diagnosis of polycystic ovary syndrome. Of these women, 171 were diagnosed with gestational diabetes mellitus in the second trimester. Univariate analysis revealed that in the first trimester, parity, family history of diabetes, age, body mass index (BMI), testosterone, low density lipoprotein cholesterol, triglyceride(TG), total cholesterol(TC), fasting plasma glucose(FPG), Hemoglobin A1c (HbA1C), diastolic blood pressure(DBP) and insulin levels were predictive factors of gestational diabetes mellitus ($P < 0.05$). Logistic analysis revealed that TG, age, HbA1C, Insulin, TC, BMI and family history of diabetes were independent risk factors for gestational diabetes mellitus. The area under the ROC curve of the gestational diabetes mellitus risk prediction model was 0.917 in this retrospective analysis, demonstrating the great ability to predict. The sensitivity and specificity of the prediction model were 0.814 and 0.871, respectively. The Hosmer–Lemeshow test also showed a good fit to the test.

Introduction

Gestational diabetes mellitus (GDM) is defined as glucose intolerance that develops or is first diagnosed after the first trimester of pregnancy and is not caused by type 1 or type 2 diabetes mellitus. GDM has long-lasting harmful effects on both the mother and infant [1]. Women with a history of GDM are almost ten times more likely to develop type 2 diabetes mellitus than those without [2]. Maternal hyperglycemia may also place infants at risk of autism spectrum disorders, obesity, hypertension, type 2 diabetes, hyperlipidemia, cardiovascular disease, deep vein thrombosis, and pulmonary embolism [3], and it may place young adults at risk of obesity with insulin resistance [4].

The incidence of gestational diabetes has always been high, and in recent years, due to advances in assisted reproductive technology and the fertility needs of older women, the incidence of gestational diabetes has increased. The global incidence of GDM ranges from 9% to 25%, while in China it ranges from 13.0% to 20.9% [5]. Over the last 10 years, the incidence in our hospital has been risen from 13.4% to 16.3%.

Polycystic ovary syndrome (PCOS) is a common disorder that affects 8% of reproductive-aged women [6, 7]. It is characterized by the presence of at least two of the following diagnostic criteria: 1) hyperandrogenism; 2) polycystic ovaries determined sonographically with 12 or more measured follicles of length between 2 and 9 mm; 3) an ovarian volume of 10 mL or more; and 4) infertility resulting from menstrual cycle disorders. The syndrome is also associated with various comorbidities that cause obesity, and have long-term sequelae such as insulin resistance and metabolic syndrome [8].

Women with PCOS have higher levels of insulin resistance, which predisposes them to developing GDM [9]. Based on extensive clinical research, the National Institutes of Health reported that PCOS is an independent risk factor for GDM. PCOS has become the most common endocrine and gynecological condition in Chinese women of childbearing age because of a shift from a traditional Chinese diet to a high-sugar, high-fat diet, and a delayed childbearing age [10].

Therefore, it is critical to identify pregnant women with PCOS who are at risk of developing GDM. The goal of this study was to identify GDM risk factors in women with PCOS to enable clinicians to undertake early prevention intervention measures effectively, and thus reduce the occurrence of GDM.

Patients And Methods

Inclusion and exclusion criteria for patients

This prospective cohort study used data from the medical records of 520 women with PCOS. The patients were between 7 and 12 weeks pregnant and have prenatal examinations in the Maternal and Child Health Hospital of Hunan Province between December 2017 and March 2020. Follow-up was performed regularly. Patients were included if they 1) were pregnant and had PCOS; 2) were older than 18 years; 3) intended to have prenatal examinations and deliver their babies at our hospital; and 4) had an oral glucose tolerance test at 24–28 weeks of pregnancy. Patients were excluded if they had 1) pregestational diabetes mellitus; 2) multiple pregnancy; or 3) cardiovascular, respiratory, thyroid, liver, kidney, or any other diseases that might affect the research outcomes. The study was approved by the ethics committee of Maternal and Child Health Hospital of Hunan Province.

Data collection

Standard sphygmomanometry was used to measure blood pressure in each patient. The measurement unit of Weight and Height were recorded and rounded to the nearest 0.1 kg and 0.1 cm, respectively. BMI was counted by dividing the weight by the square of height. After at least 8 hours of fasting in the morning, venous blood samples were collected., TG, TC, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), FBG, HbA1C and testosterone levels were measured.

Patients with a BMI of between 24.0 and 27.9 kg/m² were defined as “overweight”, while those with a BMI of more than 28.0 kg/m² were defined as “obese” [11]. Participants were described as hypertensive if their systolic blood pressure was ≥ 140 mmHg and/or their diastolic blood pressure was ≥ 90 mm Hg. Dyslipidemia was defined as having a total cholesterol level of ≥ 6.22 mmol/L, TG ≥ 2.26 mmol/L, HDL-C < 1.04 mmol/L, or LDL-C ≥ 4.14 mmol/L, according to the criteria presented in the Chinese Guidelines for the Prevention and Treatment of Dyslipidemia in Adults [12].

GDM was screened for by performing an oral glucose tolerance test on patients during weeks 24–28 of pregnancy: patients included in the study fasted for 8 to 12 hours overnight, and the following morning drank 250–300 mL water with 75g glucose powder dissolved in 5 minute period. Their venous blood was

measured before the examination, and then again after 1 and 2 hours. The diagnostic standard used for gestational diabetes was that of abide by the International Association of Diabetes and Pregnancy Study Group (IADPSG)[13]. Patients were diagnosed with GDM if they blood glucose level reached any one of it : fasting blood glucose level ≥ 5.1 mmol/L, blood glucose at 1 hour after glucose intake ≥ 10.0 mmol/L, or blood glucose at 2 hours after glucose intake ≥ 8.5 mmol/L.

Statistical analysis

Descriptive statistic analyses were performed to analysis 520 participants using IBM SPSS Statistics for Windows, Version 20.0 (Armonk, NY: IBM Corp.). All continuous variables were summarized as median (interquartile range), and categorical variables were expressed as frequency (n) and proportions (%). The chi-square and Wilcoxon tests were used to detect statistical significance.

The nomogram model was developed and implemented in three steps. First, using nonzero coefficients in the least absolute shrinkage and selection operator (LASSO) regression model, we identified independent predictive features [14]. Second, the selected variate from the LASSO regression model were identified to construct the multivariate logistic regression model, and selected the variables with statistical significance to establish the nomogram model[15]. Third, the nomogram's discrimination and calibration were evaluated using calibration curve plots, the area under the receiver (AUC), and Harrell's concordance index (C-index), respectively. The statistical package R was used to generate the nomogram model (<http://www.R-project.org>).

Result

Baseline characteristics

A total of 520 pregnant women were included in this study. The mean age of the participants was 29 years. The proportion of overweight and obese participants in the GDM group was higher than that in the non-GDM group ($p < 0.05$). In addition, the rate of family history of diabetes in the GDM group was significantly higher than that in the non-GDM group ($p < 0.05$), as were the biochemical indices levels ($p < 0.05$). There were no significant differences in systolic and diastolic blood pressure between the two groups. (Table 1)

Univariate and multivariate analysis

Optimal lambda have selected Eleven variables with nonzero coefficients(Figure 1-1). Based on one standard error criteria, the partial likelihood deviance (binomial deviance) curve was plotted vs $\log(\lambda)$ and dotted vertical lines were constructed. (Figure 1-2). According to the multivariate logistic regression model, we defined the independent risk factors for GDM to include TG, age, HbA1C, Insulin, TC, BMI and family history of diabetes (Table 2).

Nomogram and evaluation of prediction model for gestational diabetes mellitus

A nomogram of the GDM risk prediction model for pregnant women was created based on the independent risk factors for GDM before pregnancy (Figure 2). To measure the frequency of occurrence of GDM, the cumulative GDM score in the first trimester was determined based on the corresponding score of each predictor factor. The AUC of the GDM risk prediction model was 0.917, indicating a strong classification ability. The sensitivity and accuracy of the validation model were 0.814 and 0.871, respectively (Figure 3-1). The calibration of the prediction model was evaluated using the Hosmer–Lemeshow goodness of fit test, and a calibration curve was obtained. (Figure 3-2).

Discussion

Insulin resistance is a key characteristic of PCOS, and women with both PCOS and GDM are at high risk of developing type 2 diabetes mellitus. Type 2 diabetes exhibits insulin resistance and elevated blood glucose levels, which may promote pathological harm in PCOS patients [16]. It is generally accepted that Fatty acids (FAs) play a major role in the development of insulin resistance [17]. In our study, BMI is the most significant index to indicate the occurrence of GDM in pregnant women with PCOS. Consequently, it would be useful to have a clinical index that could quantify the differences between women with PCOS who have GDM and those who do not have GDM. Other factors associated with an increased risk for GDM are age, number of pregnancies, LDL-C level, and history of diabetes mellitus in first degree relatives [18]. In this study, the nomogram model that we developed could effectively predict the development of GDM in women with PCOS based on TG, age, BMI, and family history of diabetes. The sensitivity and specificity of this model were 81.4% and 87.1%, respectively. 95% of overweight women with PCOS has insulin resistance [19], supporting the outcome in our nomogram model that BMI was the most relevant factor to predict GDM. N.S. Kakoly pointed out that obese women with PCOS patients are more likely to develop insulin resistance or type 2 diabetes mellitus later, and this results were independent of race [20]. According to previous studies, the most important risk factor for GDM is being overweight or obese prior to pregnancy (BMI 25 kg/m² or above) [21]. This was not affected by the geographical distribution, parity, or a history of GDM. However, a previously reported clinical model that used BMI as the independent factor to screen for GDM found that the outcomes varied widely by race: GDM was found in more than 76% of African-Americans, 58% of Latinas, and 46% of Caucasians, but only 25% of Asians (p=0.001) [22]. A meta-analysis estimated that the risk of developing GDM increases with weight gain and is not affected by location; compared to the control group, the risk of developing GDM is approximately two, four, and eight times higher in overweight, obese, and extremely obese women, respectively [23].

Dyslipidemia, which is associated with diabetes mellitus, is the main risk factor for the development of cardiovascular disease. Diabetes-related lipid changes are attributed to an increased free fatty acid flux caused by insulin resistance. In our study, the logistic analysis found that the serum level of triglyceride is the independent factor for GDM prediction. However, in our nomogram model, the varies of LG almost have no score, it may attributed to the different value units and classification of grading methods. Several studies have reported that dyslipidemia in the first trimester of pregnancy is related to the development of GDM [24, 25]. Lean or obese women with higher TG concentrations have an increased risk of developing GDM, whereas lean women with high HDL-C levels do not. This statement is consistent with O'Malley,

who stated that maternal obesity mediates the epidemiological relationship between GDM and dyslipidemia, and only women with GDM who were obese had higher TG and lower HDL-C levels and a higher TG:HDL-C ratio than those without GDM [26]. A retrospective study indicated that women with GDM are more likely to have hyperlipidemia postpartum, particularly dyslipidemia defined by TG [27]. Hyperinsulinemia is usually concomitant with low HDL cholesterol and high LDL-cholesterol levels. The increased release of free fatty acids from insulin-resistant fat cells may be the cause of these characteristics [28].

Hemoglobin A1c(HbA1C) is the sensitive factor to indicate insulin resistance[29] and capable as an indicator to predict cardiovascular disease for Nondiabetic Adults[30]. There is an amounting evidence indicates that fasting plasma glucose (FPG) can be an indicator to prognosticate the risk rate of glucose abnormality during pregnant. An increasing number of researchers have studied the relationship between fasting plasma glucose (FPG) levels in early pregnancy and the occurrence of adverse pregnancy outcomes. They found that testing FPG levels in the first trimester is feasible, cost-effective, and may avoid the emesis and discomfort caused by the oral glucose tolerance tests. Agarwal et al. found that when serum levels of FPG <4.4 mmol/L, the sensitivity to exclude GDM was 94.7% when serum levels of FPG $\geq$ 5.3 mmol/L and the specificity for diagnosing GDM was 94%. FPG is therefore a potentially effective screening method that releases pregnant women from the need to undergo the oral glucose tolerance test, and the omission diagnostic rate is 5.8% [31].

The cult value of using FPG to define risk factors for GDM in early pregnancy is still an issue, however. Zhu et al. recommended that an FPG threshold of 5.7 mmol/L can identify 60% of pregnant women with abnormal oral glucose tolerance test at 24–28 weeks [32]. Based on several studies, E. Cosson et al. recommended that an FPG level between 5.6 and 6.9 mmol/L be used as the threshold values as the risk of giving birth to a child with macrosomia increases with higher FPG levels [33]. In consideration of the potential clinical impact of HbA1C and FPG, we incorporate these two factors into nomogram model even while the logistic analysis did not show significant difference.

Kousta et al. give evidence that even if pregnant women with GDM had normal fasting blood glucose levels in postpartum, their β -cell function and insulin response to glucose still have adverse effects. Recently, much attention has been paid to the role of other hormone factors in the development of GDM. The prediction indices included Follicle-Stimulating Hormone(FSH), Luteinizing Hormone(LH), and Thyroid peroxidase antibody (TPA). The prediction time includes the first and second trimester of pregnancy, but there is no simple and practical prediction index with high sensitivity and specificity. Therefore, we should consider other parameters for predicting GDM in women with PCOS.

We conclude that TG, FPG, BMI, HbA1C, and family history were effective risk predictors for GDM in women with PCOS in the first trimester. In particular, women with PCOS and overweight are at a substantial risk for GDM: the nomogram model shows that when BMI increased to overweight, the prediction almost reached to 85%. These women should be identified as being at high risk and be counseled before, or during the early stages of pregnancy to implement effective therapeutic strategies. Our analysis

was limited because all data used for analysis were obtained from a single site, which may have impaired our analysis. A multicenter validation using a large number of patients may produce results that are more generalizable to the broader population.

Declarations

Availability of data and materials

Not applicable.

Competing interests

The authors declare that they have no competing interests.

Ethics statement

The study was approved by the ethics committee of Maternal and Child Health Hospital of Hunan Province and informed consent was obtained from all participants. All methods carried out in this study involving human participants were in accordance with the of the international and/or national committee and With the ethical standards of the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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Authors' contributions

All authors read and approved the final manuscript.

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References

1. Buchanan, T.A. and A.H. Xiang, *Gestational diabetes mellitus*. J Clin Invest, 2005. **115**(3): p. 485-91.
2. Vounzoulaki, E., et al., *Progression to type 2 diabetes in women with a known history of gestational diabetes: systematic review and meta-analysis*. BMJ, 2020. **369**: p. m1361.
3. Xiang, A.H., et al., *Association of maternal diabetes with autism in offspring*. JAMA, 2015. **313**(14): p. 1425-34.

4. Hillier, T.A., et al., *Childhood obesity and metabolic imprinting: the ongoing effects of maternal hyperglycemia*. Diabetes Care, 2007. **30**(9): p. 2287-92.
5. Lee, K.W., et al., *Prevalence and risk factors of gestational diabetes mellitus in Asia: a systematic review and meta-analysis*. BMC Pregnancy Childbirth, 2018. **18**(1): p. 494.
6. Kakoly, N.S., et al., *Group-based developmental BMI trajectories, polycystic ovary syndrome, and gestational diabetes: a community-based longitudinal study*. BMC Med, 2017. **15**(1): p. 195.
7. Mustaniemi, S., et al., *Polycystic ovary syndrome and risk factors for gestational diabetes*. Endocr Connect, 2018. **7**(7): p. 859-869.
8. Rotterdam, E.A.-S.P.C.W.G., *Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome*. Fertil Steril, 2004. **81**(1): p. 19-25.
9. Goodarzi, M.O., et al., *Polycystic ovary syndrome: etiology, pathogenesis and diagnosis*. Nat Rev Endocrinol, 2011. **7**(4): p. 219-31.
10. Mills, G., et al., *Polycystic ovary syndrome as an independent risk factor for gestational diabetes and hypertensive disorders of pregnancy: a population-based study on 9.1 million pregnancies*. Hum Reprod, 2020. **35**(7): p. 1666-1674.
11. F. Y. Zhai, L.W.Z., C. R. Wang et al., *Validation of lipids on body mass index reference recommended by obesity working Group, international life science association of China*. Zhonghua Liu Xing Bing Xue Za Zhi, 2004. **25**(2): p. 2.
12. Adults, J.C.f.D.C.G.o.Pa.T.o.D.i., *Chinese guidelines on prevention and treatment of dyslipidemia in adults*. Zhonghua Xin Xue Guan Bing Za Zhi, 2007. **35**(5): p. 29.
13. International Association of, D., et al., *International association of diabetes and pregnancy study groups recommendations on the diagnosis and classification of hyperglycemia in pregnancy*. Diabetes Care, 2010. **33**(3): p. 676-82.
14. Alhamzawi, R. and H.T.M. Ali, *The Bayesian adaptive lasso regression*. Math Biosci, 2018. **303**: p. 75-82.
15. Balachandran, V.P., et al., *Nomograms in oncology: more than meets the eye*. The Lancet Oncology, 2015. **16**(4): p. e173-e180.
16. Stepto, N.K., et al., *Women with polycystic ovary syndrome have intrinsic insulin resistance on euglycaemic-hyperinsulinaemic clamp*. Hum Reprod, 2013. **28**(3): p. 777-84.
17. Shetty, S.S. and S. Kumari, *Fatty acids and their role in type-2 diabetes (Review)*. Exp Ther Med, 2021. **22**(1): p. 706.

18. Li, X., et al., *Association between thyroid function and risk of gestational diabetes mellitus in assisted pregnancies: A retrospective cohort study*. Diabetes Res Clin Pract, 2021. **171**: p. 108590.
19. Stepto NK, C.S., Joham AE, Hutchison SK, Harrison CL, Goldstein RF, Teede *Women with polycystic ovary syndrome have intrinsic insulin resistance on euglycaemic-hyperinsulaemic clamp*. Hum Reprod. Hum Reprod, 2013. **28**(3).
20. Kakoly, N.S., et al., *Ethnicity, obesity and the prevalence of impaired glucose tolerance and type 2 diabetes in PCOS: a systematic review and meta-regression*. Hum Reprod Update, 2018. **24**(4): p. 455-467.
21. Zhang, C. and Y. Ning, *Effect of dietary and lifestyle factors on the risk of gestational diabetes: review of epidemiologic evidence*. Am J Clin Nutr, 2011. **94**(6 Suppl): p. 1975S-1979S.
22. Shah, A., et al., *The association between body mass index and gestational diabetes mellitus varies by race/ethnicity*. Am J Perinatol, 2011. **28**(7): p. 515-20.
23. Chu, S.Y., et al., *Maternal obesity and risk of gestational diabetes mellitus*. Diabetes Care, 2007. **30**(8): p. 2070-6.
24. Li, G., et al., *Early Pregnancy Maternal Lipid Profiles and the Risk of Gestational Diabetes Mellitus Stratified for Body Mass Index*. Reprod Sci, 2015. **22**(6): p. 712-7.
25. Liu, P.J., et al., *The Predictive Ability of Two Triglyceride-Associated Indices for Gestational Diabetes Mellitus and Large for Gestational Age Infant Among Chinese Pregnancies: A Preliminary Cohort Study*. Diabetes Metab Syndr Obes, 2020. **13**: p. 2025-2035.
26. O'Malley, E.G., et al., *Maternal obesity and dyslipidemia associated with gestational diabetes mellitus (GDM)*. Eur J Obstet Gynecol Reprod Biol, 2020. **246**: p. 67-71.
27. Chodick, G., et al., *Gestational diabetes and long-term risk for dyslipidemia: a population-based historical cohort study*. BMJ Open Diabetes Res Care, 2020. **8**(1).
28. Mooradian, A.D., *Dyslipidemia in type 2 diabetes mellitus*. Nat Clin Pract Endocrinol Metab, 2009. **5**(3): p. 150-9.
29. Kanat, M., et al., *The relationship between {beta}-cell function and glycated hemoglobin: results from the veterans administration genetic epidemiology study*. Diabetes Care, 2011. **34**(4): p. 1006-10.
30. Selvin, E., et al., *Glycated hemoglobin, diabetes, and cardiovascular risk in nondiabetic adults*. N Engl J Med, 2010. **362**(9): p. 800-11.
31. Agarwal, M.M., et al., *Gestational diabetes: an evaluation of serum fructosamine as a screening test in a high-risk population*. Gynecol Obstet Invest, 2011. **71**(3): p. 207-12.

32. Zhu, W.W., et al., *Evaluation of the value of fasting plasma glucose in the first prenatal visit to diagnose gestational diabetes mellitus in china*. Diabetes Care, 2013. **36**(3): p. 586-90.
33. Cosson, E., L. Carbillon, and P. Valensi, *High Fasting Plasma Glucose during Early Pregnancy: A Review about Early Gestational Diabetes Mellitus*. J Diabetes Res, 2017. **2017**: p. 8921712.

Tables

Table1 Comparison of pre-pregnancy laboratory test and general conditions of PCOS women between the GDM and non-GDM groups

Variable	GDM group (n=171)	Non-GDM group (n=349)	P-value
Age(years)			
<35	151	325	0.064
≥35	20	24	
BMI(kg/m2)			
<24	69	301	P<0.05
24 ≤n<28	94	47	
≥28	8	1	
Parity			
0	98	260	P<0.05
≥1	73	89	
Family history with diabetes			
No	123	331	P<0.05
Yes	48	18	
Testosterone (mmol/L)	1.31(0.80,1.79)	1.49(0.92,2.01)	0.022
LDL(mmol/L)	3.06(1.28,3.34)	1.59(0.99,2.06)	0.000
HDL(mmol/L)	1.17(0.99,1.43)	1.09(0.85,1.50)	0.120
TG(mmol/L)	1.87(1.14,2.99)	1.52(1.02,2.17)	0.000
TC(mmol/L)	5.02(4.52,5.29)	3.22(2.32,3.90)	0.000
FPG(mmol/L)	4.87(4.34,5.26)	4.46(4.20,4.71)	0.000
Fasting insulin(mmol/L)	15.30(11.08–23.37)	9.98(7.16,13.66)	0.000
HbA1c(%)	5.14(4.88,5.49)	4.63(4.36,4.92)	0.000
SBP(mmHg)	117(111,125)	118(112,123)	0.560
DBP(mmHg)	75(69,81)	76(69,82)	0.477

MI body mass index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *FPG* fasting plasma glucose, *HbA1c* glycosylated hemoglobin, *TC* total cholesterol, *TG* triglycerides, *LDL-C* low-density lipoprotein cholesterol, *HDL-C* high-density lipoprotein cholesterol

Table 2 Multivariate logistic regression to predict risk of GDM in PCOS women

	β Coefficient	Wald test	P	OR(95%CI)
FPG	0.679	1.936	0.052	1.972 1.000-3.980
HbA1C	2.951	6.694	0.000	19.130 8.380-47.511
TG	-0.474	-2.654	0.007	0.622 0.432-0.874
TC	1.582	5.803	0.000	4.865 (2.950-8.627)
BMI	0.212	2.484	0.013	1.236 1.048-1.468
Family history	1.599	3.033	0.002	4.949 1.817-14.448
Age	0.140	2.425	0.015	1.150 1.029-1.292

FBG fasting blood glucose, *HbA1c* glycosylated hemoglobin, *TG* triglycerides, *TC* total cholesterol *BMI*/body mass index

Figures

1-1

1-2

Figure 1

Optimal lambda have selected Eleven variables with nonzero coefficients(Figure 1-1). Based on one standard error criteria, the partial likelihood deviance (binomial deviance) curve was plotted vs log(lambda) and dotted vertical lines were constructed. (Figure 1-2)

Figure 2

GDM nomogram for the first trimester. The risk of predicting the occurrence of GDM is quantified as the number of points marked on the axis, the score determined by each variable axis is the number corresponding to the value vertical on the total points scale, and projected the sum of all variables onto the bottom axis, yielding a personalized GDM risk for each woman with PCOS.

3-1

3-2

Figure 3

ROC curves showing the precision of the GDM nomogram in patients. The average predictive precision of the nomogram for GDM was 0.917, and the validation model's sensitivity and specificity were 0.814 and 0.871, respectively.