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Nomogram model based on magnetic resonance imaging and clinical factors for prenatal diagnosis of placenta accreta spectrum disorders

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Abstract

This study aimed to develop a magnetic resonance imaging (MRI) and clinical factor-based nomogram for prenatal diagnosis of placenta accreta spectrum disorders (PAS). Clinical and MRI data from pregnant women were retrospectively analyzed and split into training (70%) and validation (30%) sets. Binary logistic regression identified age, placenta previa, T2 dark band, placental/uterine bulge, loss of T2 hypointense interface, myometrial thinning, and abnormal placental bed vessels as significant predictors of PAS. A nomogram incorporating these factors demonstrated strong diagnostic performance via ROC analysis (AUC >0.8), calibration curves, and decision curve analysis (DCA) in both training and validation sets. The model showed high accuracy and clinical utility, supporting its use in prenatal PAS diagnosis *(Afr J Reprod Health 2025; 29 [10]: 155-166)*

Keywords: MRI; placenta accrete; spectrum disorders; prenatal diagnosis; Nomogram model

Résumé

Cette étude visait à développer une imagerie par résonance magnétique (irm) et un nomogramme basé sur des facteurs cliniques pour le diagnostic prénatal des troubles du spectre placenta accreta (apm). Les données cliniques et d'irm des femmes enceintes ont été analysées rétrospectivement et divisées en séries de formation (70%) et de validation (30%). La régression logistique binaire a identifié l'âge, le placenta previa, la bande noire T2, le renflement placentaire/utérin, la perte de l'interface hypointense T2, l'amincissement myomètre et les vaisseaux du lit placentaire anormaux comme des prédicteurs significatifs du PAS. Un nomogramme intégrant ces facteurs A démontré de solides performances diagnostiques par l'analyse ROC (AUC > 0,8), les courbes d'étalonnage et l'analyse de la courbe de décision (DCA) dans les ensembles de formation et de validation. Le modèle a montré une grande précision et une utilité clinique, soutenant son utilisation dans le diagnostic du sap prénatal. *(Afr J Reprod Health 2025; 29 [10]: 155-166).*

Mots-clés: irm; troubles du spectre placenta accrete; diagnostic prenatal; modèle nomogramme

Introduction

Placenta accreta spectrum disorders (PAS) refer to a group of diseases caused by poor development of the basal decidua and varying degrees of invasion of placental villi into the myometrium[1]. According to the depth of invasion of the placental villi, PAS can be classified as placenta accreta, placenta increta, and placenta percreta[2]. The higher the degree of placental implantation, the more severe the adverse pregnancy outcomes, such as postpartum bleeding[3]. Patients with PAS often lack clinical symptoms or have nonspecific symptoms before childbirth, making it challenging to diagnose solely based on clinical manifestations. This is because early-stage placenta accreta may not cause obvious symptoms, or its symptoms may resemble other pregnancy complications, posing a certain degree of diagnostic challenge[4-6]. It is an important precondition to alleviate severe complications and ensure the safety of mother and child to accurately identify PAS pregnant women antenatally, refer them to institutions with multidisciplinary rescue capabilities for delivery, and formulate sufficient and comprehensive rescue plans based on classification and implantation degree.

Ultrasonography is the preferred method for prenatal diagnosis of PAS, especially in assessing the relationship between the placenta and the uterine wall and identifying the type of placenta accreta.

Color Doppler ultrasound can further evaluate the blood flow of the placenta and improve the accuracy of the diagnosis[7,8]. However, when the placenta is located in the posterior wall or fundus of the uterus, influenced by amniotic fluid, maternal obesity, or excessive intestinal gas, the detection rate of PAS by ultrasound may decrease or even be difficult to detect. On the other hand, MRI is not affected by the mother's body shape, intraluminal gas, or placental location, and has an advantage in evaluating the depth of trophoblastic invasion, location of placental attachment, and the relationship with adjacent structures[9]. This study aimed to explore the value of nomogram model based on MRI and clinical factors for prenatal diagnosis of PAS.

Methods

Patients

Using 228 pregnant women who visited Affiliated Maternity and Child Health Care Hospital of Nantong University from January 2019 to December 2022 as subjects, we retrospectively analyzed their clinical data and prenatal MRI imaging data. The inclusion criteria were: gestational age ≥21 weeks; singleton pregnancies with suspected PAS; complete clinical records; complete records of postoperative diagnosis, and/or pathological diagnosis. The exclusion criteria were: pregnancies with severe artifacts and poor image quality due to fetal movement or other reasons in MR images; pregnancy outcomes containing stillbirth. Randomly divide the data into a training set and a validation set according to the ratio of 7:3.

Pregnant women were divided into PAS group and non-PAS group. The patients' information are presented in Table1.

Instruments and examination methods

The imaging examination was conducted using a Siemens 1.5 Tesla magnetic resonance scanner (model: Avanto Tim-class). The patients were positioned in the supine position during scanning, and the imaging range extended from the upper edge of the placenta to the symphysis pubis. The examination sequences included half-Fourier acquisition single-shot turbo spin-echo T2-weighted imaging (HASTE T2WI) and True fast imaging with steady-state precession (True-FISP). The scan parameters are shown in Table2.

Observed indicators

Clinical data: Age, gestational age, number of pregnancies, number of abortions, number of spontaneous births, number of cesarean deliveries, Gestational Age, Gestational hypertension, gestational diabetes mellitus, vaginal bleeding during pregnancy, Fetal lie, placenta previa (PP).

MR imaging features: T2 Dark Band (T2DB), placenta and uterine bulge (PUB), Disappearance of the T2 hypointense interface between placenta and uterus (DT2HIPU), thinning of uterine muscular layer (TUML), abnormal blood vessels in placental bed (ABVPB), uneven signaling in the placenta, asymmetrical thickness and morphology of the placenta, ischemic infarction of the placenta, fetal blood vessels within the placenta [It is defined as those that run from the placental fetus or umbilical cord (low signal on HASTE, True FISP, etc., or high signals), penetrate into the placental tissue (depth > 10mm) and can reach the myometrium through the placenta. The diameter of the blood vessels refers to the measurement of the widest part of the blood vessels.

Statistics processing

The measurement data obtained from this study were validated by the Kolmogorov-Smirnov D test to meet a normal distribution and are presented as mean ± standard deviation. Those that do not meet a normal distribution are expressed as median (interquartile range). Independent t-tests and Mann-Whitney U rank-sum test were used for intergroup comparisons, respectively. The count data was expressed as n (%), and used chi-square test to do group comparisons. Binary unconditional logistic regression was applied to identify the related factors for PAS, and to construct Nomogram model based on these related factors. The receiver operating characteristic curve (ROC), calibration curve and decision curve analysis (DCA) were used to access the clinical effectiveness of Nomogram model for PAS. Statistics processing was conducted using R language R 4.2.3 statistical software, along with Zstats v0.90 (www.medsta.cn/software).

Table1: Overall clinical data and imaging treatment status

Variables	Total (n = 228)	Non-PAS (n = 102)	PAS (n = 126)	Statistic	*P*
Age, Mean ± SD	31.7 ± 4.1	30.8 ± 4.0	32.4± 4.1	t=-3.09	0.002
Number of Pregnancies, M (Q_1, Q_3)	2.0 (1.0, 3.0)	2.0 (0.0, 3.0)	2.0 (1.0, 3.0)	Z=-1.23	0.218
Number of Abortions, M (Q_1, Q_3)	1.0 (0.0, 2.0)	1.0 (0.0, 2.0)	1.0 (0.0, 2.0)	Z=-0.87	0.387
Fetal Blood Vessels within Placenta, M (Q_1, Q_3)	0.0 (0.0, 2.5)	0.0 (0.0, 0.0)	0.0(0.0, 3.2)	Z=-1.46	0.144
Number of Spontaneous Births, M (Q_1, Q_3)	0.0 (0.0, 0.0)	0.0 (0.0, 0.0)	0.0 (0.0, 0.0)	Z=-0.77	0.439
Number of Cesarean Deliveries, M (Q_1, Q_3)	0.0 (0.0, 1.0)	0.0 (0.0, 1.0)	1.0 (0.0, 1.0)	Z=-1.10	0.271
Gestational Age, M (Q_1, Q_3)	35.0 (33.0, 37.0)	36.0 (32.0, 37.0)	35.0 (33.0, 37.0)	Z=-0.18	0.860
Gestational Hypertension, n(%)				χ^2=0.22	0.639
No	215 (94.3)	97 (95.1)	118 (93.7)		
Yes	13 (5.7)	5 (4.9)	8 (6.4)		
Gestational Diabetes Mellitus, n(%)				χ^2=1.04	0.308
No	162 (71.1)	69 (67.7)	93 (73.8)		
Yes	66 (29.0)	33 (32.4)	33 (26.2)		
Vaginal Bleeding During Pregnancy, n(%)				χ^2=3.95	0.047
No	176 (77.2)	85 (83.3)	91 (72.2)		
Yes	52 (22.8)	17 (16.7)	35 (27.8)		
Fetal Lie, n(%)				χ^2=7.64	0.006
Breech presentation	40 (17.5)	10 (9.8)	30 (23.8)		
Vertex position	188 (82.5)	92 (90.2)	96 (76.2)		
PP, n(%)				χ^2=34.03	<.001
complete PP	107 (46.9)	28 (27.5)	79 (62.7)		
low-lying PP	11 (4.8)	6 (5.9)	5 (4.0)		
marginal PP	30 (13.2)	13 (12.8)	17 (13.5)		
No	80 (35.1)	55 (53.9)	25 (19.8)		
T2DB, n(%)				χ^2=13.66	<.001
No	189 (82.9)	95 (93.1)	94 (74.6)		
Yes	39 (17.1)	7 (6.9)	32 (25.4)		
PUB, n(%)				χ^2=11.86	<.001
No	194 (85.1)	96 (94.1)	98 (77.8)		
Yes	34 (14.9)	6 (5.9)	28 (22.2)		
DT2HIPU, n(%)				χ^2=28.19	<.001
No	166 (72.8)	92 (90.2)	74 (58.7)		
Yes	62 (27.2)	10 (9.8)	52 (41.3)		
TUML, n(%)				χ^2=18.25	<.001
No	200 (87.7)	100 (98.0)	100 (79.4)		
Yes	28 (12.3)	2 (2.0)	26 (20.6)		
ABVPB, n(%)				χ^2=26.65	<.001
No	176 (77.2)	95 (93.1)	81 (64.3)		
Yes	52 (22.8)	7 (6.9)	45 (35.7)		
Uneven Signaling in Placenta, n(%)				χ^2=3.03	0.082
No	198 (86.8)	93 (91.2)	105 (83.3)		
Yes	30 (13.2)	9 (8.8)	21 (16.7)		
Asymmetrical Thickness and Morphology of Placenta, n(%)				χ^2=1.58	0.209
No	214 (93.9)	98 (96.1)	116 (92.1)		
Yes	14 (6.1)	4 (3.9)	10 (7.9)		
Ischemic Infarction of Placenta, n(%)				χ^2=0.17	0.683
No	214 (93.9)	95 (93.1)	119 (94.4)		
Yes	14 (6.1)	7 (6.9)	7 (5.6)		

Note: PAS: placenta accreta spectrum disorders; PP: placenta previa; T2DB: T2 Dark Band; PUB: placenta and uterine bulge; DT2HIPU: Disappearance of the T2 hypointense interface between placenta and uterus; TUML: thinning of the uterine muscular layer; ABVPB: abnormal blood vessels in placental bed.

Table 2: Magnetic resonance imaging sequence parameters

Sequence	TR* (ms)	TE* (ms)	Slice Thickness(mm)	Slice Gap(mm)	FOV*(mm)	Scan Direction
HASTE T2WI	1100	108	4.0	1.2	380×380	Axial Plane, Sagittal Plane, Coronal Plane,
True-FISP	3.87	1.68	4.0	1.2	380×380	Sagittal Plane

A significance level of P<0.05 was considered statistically significant.

Ethical consideration

This study was approved by the Ethical Committee of Affiliated Maternity and Child Health Care Hospital of Nantong University (Y2020001). All patients had given informed consent.

Results

Overall clinical data and imaging treatment status

Among 228 pregnant women, PAS was found in 126 cases, with an incidence rate of 55.26%. Compared with the non-PAS group, the PAS group had older women, a higher incidence rate of vaginal bleeding during pregnancy, a higher proportion of vertex positions, a higher incidence rate of complete placenta praevia, and a higher proportion of image characteristics such as T2DB, PUB, DT2HIPU, TUML, ABVPB (P<0.05) (Table-1). The data were randomly divide into a training set and a validation set according to the ratio of 7:3. There was no significant difference between the training set and validation set data (P>0.05) (Table3), which indicate that the validation set and the training set are homogeneous.

Comparison of clinical data and imaging data in the training set

In the training set, compared with the non-PAS group, the PAS group had older women, a higher incidence rate of complete PP, and a higher proportion of image characteristics such as T2DB, PUB, DT2HIPU, TUML, ABVPB (P<0.05) (Table4).

Related factors for PAS in training set

For the non-PAS group, assign the value 0, and for the PAS group, assign the value 1. Then, input age, PP, T2DB, PUB, DT2HIPU, TUML, and ABVPB into the binary logistic regression equation separately. The results show that they are related factors for PAS (P<0.05). When these indicators are entered into the binary logistic regression equation together, the results show that age, PP, and DT2HIPU are independent related factors for PAS (P<0.05) (Table5).

The evaluation of Nomogram model on its clinical effectiveness in training set

By applying a binary Logistic regression equation, we construct a Nomogram model based on related factors such as age, PP, T2DB, PUB, DT2HIPU, TUML, and ABVPB (Figur-1A), when total point was 60-165 in this model, risk for PAS was 0.10-0.90. ROC analysis shows that area under the curve (AUC) of this model for the diagnosis of PAS is 0.83, 95% CI: 0.77-0.89, with Accuracy, Sensitivity, and Specificity of 0.75, 0.78, and 0.72, respectively (Table6) (Figure-1B). This model demonstrates good discriminative ability in the diagnosis of PAS. The result of Hosmer-Lemeshow goodness-of-fit test in calibration curve analysis shows $\chi 2=1.82$, P=0.99, the calibration curve has good agreement with the ideal curve (Figure-1C).

Table 3: Comparison of clinical data and dmaging Data between training set and validation set

Variables	Total (n = 228)	test (n = 69)	train (n = 159)	Statistic	*P*
Age, Mean ± SD	31.7± 4.1	32.1 ± 4.0	31.5 ± 4.2	t=0.95	0.342
Number of Pregnancies, M (Q_1, Q_3)	2.0 (1.0, 3.0)	2.0 (1.0, 3.0)	2.0 (1.0, 3.0)	Z=-1.02	0.309
Number of Abortions, M (Q_1, Q_3)	1.0 (0.0, 2.0)	1.0 (0.0, 2.0)	1.0 (0.0, 2.0)	Z=-0.57	0.572
Fetal Blood Vessels within Placenta, M (Q_1, Q_3)	0.0 (0.0, 2.5)	0.0 (0.0, 0.0)	0.0 (0.0, 2.5)	Z=-0.10	0.920
Number of Spontaneous Births, M (Q_1, Q_3)	0.0 (0.0, 0.0)	0.0 (0.0, 0.0)	0.0 (0.0, 0.0)	Z=-0.79	0.431
Number of Cesarean Deliveries, M (Q_1, Q_3)	0.0 (0.0, 1.0)	1.0 (0.0, 1.0)	0.0 (0.0, 1.0)	Z=-1.25	0.211
Gestational Age, M (Q_1, Q_3)	35.0 (33.0, 37.0)	36.0 (32.0, 37.0)	35.0 (33.0, 37.0)	Z=-0.18	0.860
Gestational Hypertension, n(%)				χ^2=0.07	0.787
No	215 (94.3)	66 (95.7)	149 (93.7)		
Yes	13 (5.7)	3 (4.4)	10 (6.3)		
Gestational Diabetes Mellitus, n(%)				χ^2=0.41	0.520
No	162 (71.1)	47 (68.1)	115 (72.3)		
Yes	66 (29.0)	22 (31.9)	44 (27.7)		
Vaginal Bleeding During Pregnancy, n(%)				χ^2=0.19	0.664
No	176 (77.2)	52 (75.4)	124 (78.0)		
Yes	52 (22.8)	17 (24.6)	35 (22.0)		
Fetal Lie, n(%)				χ^2=1.20	0.273
Breech presentation	40 (17.5)	15 (21.7)	25 (15.7)		
Vertex position	188 (82.5)	54 (78.3)	134 (84.3)		
PP, n(%)				χ^2=1.29	0.731
complete PP	107 (46.9)	31 (44.9)	76 (47.8)		
low-lying PP	11 (4.8)	5 (7.3)	6 (3.8)		
marginal PP	30 (13.2)	9 (13.0)	21 (13.2)		
No	80 (35.1)	24 (34.8)	56 (35.2)		
T2DB, n(%)				χ^2=1.99	0.158
No	102 (44.7)	26 (37.7)	76 (47.8)		
Yes	126 (55.3)	43 (62.3)	83 (52.2)		
PUB, n(%)				χ^2=0.01	0.940
No	189 (82.9)	57 (82.6)	132 (83.0)		
Yes	39 (17.1)	12 (17.4)	27 (17.0)		
DT2HIPU, n(%)				χ^2=0.27	0.602
No	194 (85.1)	60 (87.0)	134 (84.3)		
Yes	34 (14.9)	9 (13.0)	25 (15.7)		
TUML, n(%)				χ^2=0.01	0.939
No	166 (72.8)	50 (72.5)	116 (73.0)		
Yes	62 (27.2)	19 (27.5)	43 (27.0)		
ABVPB, n(%)				χ^2=0.42	0.517
No	200 (87.7)	62 (89.9)	138 (86.8)		
Yes	28 (12.3)	7 (10.1)	21 (13.2)		
Abnormal Blood Vessels in Placental Bed, n(%)				χ^2=0.06	0.800
No	176 (77.2)	54 (78.3)	122 (76.7)		
Yes	52 (22.8)	15 (21.7)	37 (23.3)		

Variables	Total (n = 228)	test (n = 69)	train (n = 159)	Statistic	*P*
Uneven Signaling in Placenta, n(%)				χ^2=0.79	0.375
No	198 (86.8)	62 (89.9)	136 (85.5)		
Yes	30 (13.2)	7 (10.1)	23 (14.5)		
Asymmetrical Thickness and Morphology of Placenta, n(%)				χ^2=0.20	0.658
No	214 (93.9)	66 (95.7)	148 (93.1)		
Yes	14 (6.1)	3 (4.4)	11 (6.9)		
Ischemic Infarction of Placenta, n(%)				χ^2=0.20	0.658
No	214 (93.9)	66 (95.7)	148 (93.1)		
Yes	14 (6.1)	3 (4.4)	11 (6.9)		

Note: PAS: placenta accreta spectrum disorders; PP: placenta previa; T2DB: T2 Dark Band; PUB: placenta and uterine bulge; DT2HIPU: Disappearance of the T2 hypointense interface between placenta and uterus; TUML: thinning of the uterine muscular layer; ABVPB: abnormal blood vessels in placental bed.

Table 4: Comparison of Clinical Data and Imaging Data in the Training Set

Variables	Total (n = 159)	Non-PAS (n = 76)	PAS (n = 83)	Statistic	*P*
Age, Mean ± SD	31.5 ± 4.2	30.6 ± 3.9	32.4 ± 4.2	t=-2.76	0.006
Number of Pregnancies, M (Q_1, Q_3)	2.0 (1.0, 3.0)	2.0 (0.0, 3.3)	2.0 (1.0, 3.0)	Z=-0.96	0.338
Number of Abortions, M (Q_1, Q_3)	1.0 (0.0, 2.0)	1.0 (0.0, 2.0)	1.0 (0.0, 2.0)	Z=-0.22	0.826
Fetal Blood Vessels within Placenta, M (Q_1, Q_3)	0.0 (0.0, 2.5)	0.0 (0.0, 0.0)	0.0 (0.0, 3.2)	Z=-1.42	0.156
Number of Spontaneous Births, M (Q_1, Q_3)	0.0 (0.0, 0.0)	0.0 (0.0, 0.0)	0.0 (0.0, 0.0)	Z=-1.65	0.100
Number of Cesarean Deliveries, M (Q_1, Q_3)	0.0 (0.0, 1.0)	0.0 (0.0, 1.0)	0.0 (0.0, 1.0)	Z=-0.69	0.493
Gestational Hypertension, n(%)				χ^2=0.70	0.403
Gestational Age, M (Q_1, Q_3)	35.0 (33.0, 37.0)	35.0 (32.0, 37.0)	35.0 (33.0, 37.0)	Z=-1.52	0.128
No	149 (93.7)	73 (96.1)	76 (91.6)		
Yes	10 (6.3)	3 (4.0)	7 (8.4)		
Gestational Diabetes Mellitus, n(%)				χ^2=1.98	0.159
No	115 (72.3)	51 (67.1)	64 (77.1)		
Yes	44 (27.7)	25 (32.9)	19 (22.9)		
Vaginal Bleeding During Pregnancy, n(%)				χ^2=3.28	0.070
No	124 (78.0)	64 (84.2)	60 (72.3)		
Yes	35 (22.0)	12 (15.8)	23 (27.7)		
Fetal Lie, n(%)				χ^2=1.66	0.198
Breech presentation	25 (15.7)	9 (11.8)	16 (19.3)		
Vertex position	134 (84.3)	67 (88.2)	67 (80.7)		
PP, n(%)				-	<.001
complete PP	76 (47.8)	22 (29.0)	54 (65.1)		
low-lying PP	6 (3.8)	4 (5.3)	2 (2.4)		
marginal PP	21 (13.2)	9 (11.8)	12 (14.5)		
No	56 (35.2)	41 (54.0)	15 (18.1)		
T2DB, n(%)				χ^2=8.53	0.004
No	132 (83.0)	70 (92.1)	62 (74.7)		
Yes	27 (17.0)	6 (7.9)	21 (25.3)		
PUB, n(%)				χ^2=9.19	0.002
No	134 (84.3)	71 (93.4)	63 (75.9)		

Variables	Total (n = 159)	Non-PAS (n = 76)	PAS (n = 83)	Statistic	*P*
Yes	25 (15.7)	5 (6.6)	20 (24.1)		
DT2HIPU, n(%)				χ^2=27.06	<.001
No	116 (73.0)	70 (92.1)	46 (55.4)		
Yes	43 (27.0)	6 (7.9)	37 (44.6)		
TUML, n(%)				χ^2=14.21	<.001
No	138 (86.8)	74 (97.4)	64 (77.1)		
Yes	21 (13.2)	2 (2.6)	19 (22.9)		
ABVPB, n(%)				χ^2=19.28	<.001
No	122 (76.7)	70 (92.1)	52 (62.7)		
Yes	37 (23.3)	6 (7.9)	31 (37.4)		
Uneven Signaling in Placenta, n(%)				χ^2=0.81	0.368
No	136 (85.5)	67 (88.2)	69 (83.1)		
Yes	23 (14.5)	9 (11.8)	14 (16.9)		
Asymmetrical Thickness and Morphology of Placenta, n(%)				χ^2=0.62	0.431
No	148 (93.1)	72 (94.7)	76 (91.6)		
Yes	11 (6.9)	4 (5.3)	7 (8.4)		
Ischemic Infarction of Placenta, n(%)				χ^2=0.22	0.642
No	148 (93.1)	70 (92.1)	78 (94.0)		
Yes	11 (6.9)	6 (7.9)	5 (6.0)		

Note: PAS: placenta accreta spectrum disorders; PP: placenta previa; T2DB: T2 Dark Band; PUB: placenta and uterine bulge; DT2HIPU: Disappearance of the T2 hypointense interface between placenta and uterus; TUML: thinning of the uterine muscular layer; ABVPB: abnormal blood vessels in placental bed.

Table 5: The results of Binary unconditional logistic regression

Variables	single factor					multiple factors				
	β	S.E	Z	*P*	OR (95%CI)	β	S.E	Z	*P*	OR (95%CI)
Age	0.11	0.04	2.65	0.008	1.12 (1.03 ~ 1.21)	0.18	0.05	3.29	<.001	1.19 (1.07 ~ 1.33)
PP										
complete PP					1.00 (Reference)					1.00 (Reference)
low-lying PP	-1.59	0.90	-1.76	0.078	0.20 (0.03 ~ 1.19)	-1.02	1.00	-1.01	0.311	0.36 (0.05 ~ 2.59)
marginal PP	-0.61	0.51	-1.20	0.230	0.54 (0.20 ~ 1.47)	-0.24	0.57	-0.41	0.680	0.79 (0.26 ~ 2.43)
No	-1.90	0.39	-4.83	<.001	0.15 (0.07 ~ 0.32)	-1.70	0.48	-3.54	<.001	0.18 (0.07 ~ 0.47)
T2DB										
No					1.00 (Reference)					1.00 (Reference)
Yes	1.37	0.49	2.78	0.005	3.95 (1.50 ~ 10.42)	0.10	0.68	0.15	0.881	1.11 (0.29 ~ 4.24)
PUB										
No					1.00 (Reference)					1.00 (Reference)
Yes	1.51	0.53	2.85	0.004	4.51 (1.60 ~ 12.72)	-1.29	1.00	-1.28	0.199	0.28 (0.04 ~ 1.97)

Variables	single factor					multiple factors				
	β	S.E	Z	*P*	OR (95%CI)	β	S.E	Z	*P*	OR (95%CI)
DT2HIPU										
No					1.00 (Reference)					1.00 (Reference)
Yes	2.24	0.48	4.67	<.001	9.38 (3.67 ~ 24.01)	1.64	0.76	2.15	0.031	5.16 (1.16 ~ 22.96)
TUML										
No					1.00 (Reference)					1.00 (Reference)
Yes	2.40	0.76	3.14	0.002	10.98 (2.46 ~ 48.97)	1.11	1.06	1.05	0.293	3.04 (0.38 ~ 24.17)
ABVPB										
No					1.00 (Reference)					1.00 (Reference)
Yes	1.94	0.48	4.02	<.001	6.96 (2.70 ~ 17.89)	1.13	0.60	1.90	0.058	3.11 (0.96 ~ 10.05)

Note: PAS: placenta accreta spectrum disorders; PP: placenta previa; T2DB: T2 Dark Band; PUB: placenta and uterine bulge; DT2HIPU: Disappearance of the T2 hypointense interface between placenta and uterus; TUML: thinning of the uterine muscular layer; ABVPB: abnormal blood vessels in placental bed; OR: Odds Ratio; CI: Confidence Interval.

Table 6: Results of ROC analysis

Data	AUC (95%CI)	Accuracy (95%CI)	Sensitivity (95%CI)	Specificity (95%CI)	PPV (95%CI)	NPV (95%CI)	cut off
Train	0.83 (0.77-0.89)	0.75 (0.67-0.81)	0.78 (0.68 - 0.87)	0.72 (0.63 - 0.82)	0.72 (0.62 - 0.82)	0.78 (0.69 - 0.87)	0.528
Test	0.74 (0.62-0.86)	0.67 (0.54-0.78)	0.69 (0.51 - 0.87)	0.65 (0.51 - 0.79)	0.55 (0.38 - 0.72)	0.78 (0.64 - 0.91)	0.528

Note: ROC: receiver operating characteristic curve; AUC: area under the curve; CI: Confidence Interval; PPV: Positive Predictive Value; NPV: Negative Predictive Value.

ROC analysis shows that AUC of this model for the diagnosis of PAS is 0.74, 95% CI: 0.62-0.86, with Accuracy, Sensitivity, and Specificity of 0.67, 0.69, and 0.65, respectively (Table-6) (Figure-1B). The result of Hosmer-Lemeshow goodness-of-fit test in calibration curve analysis shows $\chi 2=3.30$, P=0.91, the calibration curve has good agreement with the ideal curve (Figure-1E). DCA analysis shows that when the probability threshold range from 0.40 to 0.99, PAS patients could obtain more net benefits (Figure-1F). The diagnostic performance of the model for PAS has also been well validated in the validation set.

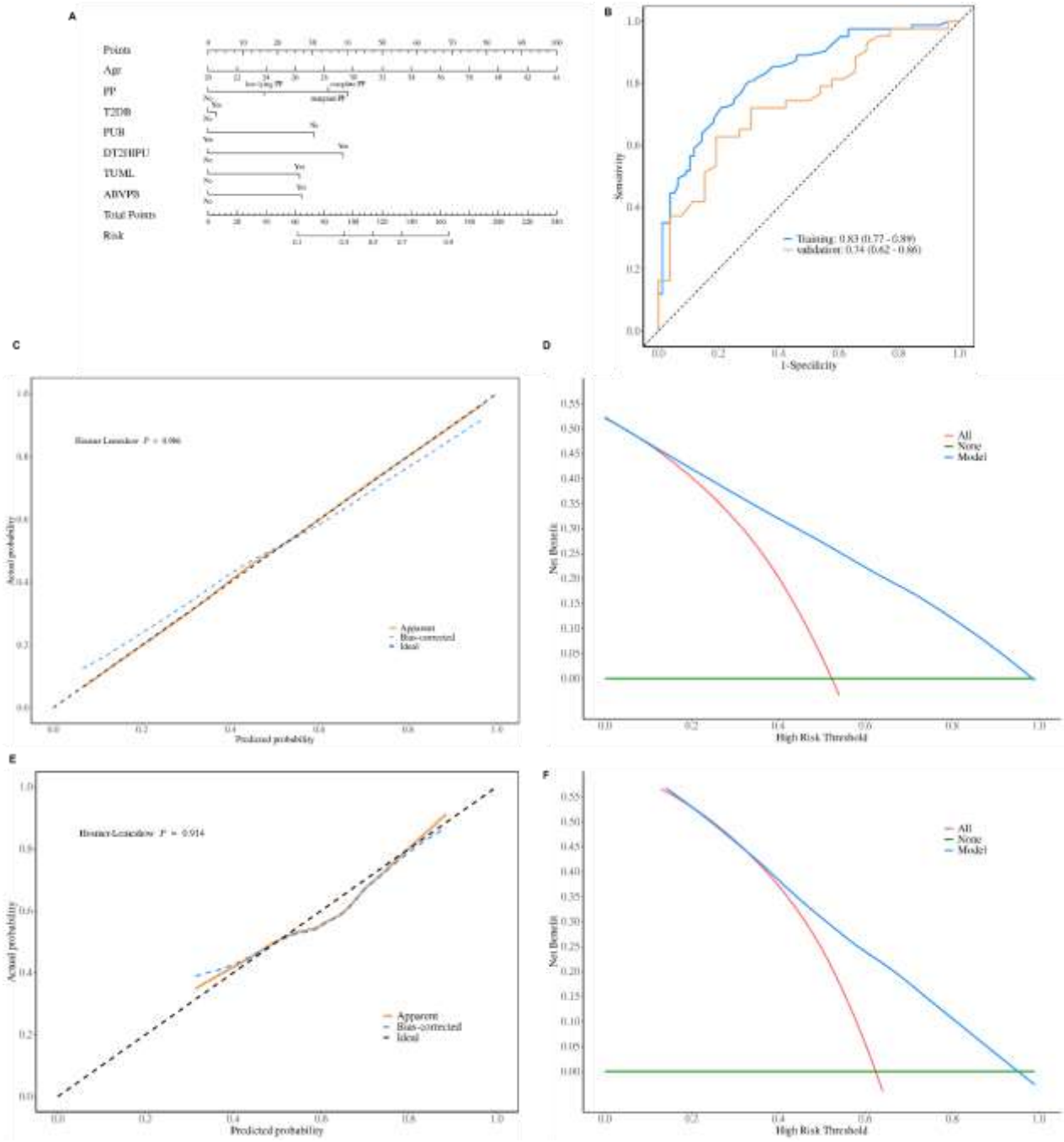

Figure1: (A) Nomogram model based on related factors such as age, PP, T2DB, PUB, DT2HIPU, TUML, and ABVPB;
(B) The results of ROC analysis in training and validation sets.
(C) The result of calibration curve analysis in training set.
(D) The results of DCA analysis in training set.
(E) The result of calibration curve analysis in validation set.
(F) The results of DCA analysis in validation set.

Discussion

PAS is a serious complication of pregnancy, and its pathological mechanism refers to the invasion of placental villi into the uterine muscle layer. This condition can easily lead to difficulties in separating

the placenta from the uterine wall, resulting in postpartum massive bleeding and disseminated intravascular coagulation and other critical conditions, seriously threatening the life of pregnant women and being an important cause of severe adverse outcomes for both mothers and fetuses[10]. As more and more women are undergoing cesarean section surgeries nowadays, the incidence of placenta increta in women with cesarean sections is also increasing year by year[11]. Therefore, the accuracy of prenatal diagnosis of placenta accreta is particularly important, which helps clinicians develop appropriate delivery plans and improve maternal and fetal outcomes.

Current research indicates that advanced maternal age, PP, and prior cesarean section are risk factors for PAS[12, 13]. The findings of this study also show that advanced maternal age and PP are independent risk factors for PAS. Advanced age is an important high-risk factor for PAS. With the increase of age, the risk of PAS increases significantly, which may be related to the physiological changes of the endometrium, making it easier for the placenta to implant into the myometrium[14]. When PP exists, the position of the placenta is lower, possibly covering or close to the cervical internal os. This situation makes it easier for the placenta to adhere abnormally to the uterine wall, thus increasing the likelihood of PAS. Especially when PP is accompanied by a history of cesarean section, the risk of PAS is even higher[4]. However, our study results do not suggest an association between previous cesarean section history and PAS. This may be related to the small sample size of our study, and we will follow up with a larger sample size in collaboration with other centers for further verification.

As MRI technology becomes widely used in the field of obstetrics, its diagnostic advantages in the assessment of placenta previa combined with PP have become evident. Currently, several consensus statements by the Society of Abdominal Radiology (SAR) and the European Society of Urogenital Radiology (ESUR) provide important guidelines for the identification of specific MRI features. These features include a short T2WI signal band in the placenta, thinning of the myometrium, interruption of the continuity of the urinary bladder wall, local bulge of the uterus, abnormal vascular proliferation beneath the placenta, placental heterogeneity, changes in morphology of the placenta – asymmetric thickening, ischemic infarcts in the placenta, and abnormal vascular shadows within the placenta[15]. By evaluating these characteristic MRI findings, clinicians can improve the accuracy of diagnosing PAS, which is crucial for the formulation of appropriate management strategies and improved outcomes for both mother and fetus. In this study, we found T2DB, PUB, DT2HIPU, TUML and ABVPB are related factors for PAS, and T2HIPU are independent related factors for PAS.

However, due to the limitations of the human eye subjective vision of radiologists and the differences between interpreters, there may be missed or misdiagnosed cases of PAS. The Nomogram model transforms complex regression equations into visual graphics, making the predictions from the models more readable and convenient for patient evaluation. The intuitive and easy-to-understand nature of this approach has led to an increasing amount of attention and application within medical research and clinical practice[16]. Compared with models that only rely on MRI morphologic features, Nomogram models, which incorporate imaging features and clinical data, significantly improve the accuracy and predictive power of diagnoses. In this study, we constructed a Nomogram model using risk factors associated with PAS, and after ROC, DCA, and calibration curve analyses, the results indicated that the model had a certain degree of discrimination and consistency in the diagnosis of PAS, patients could obtain a positive net benefit, and it was validated in the validation set.

Study strengths and limitations

The nomogram model, which integrates MRI findings and clinical factors, exhibits robust clinical effectiveness in the prenatal diagnosis of PAS, rendering it well-suited for practical clinical implementation. However, this study is a single-center retrospective study with a small sample size, which may introduce certain statistical biases. In

addition, we did not conduct external validation. These limitations will be further investigated and addressed in subsequent works

Conclusion

In a summary, Nomogram model based on MR and clinical factors demonstrates good clinical efficacy for prenatal diagnosis of PAS, and it is suitable for clinical application.

Authors contributions

DM designed this research project. JZ, QZ, PL, and HY collected and organized the data. JZ and QZ analyzed the data. JZ wrote the initial draft. All authors reviewed the manuscript.

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Conflict of interests

The authors declare that they have no conflict of interest.

Consent for publication

All patients had given informed consent.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Ethics approval

This study was approved by the Ethics Committee of Affiliated Maternity and Child Health Care Hospital of Nantong University (Y2020001).

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