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Original Article

Nomogram for predicting postpartum pelvic floor dysfunction based on ultrasound and serum biomarkers FGF2 and HBP

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Abstract: Objective: To evaluate the utility of pelvic floor ultrasound combined with serum fibroblast growth factor 2 (FGF2) and heparin-binding protein (HBP) in assessing postpartum pelvic floor dysfunction (PFD), and to construct a nomogram model for prognostic prediction. Methods: A retrospective study included 149 primiparous women at 42 days postpartum who underwent pelvic floor ultrasound at Wenzhou Central Hospital from January 2020 to December 2021. Participants were divided into a control group (cesarean section, 68 cases) and an observation group (vaginal delivery, 81 cases). Baseline characteristics, ultrasound parameters, and serum FGF2 and HBP levels were recorded. Incidence of PFD, defined as pelvic organ prolapse or stress urinary incontinence, was documented during 3-year follow-up. Correlations were analyzed, and univariate and multivariate logistic regression identified independent predictors. A nomogram was constructed and validated using ROC, calibration, and decision curve analyses. Results: Abnormal pelvic floor ultrasound findings were more frequent after vaginal delivery (64.2% vs. 52.9%). Quantitative ultrasound indices were significantly higher in the observation group, while serum FGF2 and HBP levels were significantly lower (all $P < 0.05$). Ultrasound indicators were negatively correlated with biomarker levels. During follow-up, 61 PFD cases occurred in the observation group and 22 in the control group. Independent risk factors included age ≥ 30 years, prenatal BMI ≥ 27 kg/m^2, vaginal delivery, no pelvic floor training, constipation during pregnancy, episiotomy, and neonatal birth weight ≥ 4 kg. The nomogram showed excellent discrimination (AUC=0.931, 95% CI: 0.892-0.970) and good calibration, with clinical benefit confirmed by decision curve analysis. Conclusion: Ultrasound combined with serum FGF2 and HBP enables comprehensive assessment of postpartum pelvic floor function. The nomogram model provides accurate risk prediction and may guide early identification and intervention in high-risk women.

Keywords: Pelvic floor dysfunction, ultrasound, fibroblast growth factor 2, heparin-binding protein, nomogram

Introduction

Pelvic floor dysfunction (PFD) encompasses a group of disorders characterized by pelvic organ displacement and functional impairment resulting from weakened pelvic floor support structures. Major risk factors include menopause, childbirth, and chronic constipation. Common manifestations are fecal incontinence, stress urinary incontinence, and uterine prolapse, all of which markedly affect women's health and quality of life [1]. Childbirth is one of the leading causes of PFD, with surveys reporting a postpartum incidence of 5%-45%, accounting for over 70% of all PFD cases. Thus, early and accurate evaluation of postpartum pelvic floor function and identification of high-risk women are essential for prevention and timely intervention [2].

Currently, pelvic floor function is mainly assessed through clinical symptom evaluation, pelvic floor muscle strength testing, and ultrasound examination. Owing to its noninvasive nature, high reproducibility, simple operation, and ability to dynamically visualize pelvic floor support structures, pelvic floor ultrasound has become an indispensable imaging tool [3, 4]. However, ultrasound primarily reflects static or dynamic anatomical changes and has limitations in predicting tissue repair capacity, inflammatory status, and long-term functional prognosis. In this context, serum biomarkers provide

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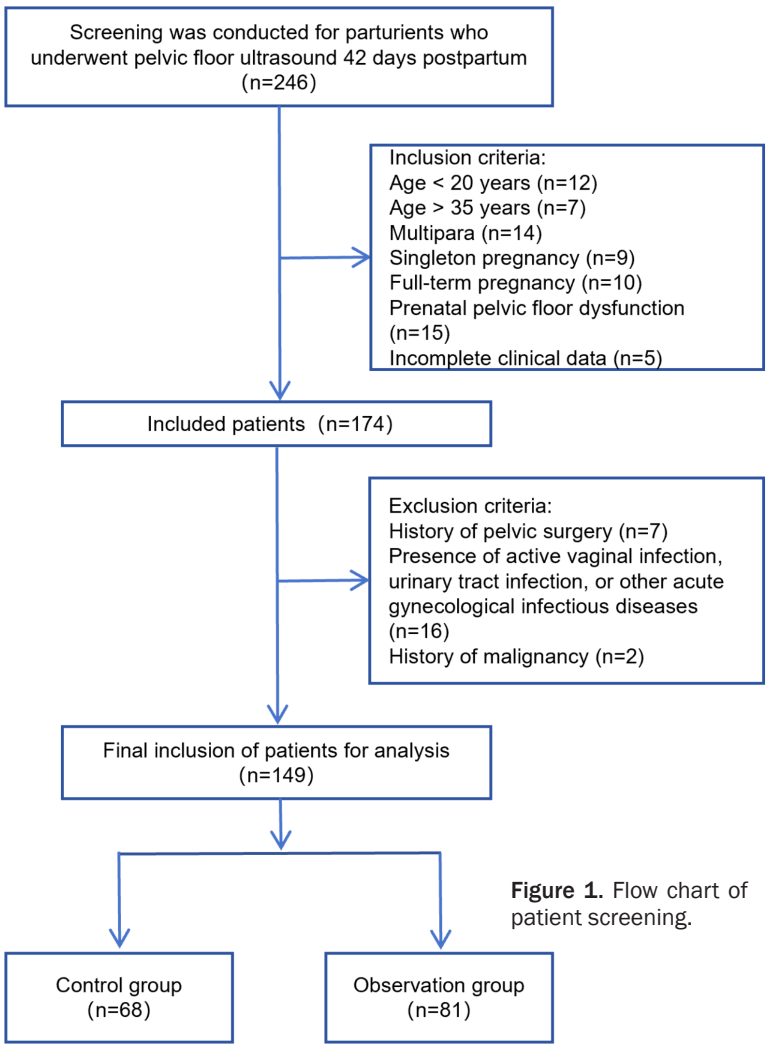

Figure 1. Flow chart of patient screening.

complementary value by reflecting underlying pathophysiological processes.

Fibroblast growth factor 2 (FGF2) and heparin-binding protein (HBP) are two key serum biomarkers. FGF2 is a potent angiogenic factor and central regulator of tissue repair, essential for the regeneration of soft tissues such as muscles and ligaments after injury [5]. Since childbirth causes traction and microinjury to pelvic floor muscles, fascia, and ligaments, postpartum serum FGF2 levels may reflect the intrinsic repair potential of these supportive tissues. HBP, a major effector released upon neutrophil activation, sensitively reflects local inflammatory responses and tissue injury [6]. Given that childbirth-related trauma or secondary infection can trigger persistent inflammation and impair recovery, HBP may serve as a valuable indicator of postpartum pelvic floor tissue damage. Although both biomarkers have been implicated in tissue injury and repair [5, 6], their roles in predicting postpartum PFD risk and severity remain largely unexplored.

Therefore, this study integrates pelvic floor ultrasound with serum FGF2 and HBP detection to comprehensively evaluate postpartum pelvic floor function. By combining imaging with molecular indicators, this approach provides structural as well as dynamic biological insights into tissue repair and inflammatory responses. A nomogram prediction model based on multifactorial analysis is further established to enable early identification of high-risk women and guide individualized clinical interventions.

Materials and methods

Subjects

A retrospective analysis was performed on 149 postpartum women who underwent pelvic floor ultrasound at 42 days after delivery at Wenzhou Central Hospital between January 2020 and December 2021. Patients were divided into a control group (cesarean section, n=68) and an observation group (vaginal delivery, n=81). All women were followed for 3 years to document the incidence of pelvic organ prolapse and stress urinary incontinence. The study protocol was approved by the Medical Ethics Committee of Wenzhou Central Hospital (approval number: L2025-05-024).

Inclusion criteria were: age 20-35 years; primiparous; singleton, full-term pregnancy; no history of PFD prior to childbirth; and complete clinical and follow-up data.

Exclusion criteria were: history of pelvic surgery; active vaginal or urinary tract infection, or other acute gynecological infection; and history of malignant tumors. The screening process is illustrated in **Figure 1**.

Data collection

Baseline characteristics: Age, prenatal BMI, education level, and occupation were extracted from medical records.

Ultrasound abnormalities: Abnormalities at 42 days postpartum were recorded, including bladder neck mobility, widened posterior bladder angle, urethral funneling, bladder bulge, uterine bulge, rectal bulge, levator ani injury, levator hiatus enlargement, and abnormal perineal body activity.

Ultrasound parameters: Quantitative indices were extracted, including bladder neck displacement, distance from bladder neck to pubic symphysis (Valsalva), distance from rectal ampulla to pubic symphysis (Valsalva), and levator hiatus area.

Serum biomarkers: On day 42 postpartum, 5 mL of fasting venous blood was drawn, centrifuged at 3,000 rpm for 10 min, and serum collected.

FGF2 levels were measured by ELISA using the BIO-RAD iMark microplate reader (Tianjin Kangerk Company) with a kit from Beijing Yitai Biotechnology Co., Ltd.

HBP levels were measured by fluorescence immunoassay using the AFS3000B analyzer (Guangzhou Lanbo Biotechnology Co., Ltd.).

Follow-up outcomes: The incidence of pelvic organ prolapse (defined as POP-Q stage ≥ II or relevant diagnostic records) and/or stress urinary incontinence (diagnosed clinically or by patient-reported leakage during exertion, coughing, or sneezing) within 3 years postpartum was recorded from outpatient charts, urogynecology consultation notes, physical therapy records, and electronic medical records.

Outcome measures

The primary outcomes were the incidence of pelvic organ prolapse and stress urinary incontinence within 3 years postpartum.

Secondary outcomes included abnormal ultrasound findings, quantitative ultrasound indices, serum FGF2 and HBP levels, and the development and validation of a nomogram prediction model.

Nomogram construction and validation

Independent predictors of PFD were identified by multivariate logistic regression, and a nomogram was constructed based on regression coefficients to assign quantitative risk scores.

Calibration: Bootstrap resampling (1,000 iterations) was used to compare predicted vs. observed probabilities.

Discrimination: Predictive performance was evaluated by receiver operating characteristic (ROC) curve analysis, with the area under the curve (AUC) representing accuracy.

Clinical utility: Decision curve analysis (DCA) was applied to assess net benefit across threshold probabilities.

All modeling was performed using the rms package in R (version 4.1.0).

Statistical methods

Data were analyzed using SPSS 23.0 and GraphPad Prism 8.0. Categorical variables were summarized as frequencies and percentages. The normality of continuous variables was assessed using the Shapiro - Wilk test. Normally distributed continuous variables are expressed as mean ± standard deviation, and intergroup comparisons were conducted using one-way analysis of variance (ANOVA), with post hoc pairwise comparisons performed using the LSD test when homogeneity of variance was assumed, or the Games - Howell test when variances were unequal. Categorical variables were compared using Pearson's chi-square test or Fisher's exact test (if expected cell frequencies were < 5). Correlations between ultrasound parameters and serum biomarker levels were assessed using Pearson correlation analysis.

After 3 years, patients were categorized into a good prognosis group and a poor prognosis group according to whether PFD occurred. Clinical variables were compared by univariate analysis, and those with $P < 0.05$ were included in multivariate logistic regression with stepwise selection to identify independent risk factors. A dynamic nomogram was then constructed based on the regression model. Its accuracy was further evaluated using ROC, calibration, and DCA curves. A two-sided $P < 0.05$ was considered statistically significant.

Table 1. Baseline data of the two groups

		Control group n=68	Observation group n=81	χ^2	*P*
Age, years	< 30	38	39	0.102	0.750
	≥ 30	30	42		
Prenatal BMI, kg/m^2	< 27	29	36	0.049	0.826
	≥ 27	39	45		
Education level	Primary school or below	16	20	1.875	0.392
	Middle school	23	36		
	University and above	27	25		
Occupation type	Manual labor	41	44	0.538	0.463
	Non-manual labor	27	37		
Number of pregnancies	One time	55	59	0.312	0.576
	≥ 2 times	15	20		
Hypertension in pregnancy	No	55	66	0.351	0.554
	Yes	11	17		
Gestational diabetes mellitus	No	41	67	0.867	0.352
	Yes	19	22		
Gestational weeks	< 37 weeks	21	23	0.110	0.740
	≥ 37 weeks	47	58		
Delivery mode	Cesarean section	36	32	2.689	0.101
	Vaginal delivery	32	49		
Pelvic floor muscle exercise	No	37	41	0.644	0.213
	Yes	31	40		
Constipation during pregnancy	No	36	32	2.689	0.101
	Yes	32	49		
Episiotomy	No	43	42	1.662	0.197
	Yes	26	39		
Newborn body mass	< 4.0 kg	30	28	2.124	0.145
	≥ 4.0 kg	36	55		

Results

Baseline data

No significant differences were observed between the control and observation groups in terms of age, prenatal BMI, education level, occupation type, gravidity, or parity (all P > 0.05) (**Table 1**).

Abnormal pelvic floor ultrasound findings

Abnormal ultrasound results were detected in 36 cases (52.94%) in the control group and 52 cases (64.20%) in the observation group. The incidence of abnormal findings was significantly higher in the observation group (P < 0.05). Specifically, the number of bladder neck mobility, widened posterior bladder angle, urethral funneling, bladder bulge, uterine bulge, rectal bulge, levator ani injury, levator hiatus enlargement, and abnormal perineal body activity in the observation group were higher than those in the control group (**Figure 2**).

Quantitative ultrasound parameters

The observation group exhibited significantly higher mean value of bladder neck displacement, distance from bladder neck to pubic symphysis (Valsalva), distance from rectal ampulla to pubic symphysis (Valsalva), and levator hiatus area compared with the control group (P < 0.05) (**Figure 3**). Representative ultrasound images are presented in **Figure 4**.

Serum FGF2 and HBP levels

Serum FGF2 and HBP levels were significantly lower in the observation group than those in the control group (both P < 0.05) (**Figure 5**).

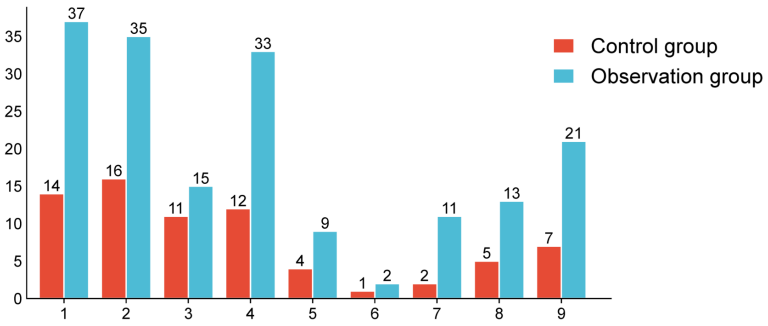

Figure 2. Abnormalities of pelvic floor ultrasound related indicators in the two groups. 1: Bladder neck mobility; 2: widened posterior bladder angle; 3: urethral funneling; 4: Bladder bulge; 5: Uterine bulge; 6: Rectal bulge; 7: Levator ani injury; 8: Levator hiatus enlargement; 9: abnormal perineal body activity.

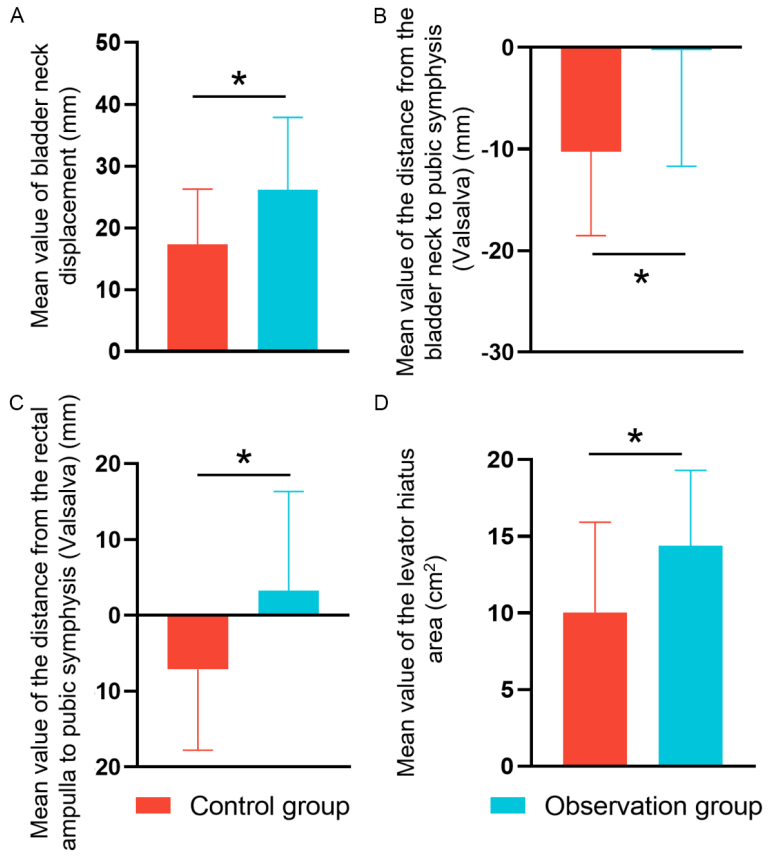

Figure 3. Levels of pelvic floor ultrasound related indicators in the two groups. A. Mean value of bladder neck displacement; B. Mean value of the distance from the bladder neck to pubic symphysis (Valsalva); C. Mean value of the distance from the rectal ampulla to pubic symphysis (Valsalva); D. Mean value of the levator hiatus area. *$P < 0.05$.

Correlation analysis

Pelvic floor ultrasound parameters were negatively correlated with serum FGF2 and HBP levels (all $P < 0.05$) (**Table 2**; **Figure 6**).

Univariate analysis

During the 3-year follow-up, 61 cases of PFD occurred in the observation group and 22 in the control group. Based on outcomes, participants were divided into good prognosis and poor prognosis groups. Compared with the good prognosis group, the poor prognosis group had significantly higher proportions of women aged ≥ 30 years, with prenatal BMI ≥ 27 kg/m^2, vaginal delivery, no pelvic floor muscle training, constipation during pregnancy, episiotomy, and neonatal birth weight ≥ 4 kg (all $P < 0.05$) (**Table 3**).

Multivariate analysis

Variables identified in univariate analysis were entered into multivariate logistic regression. Age ≥ 30 years, prenatal BMI ≥ 27 kg/m^2, vaginal delivery, no pelvic floor muscle training, constipation during pregnancy, episiotomy, and neonatal birth weight ≥ 4 kg were confirmed as independent risk factors for postpartum PFD (all $P < 0.05$) (**Table 4**).

Nomogram construction

A prognostic nomogram was developed using the independent predictors identified above, including age, prenatal BMI, mode of delivery, pelvic floor muscle training, constipation during pregnancy, and neonatal weight. Each factor was assigned a weighted score, and the total score corresponded to the predicted risk of PFD (**Figure 7**).

Model validation

The nomogram demonstrated excellent discrimination, with an AUC of 0.931 (95% CI: 0.892-0.970) (**Figure 8A**). Internal validation

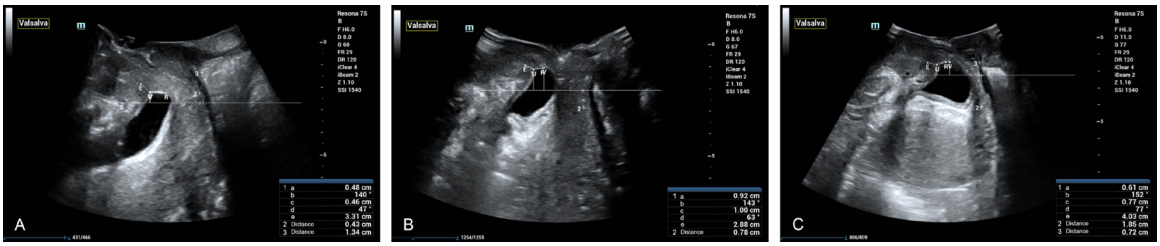

Figure 4. Pelvic floor ultrasound images of the two groups of patients. A. Primipara, 40 years old, diagnosed with stress urinary incontinence. Ultrasound images 42 days postpartum showed that the bladder neck was positioned 5 mm below the reference line, the bladder neck mobility 33 mm, the posterior bladder Angle 140°, the urethral rotation Angle 91°, the lowest point of the posterior bladder wall 5 mm below the reference line, and the cervix 4 mm below the reference line in Valsalva. B. Primipara, 28 years old. Forty-two days postpartum ultrasound images showed that in Valsalva position, the bladder neck was 9 mm below the reference line, the bladder neck mobility was 29 mm, the posterior bladder Angle was 143°, the urethral rotation Angle was 58°, the nadir of the posterior bladder wall was 10 mm below the reference line, and the cervix was 8 mm above the reference line. C. Multipara, 29 years old. Ultrasound images 42 days after delivery showed that the bladder neck was 6 mm below the reference line, the bladder neck mobility was 40 mm, the posterior bladder Angle was 152°, the urethral rotation Angle was 115°, the lowest point of the posterior bladder wall was 8 mm below the reference line, and the cervix was 19 mm below the reference line.

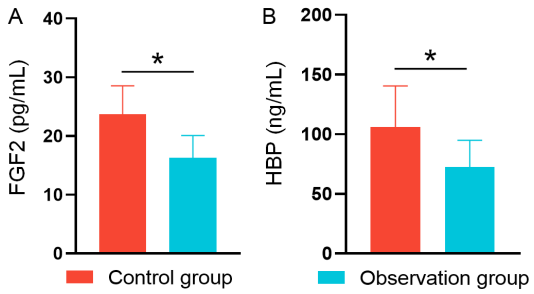

Figure 5. Serum FGF2 and HBP levels in the two groups. A. FGF2; B. HBP. FGF2 = Fibroblast Growth Factor 2; HBP = Heparin-Binding Protein. *$P < 0.05$.

using bootstrap resampling confirmed good calibration, with the calibration curve closely aligned with the ideal reference line (**Figure 8B**). DCA further indicated that the model provided meaningful clinical benefit across a wide range of threshold probabilities (**Figure 8C**).

Discussion

The female pelvic floor is a complex support system composed of muscles, fascia, ligaments, nerves, and blood vessels. It maintains the anatomical positions of the urethra, vagina, and rectum and plays a critical role in supporting pelvic organs [7]. Pregnancy and childbirth substantially affect this system, with mechanical injuries from vaginal delivery being particularly pronounced [8, 9]. In this study, the incidence of pelvic floor ultrasound abnormalities at 42 days postpartum was relatively high, with significantly greater damage observed among women who delivered vaginally compared with those who underwent cesarean section. This finding aligns with the well-established impact of vaginal delivery on pelvic floor tissues.

During the second stage of labor, the passage of the fetus through the birth canal exposes the pelvic floor to high pressure and friction [10], causing direct mechanical damage to muscles (e.g., levator ani), fascia, and ligaments. Such injury may result in muscle discontinuity or levator hiatus enlargement, with reported incidences ranging from 15% to 35% [11]. This explains the greater levator hiatus area observed in the vaginal delivery group. Furthermore, overstretching of pelvic floor tissues may induce neurogenic injury, including axonal degeneration and denervation [12], leading to impaired contractility and reduced support for pelvic organs [13]. Together, mechanical and neurogenic injury alter the biomechanical properties of pelvic floor tissues, preventing complete recovery of elasticity and tension postpartum. Consequently, irreversible relaxation of bladder neck and perirectal support structures develops, forming the pathological basis for PFD.

With the adjustment of China's fertility policies, the proportion of advanced maternal age pregnancies has increased markedly. Age-related factors such as decreased pelvic floor muscle flexibility, diminished repair capacity, and prolonged labor further exacerbate delivery-associated injuries, significantly increasing the risk

Table 2. Correlation between pelvic floor ultrasound and serum FGF2 and HBP levels

	FGF2		HBP	
	r	*P*	*r*	*P*
Mean value of bladder neck displacement	-0.632	< 0.001	-0.789	< 0.001
Mean value of the distance from the bladder neck to pubic symphysis (Valsalva)	-0.680	< 0.001	-0.896	< 0.001
Mean value of the distance from the rectal ampulla to pubic symphysis (Valsalva)	-0.505	< 0.001	-0.545	< 0.001
Mean value of the levator hiatus area	-0.690	< 0.001	-0.847	< 0.001

Note: FGF2 = Fibroblast Growth Factor 2; HBP = Heparin-Binding Protein.

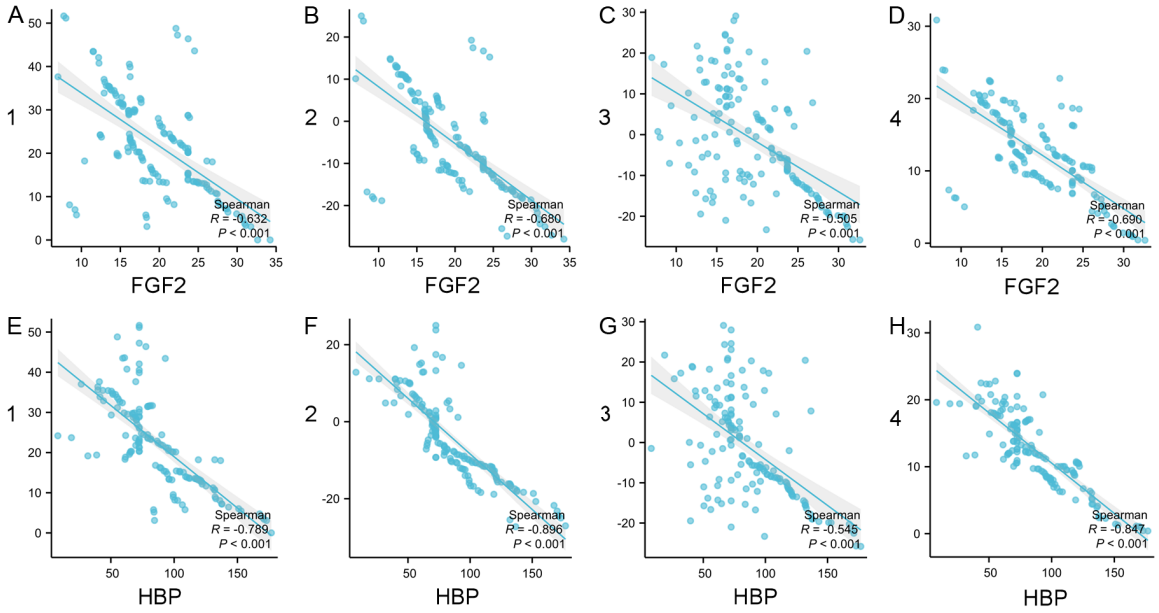

Figure 6. Scatter plot of correlation between pelvic floor ultrasound and serum FGF2 and HBP levels. 1: Mean value of bladder neck displacement; 2: Mean value of the distance from the bladder neck to pubic symphysis (Valsalva); 3: Mean value of the distance from the rectal ampulla to pubic symphysis (Valsalva); 4: Mean value of the levator hiatus area. FGF2 = Fibroblast Growth Factor 2; HBP = Heparin-Binding Protein.

of future PFD, including pelvic organ prolapse and stress urinary incontinence. Thus, early identification of pelvic floor injuries and timely rehabilitative interventions, particularly in women with vaginal delivery or advanced maternal age, are of high clinical relevance.

FGF2 is a multifunctional cytokine that stimulates fibroblast, endothelial cell, and smooth muscle proliferation, thereby promoting tissue repair [14]. In our study, serum FGF2 levels were significantly higher after cesarean section compared with vaginal delivery. Possible explanations include: (1) surgical stress activating inflammatory pathways (e.g., NF-κB), thereby upregulating FGF2 expression; and (2) decidual damage and bleeding caused by manual placental separation, which stimulates vascular repair responses and FGF2 release [15]. This is consistent with studies showing that FGF2 promotes bone marrow mesenchymal stem cells differentiation into fibroblasts, contributing to ligament and tendon reconstruction in the pelvic floor [16].

HBP, an effector protein released by neutrophils, is a key mediator of inflammation and immune regulation and is widely used as a biomarker in sepsis and acute infections [17, 18]. Our study revealed that HBP levels were also higher in women after cesarean section than after vaginal delivery. This may be explained by: (1) extensive tissue trauma from surgical incision through multiple layers, leading to neutrophil activation; (2) sutures and exposure of the placental separation surface inducing sterile inflammation; and (3) additional decidual damage and bleeding during manual placental separation [18, 19]. Layuk et al. further reported that urinary HBP levels were elevated in pre-

Table 3. Univariate analysis

		Good prognosis group n=66	Poor prognosis group n=83	χ^2	*P*
Age, years	< 30	45	32	12.923	< 0.001
	≥ 30	21	51		
Prenatal BMI, kg/m^2	< 27	41	24	16.483	< 0.001
	≥ 27	25	59		
Education level	Primary school or below	14	19	0.823	0.663
	Middle school	26	43		
	University and above	26	31		
Occupation type	Manual labor	38	47	0.014	0.908
	Non-manual labor	28	36		
Number of pregnancies	One time	52	62	0.342	0.559
	≥ 2 times	14	21		
Hypertension in pregnancy	No	55	66	0.351	0.554
	Yes	11	17		
Gestational diabetes mellitus	No	48	60	0.004	0.953
	Yes	18	23		
Gestational weeks	< 37 weeks	19	25	0.032	0.859
	≥ 37 weeks	47	58		
Delivery mode	Cesarean section	46	22	27.644	< 0.001
	Vaginal delivery	20	61		
Pelvic floor muscle exercise	No	27	51	6.216	0.013
	Yes	39	32		
Constipation during pregnancy	No	44	24	21.119	< 0.001
	Yes	22	59		
Episiotomy	No	43	41	3.710	0.054
	Yes	23	42		
Newborn body mass	< 4.0 kg	40	18	23.424	< 0.001
	≥ 4.0 kg	26	65		

Table 4. Multivariate analysis

	B	*S.E.*	*Wald*	*P*	*OR*	*95% CI*
Age ≥ 30 years	2.034	0.557	3.654	< 0.001	7.644	2.568-22.756
Prenatal BMI ≥ 27 kg/m^2	1.829	0.538	3.398	< 0.001	6.225	2.168-17.870
Vaginal delivery	2.580	0.561	4.601	< 0.001	13.196	4.397-39.602
No pelvic floor muscle training	1.549	0.538	2.881	0.004	4.706	1.641-13.496
Constipation during pregnancy	1.807	0.526	3.433	< 0.001	6.094	2.172-17.100
Newborn body mass ≥ 4.0 kg	1.778	0.559	3.178	0.001	5.919	1.977-17.720

eclampsia, highlighting its role in pregnancy-related inflammatory processes [20]. These findings suggest that HBP is closely associated with maternal inflammatory responses.

Importantly, we observed significant negative correlations between pelvic floor functional indices and both FGF2 and HBP levels. This implies that excessive inflammatory activity may activate matrix metalloproteinases, degrading the extracellular matrix, while dysregulated repair processes disrupt balanced tissue remodeling, collectively impairing pelvic floor support [21]. These results deepen our understanding of how delivery mode influences pelvic floor recovery and highlight potential molecular targets for intervention.

The development of PFD is a complex pathophysiological process involving multiple inter-

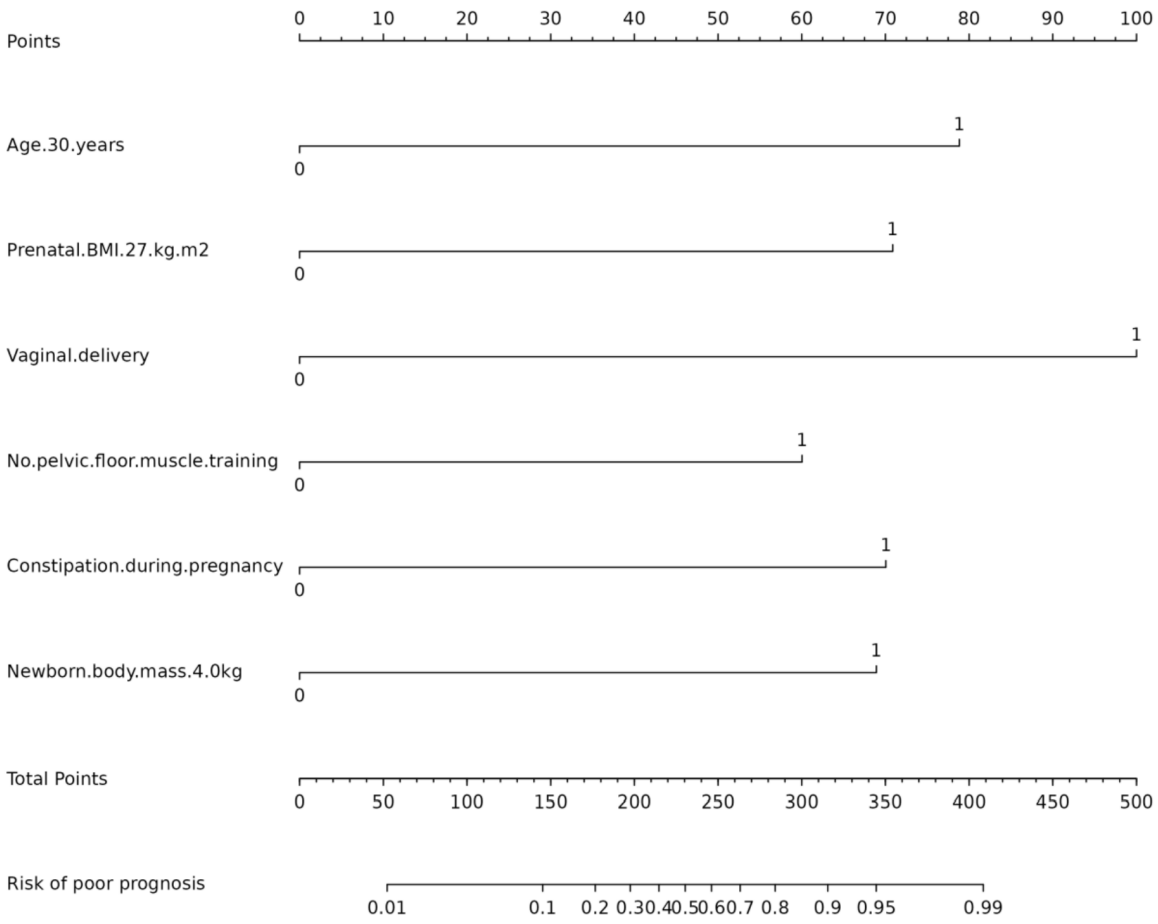

Figure 7. Nomogram prediction model.

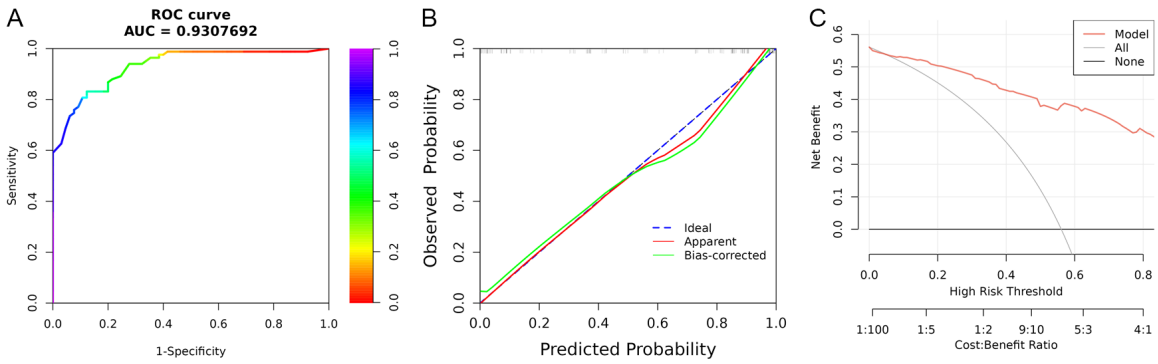

Figure 8. Validation of the nomogram prediction model. A. ROC curve; B. Calibration curve; C. DCA curve. ROC = Receiver Operating Characteristic; DCA = Decision Curve Analysis.

acting factors. In this study, systematic analysis identified age ≥ 30 years, prenatal BMI ≥ 27 kg/m^2, vaginal delivery, lack of pelvic floor muscle training, constipation during pregnancy, episiotomy, and neonatal birth weight ≥ 4 kg as independent risk factors. These factors compromise pelvic floor function mainly by damaging structural integrity, impairing neuromuscular function, and increasing mechanical load [22].

Advancing age contributes to degenerative changes in pelvic floor muscles and fascia, including muscle fiber atrophy and reduced elas-

tic fibers, resulting in weakened support [23]. Nerve function decline further slows conduction velocity, impairing coordinated contraction and relaxation. Obesity is another important contributor: elevated intra-abdominal pressure imposes chronic overload on pelvic floor muscles, while metabolic abnormalities (e.g., insulin resistance, hyperlipidemia) impair microvascular function, limiting tissue repair [24].

Mode of delivery is a key determinant of pelvic floor outcomes. Although clinical evidence remains somewhat controversial, most studies suggest vaginal delivery increases the risk of pelvic organ prolapse and stress urinary incontinence, particularly in cases of macrosomia (≥ 4 kg), where excessive stretching and compression may cause muscular, fascial, and even neural injury [25]. Episiotomy directly disrupts anatomic continuity, and subsequent scar formation may further impair pelvic floor muscle function [26]. Importantly, some evidence suggests that the long-term impact of delivery mode may diminish over time, underscoring the value of postpartum rehabilitation. Without timely pelvic floor muscle training, strength and endurance are difficult to restore, leaving organ support persistently compromised [27]. In addition, pregnancy-related factors that elevate intra-abdominal pressure, such as constipation, exacerbate pelvic floor damage [28].

Early recognition of these risk factors, coupled with targeted interventions such as pelvic floor muscle training and lifestyle modification, is therefore critical for prevention and recovery.

This study integrated multiple predictive indicators to develop a nomogram model for postpartum PFD. The model demonstrated excellent performance, with an AUC of 0.931, reflecting strong discriminative ability. The calibration curve closely aligned with observed outcomes, confirming good reliability. This model enables individualized risk prediction and provides a practical tool to guide clinical decision-making and prognosis assessment.

Several limitations must be acknowledged. First, this was a single-center cross-sectional observational study with a relatively homogeneous patient population, which may introduce selection bias. Second, the proportions of advanced maternal age (≥ 35 years) and macrosomic births were low, limiting the ability to fully evaluate these high-risk subgroups. Larger, multicenter studies with more diverse populations are needed to enhance generalizability. Third, although we observed negative correlations between FGF2/HBP levels and pelvic floor function, histological or molecular experiments were not performed to validate the proposed "inflammation-repair imbalance" mechanism. Hence, the mechanistic explanation remains speculative.

In summary, the present study clarified the effects of multiple risk factors on postpartum PFD and established a nomogram prediction model with high predictive accuracy. The findings provide valuable clinical guidance for early identification of high-risk women and for implementing preventive and rehabilitative strategies, including pelvic floor muscle training, lifestyle adjustment, and prenatal health management. Future research should explore the long-term effects of delivery mode on pelvic floor function and develop optimized rehabilitation strategies to prevent and mitigate PFD more effectively.

Disclosure of conflict of interest

None.

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References

[1] Quaghebeur J, Petros P, Wyndaele JJ and De Wachter S. Pelvic-floor function, dysfunction, and treatment. Eur J Obstet Gynecol Reprod Biol 2021; 265: 143-149.

[2] Peinado-Molina RA, Hernández-Martínez A, Martínez-Vázquez S, Rodríguez-Almagro J and Martínez-Galiano JM. Pelvic floor dysfunction: prevalence and associated factors. BMC Public Health 2023; 23: 2005.

[3] Yao L, Li F, Wang D and Sheng S. Evaluation of acupuncture treatments of postpartum female pelvic floor dysfunction by four-dimensional transperineal pelvic floor ultrasound. Medicine (Baltimore) 2021; 100: e27236.

[4] Sun S, Li H, Liu M, Shang Q, Tan Q and Yin W. An evaluation of the effects of gestational weight gain on the early postpartum pelvic floor using transperineal ultrasound. J Ultrasound Med 2023; 42: 2331-2338.

[5] Nickle A, Ko S and Merrill AE. Fibroblast growth factor 2. Differentiation 2024; 139: 100733.
[6] Kong D, Lei Z, Wang Z, Yu M, Li J, Chai W and Zhao X. A novel HCP (heparin-binding protein-C reactive protein-procalcitonin) inflammatory composite model can predict severe acute pancreatitis. Sci Rep 2023; 13: 9440.
[7] Wang Z, Saija C, Raison N, Aydin A, Xu Z, Zuo K, Rhode K and Pontiki A. Low-cost male urogenital simulator for penile implant surgery training: a 3D printing approach. 3D Print Med 2025; 11: 1.
[8] Cattani L, Van Schoubroeck D, Samešova A, Packet B, Housmans S and Deprest J. Pelvic floor ultrasound findings and symptoms of pelvic floor dysfunction during pregnancy. Int Urogynecol J 2024; 35: 2423-2430.
[9] Hegde A, Huebner M, Ibrahim S, Mastrolia SA, David-Montefiore E and Weintraub AY. Impact of childbirth on pelvic floor dysfunction in women who have undergone previous pelvic floor reconstructive surgery: systematic review and meta-analysis. Int Urogynecol J 2024; 35: 3-17.
[10] Martinho N, Friedman T, Turel F, Robledo K, Riccetto C and Dietz HP. Birthweight and pelvic floor trauma after vaginal childbirth. Int Urogynecol J 2019; 30: 985-990.
[11] O'Leary BD and Keane DP. Effect of the length of the second stage of labor on pelvic floor dysfunction. Am J Obstet Gynecol MFM 2023; 5: 100795.
[12] Mastwyk S, McClelland J, Cooper MM and Frawley HC. Pelvic floor muscle function in the standing position in women with pelvic floor dysfunction. Int Urogynecol J 2022; 33: 2435-2444.
[13] Augustina SJ, Suganthirababu P, Rajagopal V, Kumaresan A, Srinivasan V, Ramana K, Kamalakannan M, Alagesan J and Prathap L. Pelvic floor dysfunction and its impact on quality of life among female health care employees. Work 2023; 74: 1055-1059.
[14] Wang B, Wu W, Xu K and Wu H. MicroRNA-223-3p is involved in fracture healing by regulating fibroblast growth factor receptor 2. Bioengineered 2021; 12: 12040-12048.
[15] Kumar S, Chu A, Theis T, Rastogi S, Costea DM, Banerjee R, Das BC, Yarmush ML, Hsia H, Cohen R, Schachner M and Berthiaume F. Self-assembled fibroblast growth factor nanoparticles as a therapeutic for oxidant-induced neuronal and skin cell injury. ACS Appl Bio Mater 2024; 7: 5158-5170.
[16] Cheng J, Zhao ZW, Wen JR, Wang L, Huang LW, Yang YL, Zhao FN, Xiao JY, Fang F, Wu J and Miao YL. Status, challenges, and future prospects of stem cell therapy in pelvic floor disorders. World J Clin Cases 2020; 8: 1400-1413.
[17] Xiao X, Hong Y, Wang S, Ma M and Xu Z. Diagnostic value of plasma heparin-binding protein and the heparin-binding protein-to-albumin ratio in patients with community-acquired Pneumonia: a retrospective study. BMC Infect Dis 2023; 23: 777.
[18] Wu YL, Yo CH, Hsu WT, Qian F, Wu BS, Dou QL and Lee CC. Accuracy of heparin-binding protein in diagnosing sepsis: a systematic review and meta-analysis. Crit Care Med 2021; 49: e80-e90.
[19] Li L, Tian XX, Feng GL and Chen B. The predictive value of heparin-binding protein for bacterial infections in patients with severe polytrauma. PLoS One 2024; 19: e0300692.
[20] Layuk N, Wahyuni S and Arifuddin S. Differences of heparin binding protein levels in preeclampsian and non preeclampsian women. Int J Health Sci 2021.
[21] Guo W, Gao X, Zhan R, Zhao Z, Xu K and Tang B. Tricolor imaging of MMPs to investigate the promoting roles of inflammation on invasion and migration of tumor cells. Talanta 2021; 222: 121525.
[22] Murad-Regadas SM, Vilarinho ADS, Borges L, Veras LB, Macedo M and Lima DMDR. Correlation between pelvic floor dysfunction on dynamic 3D ultrasound and vaginal delivery, parity, and age in women with obstructed defecation symptoms. Arq Gastroenterol 2021; 58: 302-307.
[23] Silva RRL, Coutinho JFV, Vasconcelos CTM, Vasconcelos Neto JA, Barbosa RGB, B Marques M, Saboia DM and C Maia J. Prevalence of sarcopenia in older women with pelvic floor dysfunction. Eur J Obstet Gynecol Reprod Biol 2021; 263: 159-163.
[24] Chilaka C, Toozs-Hobson P and Chilaka V. Pelvic floor dysfunction and obesity. Best Pract Res Clin Obstet Gynaecol 2023; 90: 102389.
[25] Braga A and Serati M. New advances in female pelvic floor dysfunction management. Medicina (Kaunas) 2023; 59: 1010.
[26] Lemos N and Doumouchtsis SK. ICS debate article-childbirth, modes of delivery, and pelvic floor dysfunction-challenges in educating women about modes of delivery and granting an informed decision process. Neurourol Urodyn 2019; 38: 2027-2030.
[27] Jorge CH, Bø K, Chiazuto Catai C, Oliveira Brito LG, Driusso P and Kolberg Tennfjord M. Pelvic floor muscle training as treatment for female sexual dysfunction: a systematic review and meta-analysis. Am J Obstet Gynecol 2024; 231: 51-66, e51.
[28] Gao L, Ding S, Ding Y, Jin X, Chen Q, Zhou H, Li M, Wang J, Cao J and Zhang J. Symptom distribution of female pelvic floor dysfunction patients with constipation as chief complaint. Zhonghua Wei Chang Wai Ke Za Zhi 2018; 21: 798-802.