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Construction of a Predictive Model for Influencing Factors of Patent Ductus Arteriosus in Premature Infants Based on Prenatal Indicators

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Abstract: *This study aimed to develop a risk prediction model for patent ductus arteriosus (PDA) in premature infants based on prenatal and perinatal indicators. A retrospective study was conducted. Fifty premature infants diagnosed with PDA admitted to our hospital between September 2022 and September 2025 were enrolled as the PDA group, and 50 premature infants without PDA admitted during the same period were included as the control group. Prenatal, perinatal and clinical data were compared between the two groups. Multivariate logistic regression was performed to identify independent influencing factors, based on which the risk prediction model was constructed. The receiver operating characteristic (ROC) curve was used to validate the model's predictive performance. Univariate analysis showed that compared with the control group, the PDA group had significantly higher proportions of maternal gestational hypertension and prenatal infection, as well as a lower proportion of complete antenatal glucocorticoid treatment (all P<0.05). Multivariate logistic regression analysis identified that the following factors were independent predictors of PDA in premature infants (all P<0.05): lower gestational age at birth (OR=0.902, 95%CI: 0.842-0.966), lower birth weight (OR=0.897, 95%CI: 0.818-0.983), maternal gestational hypertension (OR=2.726, 95%CI: 1.192-6.235), incomplete antenatal glucocorticoid course before delivery (OR=2.710, 95%CI: 1.199-6.125), and prenatal infection (OR=2.751, 95%CI: 1.301-5.817). Model validation showed that the area under the ROC curve (AUC) of the model was 0.885 (95% CI: 0.812-0.957), indicating good discriminative ability. In conclusion, lower gestational age, low birth weight, maternal gestational hypertension, incomplete antenatal glucocorticoid treatment before delivery, and prenatal infection are independent risk factors for PDA in premature infants. The predictive model constructed based on these factors has good efficacy for early risk identification of PDA.*

Keywords: *Premature infants; Patent ductus arteriosus; Influencing factors; Predictive model*

1. Introduction

Ductus arteriosus is an important physiological pathway connecting the pulmonary artery and aorta during the fetal period, usually after birth It usually functionally closes within a few hours after birth and undergoes complete anatomical closure within several weeks[1]. However, if this process is obstructed and the ductus arteriosus remains closed 3 days after birth in term infants and 4 weeks after birth in premature infants, it is called patent ductus arteriosus (PDA)[2]. The incidence of PDA in premature infants has increased significantly, and has become one of the key factors affecting their survival rate and long-term quality of life [3]. PDA can cause hemodynamic disorders, cause a series of serious complications such as pulmonary edema, congestive heart failure, and bronchopulmonary dysplasia, and significantly increase the mortality and disability rate of premature infants [4]. At present, although there are many treatment options for premature infants with PDA, all of which are controversial and risky. Therefore, early and accurate identification of high-risk premature infants with PDA, and individualized risk assessment and hierarchical management are of great significance to optimizing clinical decision-making and improving the prognosis of children. Currently, studies have focused on the association between single factors such as bronchopulmonary dysplasia and PDA [5]. However, there is a lack of systematic analysis of the interaction of multiple factors, and most current prediction models mainly rely on the construction of indicators during the postpartum diagnosis process. The prediction model uses clinical indicators of maternal, fetal and pregnancy obtained before and immediately after birth for comprehensive risk assessment, but such models are not yet well-established. Based on this, this study aims to build a risk prediction model for the occurrence of

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PDA in premature infants based on prenatal indicators obtained before and immediately at birth, in order to provide clinicians with an early risk early warning tool to achieve high-risk premature infants. Early intervention is reported below.

2. Data and methods

2.1 General information

Fifty premature infants with PDA who were treated in our hospital from September 2022 to September 2025 were retrospectively selected as the PDA group, and 50 premature infants without PDA were selected as the control group. **Inclusion criteria:** (1) All infants were premature. (2) Transferred to the neonatal intensive care unit of our hospital for treatment, and the hospital stay was ≥72 hours. (3) Single fetus. (4) Bedside cardiac ultrasound completed within 7 days of birth. (5) Complete clinical data. **Exclusion criteria:** (1) Transfer or death within 7 days of birth. (2) Combined cardiac structural malformations other than PDA. (3) Congenital malformations or chromosomal genetic abnormalities. This study was approved by the ethics committee of the hospital.

2.2 Data collection

Indicators available before and immediately after birth are collected through the hospital's electronic medical record system and perinatal information management system. Including (1) fetal and birth indicators: birth age, birth weight, gender, delivery method (natural labor/cesarean section), 1-minute and 5-minute Apgar scores. (2) Maternal pregnancy indicators: maternal age, presence of hypertensive disorder complicating pregnancy, gestational diabetes, prenatal infection (clinically diagnosed as chorioamnionitis), premature rupture of membranes (duration ≥18 hours) and other pregnancy complications. (3) Intervention measures: Whether the mother applied a full course of glucocorticoid therapy before delivery (defined as two intramuscular injections of dexamethasone or betalutamide completed within 24 hours to 7 days before delivery). All data were independently extracted and entered by two uniformly trained researchers.

2.3 Relevant definitions

At present, there is no unified standard for the diagnosis of PDA internationally, and comprehensive evaluation needs to be carried out based on clinical symptoms and echocardiography results. Based on relevant literature from Practical Neonatology[6] and HamrickSEG et al [7], the diagnostic criteria for PDA are set as follows: In terms of clinical symptoms, at least one of continuous murmur at the second intercostal space on the left margin of the sternum, enhanced beats in the precordial area, heart rate at rest>180 beats/min, watery pulse, increased pulse pressure difference or decreased diastolic pressure, and worsening respiratory conditions are required. In terms of echocardiography, at least two of the inner diameter of arterial catheter>1.5mm, left-to-right shunt, left atrium to aortic root inner diameter ratio>1.4, and mitral blood flow E/A ratio ≥1.0 must be met. Those who meet the above clinical and ultrasound criteria are diagnosed with PDA.

2.4 Statistical methods

Data were processed using SPSS28.0 statistical software. Measurement data were expressed as ($\bar{x} \pm s$), and inter-group comparisons were performed using independent sample t-test; counting data were expressed as n (%), and χ^2 test was used. The factors influencing PDA were identified through multivariate logistic regression analysis, with only statistically significant items from the univariate analysis being included. The receiver operating characteristic (ROC) curve was plotted to analyze the model's predicted value for PDA occurrence. $P<0.05$ was considered statistically significant.

3. Results

3.1 Univariate analysis of PDA in premature infants

Compared with the control group, the PDA group had significantly lower gestational age, birth weight, and the proportion of mothers receiving a complete course of prenatal glucocorticoid treatment,

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and significantly higher proportions of mothers with gestational hypertension and prenatal infection (P<0.05). There was no significant difference in other clinical data between the two groups (P>0.05), as shown in Table 1.

Table 1 Univariate analysis of PDA incidence in premature infants [$\bar{x} \pm s$, n (%)]

Project	PDA group (n=50)	Control group (n=50)	χ^2/t	P
Birth age (weeks)	32.04±2.15	34.28±1.77	5.687	<0.001
Gender of premature infants			0.361	0.548
Male	28(56.00)	25(50.00)		
Female	22(44.00)	25(50.00)		
Delivery method			0.457	0.499
Vaginal delivery	15(30.00)	12(24.00)		
Cesarean section	35(70.00)	38(76.00)		
Birth weight (g)	1671.65±322.45	1880.35±410.42	2.827	0.005
Mother's age (years)	27.61±2.14	27.25±2.06	0.856	0.393
Mother's gestational hypertension	18(36.00)	8(16.00)	5.198	0.023
Mother's gestational diabetes	7(14.00)	10(20.00)	0.638	0.424
Mother's prenatal application of complete course of glucocorticoid treatment	20(40.00)	36(72.00)	10.390	0.001
Prenatal infection	15(30.00)	6(12.00)	4.882	0.027
Fetal membrane rupture time≥18 hours	22(44.00)	15(30.00)	2.102	0.147
1-minute Apgar score≤7 points	19(38.00)	11(22.00)	3.048	0.081
5-minute Apgar score≤7 points	8(16.00)	3(6.00)	2.554	0.110

3.2 Multiple factor analysis of PDA in premature infants

Taking the occurrence of PDA as the dependent variable (0=no occurrence of PDA, 1=occurrence of PDA), and 5 statistically significant items in the factor analysis as independent variables. The values were assigned as follows: birth age (original value input), birth weight (original value input), maternal hypertension during pregnancy (0=No, 1=Yes), maternal prenatal application of a full course of glucocorticoid treatment (0=Yes, 1=No), prenatal infection (0=No, 1=Yes). Multivariate logistic regression analysis results showed that birth age, birth weight, maternal hypertension during pregnancy, mother's failure to apply a full course of glucocorticoid therapy before delivery, and prenatal infection were independent influencing factors for the development of PDA in premature infants (P<0.05), as shown in Table 2.

Table 2 Multivariate logistic regression analysis of PDA in premature infants

Variable	β	SE	$wald\chi^2$	OR	P	95%CI
Birth age	-0.103	0.035	8.660	0.902	0.003	0.842~0.966
Birth weight	-0.109	0.047	5.378	0.897	0.020	0.818~0.983
Mother's gestational hypertension	1.003	0.422	5.649	2.726	0.017	1.192~6.235
Mother's prenatal use of complete course of glucocorticoid therapy	0.997	0.416	5.744	2.710	0.016	1.199~6.125
Prenatal infection	1.012	0.382	7.018	2.751	0.008	1.301~5.817

3.3 Construction and verification of a risk prediction model for premature infants with PDA

Based on the independent factors selected in Table 2, a risk prediction model for premature infants

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developing PDA was constructed. Logit (*P*) = birth age×(-0.103)+birth weight×(-0.109) + mother's gestational hypertension×1.003+mother's prenatal use of complete course of glucocorticoid therapy×0.997+prenatal infection×1.012 + 6.254. ROC curve analysis showed that the area under the curve was 0.885, the 95% *CI* was 0.812~0.957, the sensitivity was 0.820, and the specificity was 0.900, indicating that the model has good discrimination, as shown in Figure 1.

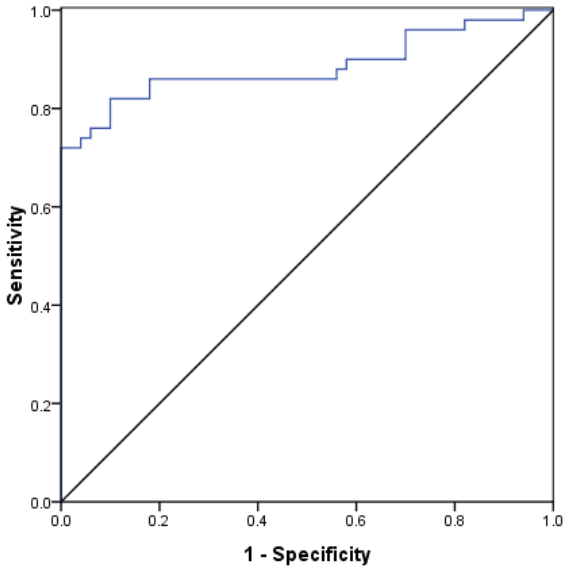

Figure 1 ROC curve of predictive model predicting the occurrence of PDA in premature infants

4. Conclusion

PDA is one of the most common circulatory complications in premature infants, and its occurrence is significantly associated with poor prognosis. Studies have shown that the overall incidence of PDA in premature infants can reach 12.9%, and among premature infants with a gestational age of less than 34 weeks, this proportion can rise to more than 17.7% [8]. The core of its pathophysiology is that the continuous opening of the arterial duct leads to left-to-right shunting of the systemic circulation to the pulmonary circulation, resulting in increased pulmonary blood flow and overloading the pulmonary vascular bed, which can induce or aggravate pulmonary edema, respiratory dysfunction, and even progress to respiratory failure. Therefore, early identification of high-risk factors for PDA and effective intervention are crucial to improve clinical outcomes in premature infants.

Through multivariate logistic regression analysis, this study concluded that birth age, birth weight, maternal hypertension during pregnancy, not receiving a full course of prenatal glucocorticoid therapy, and prenatal infection were independent factors affecting the development of PDA in premature infants. Closure of the arterial duct is a complex physiological process that relies on an increase in partial oxygen pressure, a decrease in circulating prostaglandin levels, and the functional maturation of smooth muscle cells in the duct wall. The younger the gestational age and lower the weight of premature infants, the more imperfect the smooth muscle development of the ducts and the slower the contractile response to oxygen. At the same time, the more immature the prostaglandin metabolic pathway in the body, resulting in the continuous opening of the ducts, thus the higher the risk of PDA development. This finding is basically consistent with the study of Wang Shanshan et al [9] that the incidence of PDA in premature infants with gestational age is higher. In this study, failure to receive a full course of prenatal corticosteroid therapy was an independent risk factor for PDA. Glucocorticoids can significantly promote the synthesis and release of fetal lung surfactants, improve lung maturity, thereby reducing the incidence and severity of severe respiratory distress syndrome after birth [10]. Severe respiratory distress syndrome is often accompanied by hypoxemia, acidosis, and hypercapnia, which are environmental factors that strongly inhibit catheter contraction and keep it open [11]. In this study, maternal hypertension disorder complicating pregnancy was considered an independent factor in PDA. Hypertensive disorder complicating pregnancy is often accompanied by uteroplacental poor perfusion, which may cause the fetus to be in a state of chronic hypoxia and stress, which may affect the development and adaptive regulation of the fetal cardiovascular system, and may cause the responsiveness of catheter smooth muscle to postnatal elevated oxygen. Change, thereby increasing the

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risk of PDA [12]. Prenatal infection was confirmed to be an independent factor in PDA. Intrauterine infection can trigger a systemic inflammatory response in the fetus and release a large amount of prostaglandin E2, which is one of the strongest known vasodilators and can directly act on the arterial ductus to relax and maintain it open [13]. Therefore, in premature infants exposed to intrauterine infections, circulating prostaglandin levels may remain high after birth, seriously hindering physiological closure of the catheter, leading to PDA. The prediction model constructed in the study was verified by the ROC curve, and the area under the curve reached 0.885, indicating that the model can effectively predict the risk of developing PDA in premature infants.

In summary, birth age/weight, prenatal infection, failure to receive a full course of prenatal glucocorticoid therapy, and maternal hypertension are all independent factors for the occurrence of PDA in premature infants. The predictive model built based on the above variables has predictive value for the occurrence of PDA.

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