The Relationship between Morphological and Functional Features of Preterm Placentas and the Results of Bacteriological Examination of the Discharge from the Cervical Canal of Women with Preterm Birth

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Abstract

The aim of this study was to determine the relationship between the morpho-functional features of placentas from preterm births (PBs) with the results of bacteriological examination of the discharge from the cervical canal in women with spontaneous preterm birth (SPB). The study included 227 pregnant women at gestational age between 28 and 36 weeks and 6 days, who underwent examination in the period from 2017 to 2018. Depending on the gestational age, they were divided into 3 groups. In general, a strong relationship was found between the presence of pathological changes in placentas and the presence of opportunistic flora in women ($R=0.722$, $P<0.001$). The constructed mathematical models make it possible to determine, with a high degree of certainty, the main bacteria for all the studied groups of pregnant women, thereby identifying the risk group of women at the stage of pregnancy planning, predicting complications and increasing the possibility of deliver a full term baby. (**International Journal of Biomedicine. 2019;9(4):313-319.**)

Key Words: preterm birth • placenta • opportunistic flora • placental insufficiency

Introduction

Preterm birth (PB) is a multifactorial syndrome with a variety of risk factors and long-term health consequences for the child.⁽¹⁻³⁾ Every year, an estimated 15 million babies are born preterm (before 37 completed weeks of gestation), and this number is rising.⁽³⁾ Approximately 1 million children die each year due to complications of PB.⁽⁴⁾ Common causes of PB include multiple pregnancies, infections and chronic conditions such as diabetes and high blood pressure.⁽³⁾

There is a large body of evidence that a cascade of activations of cellular components and mediators of inflammatory pathways results in onset of labor and membrane rupture.^(5,6) Supporting the fetus through the preceding gestation, the placenta is a very critical

organ in explaining the pathogenesis of spontaneous preterm birth (SPB). Placental pathology provides important diagnostic information to ascertain the cause of PB. Intra-amniotic infection, as is well known, is one risk factor of SPB.⁽⁶⁻⁸⁾ Bacterial infection and the subsequent inflammatory response are recognized as an important cause of PB. It is hypothesized that these organisms ascend the cervical canal, colonize placental tissues, cause chorioamnionitis and in severe cases infect amniotic fluid and the fetus.⁽⁹⁾ However, after 32 weeks' gestation, infection may be a less common cause of PB; instead, many cases of spontaneous preterm labor leading to PB appear to be caused by placental insufficiency (PI).⁽¹⁾ The pathoanatomical investigation of placentas from PBs is useful for assessing the etiology of SPB and the prognosis for the child.⁽¹⁰⁾

The aim of this study was to determine the relationship between the morpho-functional features of placentas from PBs with the results of bacteriological examination of the discharge from the cervical canal in women with SPB.

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Materials and Methods

Our study included 227 pregnant women at gestational age between 28 and 36 weeks and 6 days, who underwent examination in the period from 2017 to 2018. Depending on the gestational age, they were divided into 3 groups. Group 1 included 73 women at gestational age between 28 and 30 weeks and 6 days; Group 2 included 81 women at gestational age between 31 and 33 weeks and 6 days, Group 3 included 73 women at gestational age between 34 and 36 weeks and 6 days.

All women underwent an assessment of vaginal microecosis and the quantitative and qualitative composition of the biotope of the cervical discharge, as we described earlier.⁽⁸⁾

The examination of placentas was carried out according to a standardized scheme, including macroscopic analysis, material sampling and histological analysis in 3 stages. At Stage 1 of the histological examination, we performed an assessment of placental maturity;⁽¹¹⁾ at Stage 2 - a semi-quantitative assessment of the severity of certain structural indicators of the placenta (from 1 to 3 points); and at Stage 3 - the determination of the degree of PI by the totality of the mass of the fetus and placenta, the degree of maturity or immaturity of the villi, the severity of compensatory reactions, and involutive changes.

The study was conducted in accordance with ethical principles of the WMA Declaration of Helsinki (1964, ed. 2013) and approved by the RUDN University Ethics Committee. Written informed consent was obtained from all participants.

Statistical analysis was performed using the Statistica 8.0 software package (StatSoft Inc, USA). Baseline characteristics were summarized as frequencies and percentages for categorical variables and as mean $\pm$ SEM for continuous variables. Mann-Whitney U test and Kruskal-Wallis test were used, respectively, to compare means of 2 and 3 or more groups of variables not normally distributed. The frequencies of categorical variables were compared using Pearson χ^2 or Fisher's exact test, when appropriate. The Pearson's correlation coefficient and Spearman's rank correlation coefficient were used to determine the strength and direction of the relationship between two variables. The odds ratio (OR), its standard error and 95% confidence interval (CI) were calculated. Logistic regression was used to model dichotomous outcome variables. We calculated the sensitivity, specificity, positive predictive values, negative predictive values, and accuracy of the test results. A value of $P < 0.05$ was considered statistically significant.

Results

The average weight of placentas in Groups 1, 2 and 3 was 237.3 $\pm$ 49.1g, 228.3 $\pm$ 94.1g, and 318.1 $\pm$ 90.5g, respectively (Kruskal Wallis test: $H=18.30641$, $P=0.0001$). Macroscopically, placentas had an ovoid or irregular shape, small size, with additional bulges, more thickened in the center, with thinning at the edges. The umbilical cord was attached at the edges (paraplacental), in single observations—in the central zone. The length of the umbilical cord was in the range from 40 cm to 68 cm (an average of 46 $\pm$ 0.6 cm). It was noted that the vessels passing through the umbilical cord had a less than normal

winding and spiral course. The fetal surface of placentas was smooth, individual placentas had whitish areas. The maternal surface had smoothed or flattened cotyledons. In all studies, fine-grained calcifications were noted. Morphological changes in placentas are presented in Figures 1-8.

Fig.1. Leukocyte infiltration in placentas (H&E staining, x400).

Fig.2. Signs of productive inflammation (H&E staining, x400)

Fig.3. Signs of inflammation in the amnion (H&E staining, x400).

Fig.4. Intermediate chorionic villi with a small number of capillaries, sometimes surrounded by fibrinoid masses (H&E staining, x400).

(A)

(B)

Fig.5 (A,B). Intermediate chorionic villi (the 1st and 2nd ramifications). In many fields of view, the capillaries of the supporting and intermediate villi are "empty" (H&E staining; x400)

(A)

(B)

Fig.6 (A,B). Diffuse inflammation with foci of calcareous deposits in the form of calcifications in decidual tissue.

In some places, the immature terminal chorionic villi are closely adjacent to each other; therefore, the intervillous space is weakly expressed. The chorionic villi are partially surrounded by a fibrinoid roller. There is plethora of immature villi and their "desolation" in some places. In the intervillous space, erythrocyte "fields" are noted, as well as foci of calcareous deposits in the form of calcifications (H&E staining, x 400).

Fig.7. Pathological immaturity of terminal chorionic villi (H&E staining, x 400).

Fig.8. Choriodecidualite. Severe leukocyte infiltration within the decidua and trophoblastic layers. Moderate edema of spongy space. (H&E staining; A - x400, B - x200).

The data from the correlation analysis of the relationship between the state of the placenta and the data from bacteriological examination of the cervical discharge are presented in Tables 1-9.

Table 1.

Placentas with histological signs of inflammation and necrotic changes in Group 1

Group 1	Signs + n=36	Signs - n=37	P	OR (95% CI)	R
E.coli	6(16.7%)	12(32.4%)	0.1153	0.51(0.22-1.22)	
Candida spp	4(11.1%)	6(16.2%)	0.5259	0.69(0.21-2.23)	
Enterococcus faecalis	15(41.7%)	12(32.4%)	0.4139	1.28(0.7-2.35)	
Str. agalactiae	10(27.8%)	3(8.1%)	0.0281	3.43(1.03-11.44)	0.472 P=0.003
Staph. epidermidis	10(27.8%)	4(10.8%)	0.0656	2.57(0.89-7.45)	0.307 P=0.042
Staph. aureus	3(8.3%)	4(10.8%)	0.7193	0.77(0.19-3.2)	
Staph. haemolyticus	8(22.2%)	2(5.4%)	0.0367	4.11(0.94-18.06)	0.328 P=0.029
Enterobacter aerogenes	9(25%)	2(5.4%)	0.0193	4.63(1.07-19.95)	0.472 P=0.003
Acinebacter	10(27.8%)	3(8.1%)	0.0281	3.43(1.03-11.44)	0.603 P<0.001
Str. viridans	3(8.3%)	6(16.2%)	0.3057	0.51(0.14-1.9)	
Staph. warneri	5(13.9%)	7(18.9%)	0.5621	0.73(0.26-2.1)	
Serratia marcescens	3(8.3%)	5(13.5%)	0.4787	0.62(0.16-2.39)	

Table 2.

Placentas with histological signs of pathological immaturity in Group 1

Group 1	Signs+ n=38	Signs- n=35	P	OR (95% CI)	R
E.coli	6(15.8%)	12(34.3%)	0.0670	0.46(0.19-1.09)	
Candida spp.	4(10.5%)	6(17.1%)	0.4114	0.61(0.19-2)	
Enterococcus faecalis	20(52.6%)	7(20%)	0.0039	2.63(1.27-5.45)	0.472 P=0.003
Str. agalactiae	10(23.7%)	3(8.6%)	0.0818	2.76(0.81-9.39)	0.307 P=0.042
Staph. epidermidis	11(28.9%)	3(8.6%)	0.0272	3.38(1.03-11.11)	0.328 P=0.029
Staph. aureus	3(7.9%)	4(11.4%)	0.6085	0.69(0.17-2.87)	
Staph. haemolyticus	3(7.9%)	7(20%)	0.1329	0.39(0.11-1.41)	
Enterobacter aerogenes	5(13.2%)	6(17.1%)	0.6345	0.77(0.26-2.29)	
Acinebacter	10(26.3%)	3(8.6%)	0.0477	3.07(0.92-10.25)	0.405 P=0.008
Str. viridans	8(21.1%)	1(2.9%)	0.0182	7.37(0.97-55.97)	0.419 P=0.005
Staph. warneri	4(10.5%)	8(22.9%)	0.1556	0.46(0.15-1.4)	
Serratia marcescens	3 (7.9%)	5 (14.3%)	0.3825	0.55 (0.14- .14)	

Table 3.

Placentas with histological signs of placental insufficiency and hypertension in Group 1

Group 1	Signs + n=28	Signs - n=45	P	OR (95% CI)	R
E.coli	12(42.9%)	6(13.3%)	0.0070	3.21(1.36-7.59)	0.378 P=0.008
Candida spp.	9(32.1%)	1(2.2%)	0.0003	14.46(1.94-108.11)	0.405 P=0.004
Enterococcus faecalis	15(53.6%)	12(26.7%)	0.0206	2.01(1.11-3.64)	0.326 P=0.031
Staph. haemolyticus	7(25%)	6(13.3%)	0.2295	1.88(0.70-5.01)	
Staph. epidermidis	9(32.1%)	5(11.1%)	0.0265	2.89(1.08-7.76)	0.339 P=0.027
Staph. aureus	5(17.9%)	2(4.4%)	0.0584	4.02(0.84-19.32)	0.386 P=0.008
Enterobacter aerogenes	9(32.1%)	2(4.4%)	0.0013	7.23(1.68-31.07)	0.563 P<0.001
Acinebacter	5(17.9%)	8(17.8%)	0.9931	1(0.36-2.77)	
Str. viridans	8(28.6%)	1(2.2%)	0.0009	12.86(1.7-97.37)	0.559 P<0.001
Staph. warneri	3(10.7%)	9(20%)	0.2979	0.54(0.16-1.81)	
Serratia marcescens	6(21.4%)	2(4.4%)	0.0239	4.82(1.04-22.25)	0.350 P=0.014

In Group 1, the number of placentas with signs of inflammation and necrotic changes was significantly higher in women with isolated *Acinetobacter*, *Strep. agalactiae*, *Staph. haemolyticus*, and *Enterobacter aerogenes*. The number of placentas with signs of pathological immaturity was higher in women with isolated *Acinetobacter*, *Enterococcus faecalis*, *Staph. Epidermidis*, and *Str. Viridans*. The number of placentas with signs of PI and hypertension was higher in women with isolated *E. coli*, *Candida spp.*, *Enterococcus faecalis*, *Staph. Epidermidis*, *Enterobacter aerogenes*, *Str. Viridans*, and *Serratia marcescens*.

Table 4.

Placentas with histological signs of inflammation and necrotic changes in Group 2

Group 2	Signs + n=47	Signs - n=34	P	OR (95% CI)	R
E.coli	6(12.8%)	9(26.5%)	0.1171	0.48(0.19-1.23)	
Candida spp.	4(8.5%)	8(23.5%)	0.0604	0.36(0.12-1.1)	
Enterococcus faecalis	14(29.8%)	4(11.8%)	0.0542	2.53(0.91-7.02)	0.260 P=0.024
Str. agalactiae	6(12.8%)	4(11.8%)	0.8925	1.09(0.33-3.55)	
Staph. epidermidis	11(23.4%)	4(11.8%)	0.1832	1.99(0.69-5.72)	0.267 P=0.033
Staph. aureus	3(6.4%)	6(17.6%)	0.1114	0.36(0.1-1.35)	
Staph. haemolyticus	5(10.6%)	7(20.6%)	0.2135	0.52(0.18-1.49)	
Enterobacter aerogenes	8(17%)	2(5.9%)	0.1326	2.89(0.66-12.78)	0.257 P=0.036
Acinebacter	12(25.5%)	2(5.9%)	0.0210	4.34(1.04-18.15)	0.817 P<0.001
Str. viridans	8(17%)	1 (2.9%)	0.0466	5.79(0.76-44.13)	0.484 P<0.00
Staph. warneri	10(21.3%)	1 (2.9%)	0.0174	7.23(0.97-53.87)	0.491 P<0.001
Serratia marcescens	3(6.4%)	3 (8.8%)	0.6789	0.72(0.16-3.37)	

In Group 2, the number of placentas with signs of inflammation and necrotic changes was significantly higher in women with isolated *Str. Viridans*, *Acinetobacter*, and *Staph. Warneri*. The number of placentas with signs of pathological immaturity was higher in women with isolated *E. coli*, *Candida spp.*, *Enterococcus faecalis*, *Staph. Epidermidis*, and *Acinetobacter*. The number of placentas with signs of PI and hypertension was higher in women with isolated *E. coli*, *Staph. aureus*, *Staph. haemolyticus*, *Acinetobacter*, and *Str. Viridans*.

In Group 3, the number of placentas with signs of inflammation and necrotic changes was significantly higher in women with isolated *Enterococcus faecalis* and *Staph. Warneri*. The number of placentas with signs of pathological immaturity was higher in women with isolated *Enterococcus faecalis* and *Staph. aureus*. The number of placentas with signs of PI and hypertension was higher in women with isolated *E. coli*, *Staph. aureus*, and *Acinetobacter*.

Table 5.

Placentas with histological signs of pathological immaturity in Group 2

Group 2	Signs + n=48	Signs - n=33	P	OP (95% CI)	R
E.coli	13(27.1%)	2(6.1%)	0.0167	4.47(1.08-18.51)	0.319 P=0.041
Candida spp.	1(2.1%)	11(33.3%)	0.0001	0.06(0.01-0.46)	
Enterococcus faecalis	15(31.3%)	3(9.1%)	0.0184	3.44(1.08-10.94)	0.335 P=0.001
Strep. agalactiae	4(8.3%)	6(18.2%)	0.1855	0.46(0.14-1.5)	
Staph. epidermidis	13(27.1%)	2(6.1%)	0.0167	4.47(1.08-18.51)	0.527 P<0.001
Staph. aureus	8(16.7%)	1(3%)	0.0550	5.5(0.72-41.92)	0.493 P<0.001
Staph. haemolyticus	6(12.5%)	6(18.2%)	0.4794	0.69(0.24- 1.95)	
Enterobacter aerogenes	7(14.6%)	3(9.1%)	0.4603	1.6(0.45-5.76)	
Acinebacter	12(25%)	2(6.1%)	0.0268	4.13(0.99-17.23)	0.607 P<0.001
Str. viridans	8(16.7%)	1(3%)	0.0550	5.5(0.72-41.92)	0.476 P<0.001
Staph. warneri	4(8.3%)	7(21.2%)	0.0964	0.39(0.12-1.24)	
Serratia marcescens	2 (4.2%)	4 (12.1%)	0.1792	0.34 (0.07-1.77)	

Table 6.

Placentas with histological signs of PI and hypertension in Group 2

Group 2	Signs + n=44	Signs - n=37	P	OP (95% CI)	R
E.coli	14(31.8%)	1(2.7%)	0.0008	11.77(1.62-85.36)	0.793 P<0.001
Candida spp.	9(20.5%)	3(8.1%)	0.1192	2.52(0.74-8.64)	0.283 P=0.051
Enterococcus faecalis	1(2.3%)	17(45.9%)	0.0000	0.05(0.01-0.35)	
Str. agalactiae	4(9.1%)	6(16.2%)	0.3315	0.56(0.17-1.84)	
Staph. epidermidis	6(13.6%)	9(24.3%)	0.2174	0.56(0.22-1.43)	
Staph. aureus	8(18.2%)	1(2.7%)	0.0272	6.73(0.88-51.35)	0.317 P=0.009
Staph. haemolyticus	3(6.8%)	9(24.3%)	0.0272	0.28(0.08-0.96)	
Enterobacter aerogenes	8(18.2%)	2(5.4%)	0.0816	3.36(0.76-14.87)	0.276 P=0.032
Acinebacter	12(27.3%)	2(5.4%)	0.0095	5.05(1.21-21.12)	0.727 P<0.001
Str. viridans	8(18.2%)	1(2.7%)	0.0272	6.73(0.88-51.35)	
Staph. warneri	3(6.8%)	8(21.6%)	0.0527	0.32(0.09-1.1)	
Serratia marcescens	2(4.5%)	4(10.8%)	0.2835	0.42(0.08-2.17)	

Table 7.**Placentas with histological signs of inflammation and necrotic changes in Group 3**

Group 3	Signs + n=49	Signs - n=24	P	OR (95% CI)	R
E.coli	13(26.5%)	2(8.3%)	0.0707	3.18(0.78-12.99)	0.324 <i>P</i> <0.001
Candida spp.	4(8.2%)	6(25.0%)	0.0859	0.33(0.10-1.05)	
Enterococcus faecalis	25(51%)	6(25%)	0.0346	2.04(0.97-4.3)	0.6483 <i>P</i> <0.001
Str. agalactiae	6(12.2%)	3(12.5%)	0.9752	0.98(0.27-3.58)	
Staph. epidermidis	11(22.4%)	4(16.7%)	0.5657	1.35(0.48-3.79)	
Staph. aureus	30(61.2%)	9(37.5%)	0.0563	1.63(0.93-2.87)	0.358 <i>P</i> =0.009
Staph. haemolyticus	7(14.3%)	5(20.8%)	0.4783	0.69(0.24-1.94)	
Enterobacter aerogenes	8(16.3%)	7(29.2%)	0.2021	0.56(0.23-1.36)	
Acinebacter	12(24.5%)	7(29.2%)	0.6688	0.84(0.38-1.86)	
Str. viridans	8(16.3%)	1(4.2%)	0.1377	3.92(0.52-29.56)	
Staph. warneri	4(8.2%)	6(25%)	0.0494	0.33(0.1-1.05)	
Serratia marcescens	3(6.1%)	4(16.7%)	0.1506	0.37(0.09-1.51)	

Table 8.**Placentas with histological signs of pathological immaturity in Group 3**

Group 3	Signs + n=48	Signs - n=25	P	OR (95% CI)	R
E.coli	13(27.1%)	2(8%)	0.0555	3.39(0.83-13.84)	0.324 <i>P</i> <0.001
Candida spp	5(10.4%)	5(20%)	0.2977	0.52(0.17-1.63)	
Enterococcus faecalis	15(31.3%)	16(64%)	0.0072	0.49(0.29-0.82)	0.577 <i>P</i> <0.001
Str. agalactiae	5(10.4%)	4(16%)	0.4911	0.65(0.19-2.21)	
Staph. epidermidis	9(18.8%)	6(24%)	0.5983	0.78(0.31-1.95)	
Staph. aureus	32(66.7%)	7(28%)	0.0017	2.38(1.23-4.61)	0.713 <i>P</i> <0.001
Staph. haemolyticus	7(14.6%)	5(20%)	0.5535	0.73(0.26-2.06)	
Enterobacter aerogenes	7(14.6%)	8(32%)	0.0805	0.46(0.19-1.11)	
Acinebacter	12(25%)	7 28%)	0.7816	0.89(0.4-1.98)	
Str. viridans	8(16.7%)	1(4%)	0.1183	4.17(0.55-31.47)	0.256 <i>P</i> =0.057
Staph. warneri	5(10.4%)	5(20%)	0.2585	0.52(0.17-1.63)	
Serratia marcescens	3(6.3%)	4(16%)	0.1794	0.39(0.09-1.61)	

Table 9.**Placentas with histological signs of PI and hypertension in Group 3**

Group 3	Signs + n=48	Signs - n=25	P	OR (95% CI)	R
E.coli	14(29.2%)	1(4%)	0.0116	7.29(1.02-52.3)	0.493 <i>P</i> <0.001
Candida spp.	9(18.8%)	1(4%)	0.0820	4.69(0.63-34.94)	0.304 <i>P</i> =0.001
Enterococcus faecalis	24(50%)	7(28%)	0.0711	1.79(0.9-3.56)	0.277 <i>P</i> =0.012
Str. agalactiae	5(10.4%)	4(16%)	0.4911	0.65(0.19-2.21)	
Staph. epidermidis	9(18.8%)	6(24%)	0.5983	0.78(0.31-1.95)	
Staph. aureus	32(66.7%)	7(28%)	0.0017	2.38(1.23-4.61)	0.372 <i>P</i> =0.001
Staph. haemolyticus	7(14.6%)	5(20%)	0.5535	0.73(0.26-2.06)	
Enterobacter aerogenes	7(14.6%)	8(32%)	0.0805	0.46(0.19-1.11)	
Acinebacter	16(33.3%)	3(12%)	0.0487	2.78(0.89-8.64)	0.309 <i>P</i> =0.002
Str. viridans	8(16.7%)	1(4 %)	0.1183	4.17(0.55-31.47)	
Staph. warneri	6(12.5%)	4(16%)	0.6798	0.78(0.24-2.52)	
Serratia marcescens	5(10.4%)	2(8%)	0.7393	1.3(0.27-6.24)	

In general, a strong relationship was found between the presence of pathological changes in placentas and the presence of opportunistic flora in women ($R=0.722$, $P<0.001$). The risk of developing pathological changes in the placenta increased by more than 6 times ($OR=6.14$, 95% $CI=2.32-9.48$) with the presence of opportunistic flora. Binary logistic regression analysis made it possible to obtain a mathematical model that reliably identifies significant bacteria, the presence of which allows us to predict the revealed changes in the structures of placentas (Table 10-12).

Table 10.**Mathematical model for placentas with signs of inflammation and necrotic changes**

Logistic regression (logit) Dep. Var: placentas with signs of inflammation and necrotic changes. Loss: Max likelihood (MS-err. scaled to 1) Final loss: 99.064595337 Chi-square (3)=69.181 $P=.00000$

	Const.B0	E.coli -	Staph. aureus	Acinebacter
Estimate	0.2001	-1.5361	-1.672	-2.1480
Standard Error	0.2144	0.4014	0.79112	0.771
t(228)	0.93308	-3.826	-2.1134	-2.783
P-value	0.3517	0.0001	0.03564	0.0058
-95%CL	-0.222	-2.3271	-3.2308	-3.668
+95%CL	0.6226	-0.7451	-0.11314	-0.627
Wald's Chi-square	0.870	14.643	4.46662	7.749
P-value	0.3507	0.00013	0.03457	0.0053
Odds ratio (unit ch)	1.2215	0.215	0.18787	0.116
-95%CL	0.8005	0.0975	0.0395	0.025
+95%CL	1.863	0.474	0.89301	0.533
Odds ratio (range)		0.215	0.18787	0.1167
-95%CL		0.097	0.039	0.025
+95%CL		0.474	0.893	0.533

Classification of Cases OR: 40.04 Perc. correct: 84.14%

	Pred. - 1	Pred. - 0	Percent - Correct
1	127	7	94.78
0	29	64	68.82

Mathematical model for placentas with signs of inflammation and necrotic changes: sensitivity -77.27%, specificity-78.95%, and accuracy -77.97%

Table 11.

Mathematical model for placentas with signs of pathological immaturity

Logistic regression (logit) Dep. Var: placentas with signs of pathological immaturity. Loss: Max likelihood (MS-err. scaled to 1) Final loss: 110.43963104 Chi-square 3)=90.738 $P=0.0000$

	Const.B0	Staph. aureus	Candida spp.	Staph. epidermidis
Estimate	-1.2375	3.8453	2.8023	1.4181
Standard Error	0.18690	0.62581	1.104293	0.3792
t(228)	-6.6211	6.1445	2.5377	3.7395
P-value	<0.0001	<0.0001	0.01182	0.0002
-95%CI	-1.6058	2.61222	0.62646	0.670
+95%CL	-0.86927	5.0784	4.97832	2.165
Wald's Chi-square	43.84013	37.754	6.4400	13.98
P-value	<0.0001	<0.0001	0.01116	0.00018
OR (unit ch)	0.2900	46.77496	16.484	4.1295
-95%CL	0.2007176	13.629	1.8709	1.956
+95%CL	0.4192561	160.52	145.23	8.718
OR (range)		46.774	16.484	4.129
-95%CL		13.629	1.8709	1.956
+95%CL		160.52	145.2301	8.718

Classification of Cases OR: 40.04 Perc. correct: 84.14%

	Pred. - 1	Pred. - 0	Percent - Correct
1	127	7	94.78
0	29	64	68.82

Mathematical model for placentas with signs of pathological immaturity: sensitivity - 94.78% (95% CI: 89.61%-97.45%), specificity - 68.82% (95% CI: 58.81% - 77.33%), and accuracy -84.14% (95% CI: 78.29%-88.64%)

Table 11.

Mathematical model for placentas with signs of PI and hypertension

Model: Logistic regression (logit) Dep. var: placentas with signs of PI and hypertension. Loss: Max likelihood (MS-err. scaled to 1) Final loss: 106.99411981 Chi-square (3)=100.20 $P=0.0000$

	Const.B0	Enterobacter aerogenes	Candida spp.	E.coli -
Estimate	0.5749	1.606	4.4158	-2.9758
Standard Error	0.22071	0.618	1.10256	0.3818
t(240)	2.60481	2.5963	4.00511	-7.7929
p-value	0.00976	0.0100	<0.0001	<0.0001
-95%CL	0.14013	0.3875	2.2439	-3.7281
+95%CL	1.0097	2.825	6.5878	-2.2236
Wald's Chi-square	6.7850	6.7412	16.04094	60.729
P-value	0.00919	0.0094	<0.0001	<0.0001
OR (unit ch)	1.7769	4.9844	82.75615	0.05100
-95%CL	1.1504	1.473	9.4305	0.0240
+95%CL	2.744813	16.861	726.209	0.1082
OR (range)		4.984437	82.75615	0.0510
-95%CL		1.47343	9.430586	0.0240
+95%CL		16.86175	726.209	0.10821

Classification of Cases OR: 17.60 Perc. correct: 80.62%

	Pred. - 1	Pred. - 0	Percent - Correct
1	95	25	79.17
0	19	88	82.24

Mathematical model for placentas with signs of PI and hypertension: sensitivity -79.17% (95% CI: 71.05%-85.47%), specificity - 82.24% (95% CI: 73.92%-88.33%), and accuracy - 80.62% (95% CI: 74.28%-85.69%).

Conclusion

In pregnant women, the growth of opportunistic flora in diagnostically significant titers in the discharge from the cervical canal has a negative effect on the morphological and functional state of the placental complex in SPB. The constructed mathematical models make it possible to determine, with a high degree of certainty, the main bacteria for all the studied groups of pregnant women, thereby identifying the risk group of women at the stage of pregnancy planning, predicting complications and increasing the possibility of delivering a full-term baby.

Competing Interests

The authors declare that they have no competing interests.

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