ORIGINAL ARTICLE

Human Papillomavirus infection in pregnancy: Clinical and perinatal outcomes in a regional cohort and modern approaches to management

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ABSTRACT

Background and Aim: Human papillomavirus (HPV) is among the most common sexually transmitted infections and may be associated with alterations in placental and endocrine function, potentially contributing to adverse pregnancy outcomes. This study aimed to evaluate clinical, hormonal, and perinatal outcomes of HPV-associated genital lesions during pregnancy in Azerbaijan.

Methods: A total of 206 pregnant women were examined at Maternity Hospital No. 1 and Antenatal Clinic No. 6 between 2015 and 2022. The study group included 152 women with HPV-associated lesions confirmed by colposcopy, cytology, histology, and polymerase chain reaction (PCR); 54 HPV-negative women served as controls. Clinical, hormonal, microbiological, and instrumental assessments (ultrasound, cardiotocography) were performed. Neonates underwent nasopharyngeal PCR testing 8–12 hours after birth.

Results: HPV-positive women had significantly higher rates of threatened miscarriage (28.3% vs 11.1%), anemia (24.3% vs 7.4%), intrauterine infection (18.4% vs 5.6%), and preterm birth (21.1% vs 9.3%) compared with controls. Neonatal complications included low birth weight (19.1% vs 7.4%), intrauterine growth restriction (12.5% vs 3.7%), and reduced Apgar scores (<7 in 15.8% vs 5.6%). Hormonal assays showed decreased β-human chorionic gonadotropin (β-hCG), placental lactogen, and α-fetoprotein (AFP) levels in HPV-positive women. HPV DNA was detected in 32.2% of neonatal nasopharyngeal swabs (49/152), confirming vertical transmission.

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Conclusions: HPV infection during pregnancy is associated with adverse obstetric and perinatal outcomes and reduced levels of key placental endocrine markers. These findings underscore the importance of early detection, HPV vaccination, and monitoring of placental endocrine function. However, observed hormonal changes should be interpreted as associations rather than confirmed mechanistic effects. (www.actabiomedica.it)

Key words: Human Papillomavirus, pregnancy, vertical transmission, obstetric complications, neonatal outcomes

Introduction

Human papillomavirus (HPV) is one of the most prevalent sexually transmitted infections worldwide, affecting up to 40% of women of reproductive age. High-risk genotypes, particularly HPV 16 and 18, are strongly associated with cervical intraepithelial neoplasia (CIN) and cervical cancer, whereas low-risk types (HPV 6 and 11) typically cause benign genital warts (1). Pregnancy alters the course of HPV infection due to immunological and hormonal changes. These modifications contribute to HPV persistence, progression of cervical lesions, and higher recurrence rates (2,3). Previous studies have demonstrated increased risk of obstetric complications in HPV-positive women, including miscarriage, preterm birth, intrauterine infection, and anemia, as well as adverse neonatal outcomes such as intrauterine growth restriction (IUGR) and low Apgar scores (4-6). Vertical transmission of HPV is an important concern, with viral DNA detected in 5–25% of neonates (7), suggesting possible perinatal or intrapartum exposure. Despite extensive global research, regional data from the Caucasus and Eastern Europe remain limited (8). The present study evaluates maternal and perinatal outcomes in HPV-positive pregnant women in Azerbaijan, while integrating these findings with updated international evidence. Recent studies also suggest that HPV infection may influence placental endocrine function. Altered secretion of β-hCG, placental lactogen, and α-fetoprotein (AFP) has been associated with impaired trophoblastic activity and increased risk of miscarriage, preterm birth, and intrauterine growth restriction (9-11). Therefore, evaluation of hormonal markers in HPV-positive pregnancies provides important insights into the mechanisms of adverse outcomes. Although the precise biological pathways remain insufficiently defined, experimental and in vitro data suggest that the viral oncoproteins E6 and E7 of high-risk HPV types can interact with tumor suppressor proteins p53 and pRb, potentially contributing to trophoblastic apoptosis and reduced placental hormone synthesis. In addition, HPV-associated inflammation—characterized by elevated cytokines such as IL-6, TNF-α, and VEGF—may alter placental angiogenesis and oxygen exchange, offering a plausible explanation for endocrine and functional disturbances observed in some clinical studies (5). These proposed mechanisms remain unverified in human placental tissue, but they provide a biologically plausible framework for understanding multifactorial HPV-related obstetric complications. The primary aim of this study was to evaluate maternal and perinatal outcomes in pregnant women with human papillomavirus (HPV) infection in a regional cohort from Azerbaijan. The secondary aims were: 1) to assess the association between HPV infection and alterations in key placental endocrine markers, including β-human chorionic gonadotropin (β-hCG), placental lactogen, and alpha-fetoprotein (AFP); 2) to analyze the relationship between HPV infection and adverse obstetric and neonatal outcomes, such as miscarriage, preterm birth, intrauterine growth restriction, and neonatal condition at birth; 3) to evaluate the frequency of vertical HPV transmission by detecting viral DNA in neonatal nasopharyngeal samples; 4) to contextualize the obtained regional clinical data within current

international evidence in order to contribute to a better understanding of potential mechanisms underlying HPV-associated pregnancy complications.

Patients and methods

Study design and setting

This observational clinical study was conducted between 2015 and 2022 at the Educational-Surgical Clinic by Azerbaijan Medical University. The research protocol was approved by the institutional ethics committee, and informed consent was obtained from all participants.

Study population

206 pregnant women were enrolled. The study group included 152 women with HPV-associated genital lesions confirmed by colposcopy, cytology, histology, and PCR. The control group consisted of 54 HPV-negative women, verified by negative PCR testing and absence of clinical or colposcopic evidence of HPV.

Group stratification

HPV-positive women were subdivided according to HPV genotype profile and lesion severity:

- Group I (n = 88): single low-risk or single high-risk HPV type, ASC-US/LSIL.
- Group II (n = 64): multiple HPV genotypes and/or high-risk types (including HPV 16/18), or HSIL.

This stratification allowed assessment of the relationship between HPV burden and clinical, hormonal, and perinatal outcomes.

Maternal age and gestational age

The mean maternal age was 27.4 ± 4.8 years with no significant differences between groups (P = 0.42). Women were enrolled between 11 and 36 weeks of gestation, most during the second trimester (52.3%). Gestational age at inclusion did not differ significantly between groups.

Diagnostic procedures

All women underwent standard gynecological and obstetric examinations. The diagnostic protocol included colposcopy, cytology, histopathology, PCR, microbiological testing, ultrasound, and cardiotocography.

Biochemical assessment

Biochemical evaluation focused on key markers of placental function and fetal development, including β-human chorionic gonadotropin (β-hCG), placental lactogen, and α-fetoprotein (AFP).

Interpretation of hormonal markers

Decreased β-hCG and placental lactogen levels were interpreted as indicators of trophoblastic insufficiency and impaired placental hormone production.

AFP levels were evaluated in the context of fetoplacental function, with reduced concentrations considered consistent with diminished fetoplacental production or transfer, particularly when occurring together with reduced β-hCG and placental lactogen.

Timing of blood collection

Venous blood sampling was performed between 12 and 14 weeks of gestation for all participants to minimize physiological variability in hormonal levels. Samples were collected in the morning after an overnight fast. Serum was obtained by centrifugation at 3000 rpm for 10 minutes and stored at –20°C until analysis. Concentrations of β-hCG (mIU/mL), placental lactogen (μg/mL), and AFP (mIU/mL) were measured by enzyme-linked immunosorbent assay (ELISA) using certified commercial kits (Diagnostic Systems, Germany). All measurements were performed in duplicate, with internal quality control samples included in each run. These parameters were selected as the most informative biochemical indicators of placental endocrine function and maternal–fetal status. Together, they provided objective criteria for evaluating placental function and predicting perinatal complications in HPV-associated pregnancy.

Neonatal evaluation

To assess vertical transmission, PCR testing for HPV DNA was performed using nasopharyngeal swabs obtained 8–12 hours after birth (7).

Statistical analysis

Data were analyzed using SPSS software (version 16.0; IBM Corp., Armonk, NY, USA). Categorical variables were analyzed using the χ^2 test, whereas continuous variables were evaluated using Student's *t*-test or the Mann–Whitney *U* test, as appropriate. Correlation analysis was performed using Pearson's correlation coefficient. A *P* value of <0.05 was considered statistically significant. Normality of data distribution was assessed using the Kolmogorov–Smirnov test.

Results

Among 206 pregnant women enrolled in the study, 152 were HPV-positive and 54 were HPV-negative (controls). The mean maternal age did not differ significantly between groups (P = 0.42; Student's t-test). HPV-positive women had a significantly higher incidence of adverse maternal outcomes compared with controls (χ^2 test). The most frequent complication was threatened miscarriage, which occurred in 43 of 152 women (28.3%) in the HPV-positive group and 6 of 54 women (11.1%) in the control group (P = 0.009; χ^2 test). Anemia was observed in 37 of 152 (24.3%) versus 4 of 54 (7.4%) (P = 0.021; χ^2 test). Intrauterine infection was diagnosed in 28 of 152 (18.4%) and 3 of 54 (5.6%), respectively (P = 0.034; χ^2 test). Preeclampsia complicated 22 of 152 pregnancies (14.5%) compared with 2 of 54 (3.7%) in controls (P = 0.028; χ^2 test). Preterm birth before 37 weeks occurred in 32 of 152 (21.1%) HPV-positive women versus 5 of 54 (9.3%) HPV-negative women (P = 0.047; χ^2 test). Neonatal complications were more frequent among infants born to HPV-positive mothers. Apgar scores <7 at 1 minute were observed in 24 of 152 neonates (15.8%) in the HPV-positive group compared with 3 of 54 (5.6%) in the control group (P = 0.041; χ^2 test). Low birth weight (<2500 g) was recorded in 29 of 152 neonates (19.1%) versus 4 of 54 (7.4%) in controls (P = 0.043; χ^2 test). Intrauterine growth restriction occurred in 19 of 152 (12.5%) compared with 2 of 54 (3.7%) (P = 0.039; χ^2 test). Respiratory distress was registered in 16 of 152 neonates (10.5%) and in 2 of 54 controls (3.7%) (P = 0.048; χ^2 test). PCR analysis of nasopharyngeal swabs obtained 8–12 hours after birth revealed HPV DNA in 49 of 152 neonates (32.2%) born to HPV-positive mothers, while none of the 54 control infants tested positive (P < 0.001; χ^2 test). In addition, hormonal studies revealed significantly reduced β-hCG, placental lactogen, and α-fetoprotein levels in HPV-positive women compared with controls (Table 1). Detailed analysis of maternal serum hormone concentrations demonstrated a consistent decline in all three placental markers among HPV-infected pregnant women (n = 152) compared with controls (n = 54). In Group I (n = 88), mean β-hCG concentration was 9435 ± 92 mIU/mL, while in Group II (n = 64) it was further reduced to 8470 ± 107 mIU/mL, compared with 9786 ± 71 mIU/mL in controls (P = 0.018 and P = 0.006, respectively; Student's t-test). Placental lactogen levels also decreased progressively — 8.78 ± 0.23 μg/mL and 6.03 ± 0.27 μg/mL versus 10.62 ± 0.32 μg/mL in controls (P = 0.004 and P < 0.001, respectively; Student's t-test). A similar trend was noted for α-fetoprotein, which declined to 73.7 ± 1.95 mIU/mL and 66.8±2.50 mIU/mL versus 91.3±2.60 mIU/mL in controls (P < 0.001 for both comparisons; Student's t-test).

Table 1. Maternal serum hormone levels in study groups.

Marker (units)	Group I (n=88)	Group II (n=64)	Control (n=54)
β-hCG (mIU/mL)	9435±92*	8470 ±107**	9786±71
Placental lactogen (μg/mL)	8.78±0.23**	6.03±0.27***	10.62±0.32
α-fetoprotein (mIU/mL)	73.7±1.95***	66.8±2.50***	91.3±2.60

*P<0.05; **P<0.01; ***P<0.001 vs control.

Ultrasound findings

Ultrasound examination revealed additional indicators supporting placental dysfunction in HPV-positive pregnancies. Although fetal biometric parameters were largely within normal limits except for cases of intrauterine growth restriction, several functional placental markers differed between groups. Placental maturity grades were advanced for gestational age in 21 of 152 HPV-positive women (13.8%) compared with 2 of 54 controls (3.7%) (P = 0.028; χ^2 test). Placental thickness below the 10th percentile was observed in 17 of 152 women (11.2%) in the study group versus 1 of 54 (1.9%) in controls (P = 0.036; χ^2 test). Placental thickness below the 10th percentile was defined according to gestational age-specific reference ranges (12). Increased uterine artery resistance (RI > 0.58) or early diastolic notching was detected in 23 of 152 HPV-positive pregnancies (15.1%) compared with 3 of 54 (5.6%) in controls (P = 0.041; χ^2 test). A decrease in maternal serum α-fetoprotein was observed in HPV-positive women and occurred concurrently with reduced β-hCG and placental lactogen levels. Correlation analysis demonstrated significant associations between reduced β-hCG and placental lactogen levels and the frequency of threatened miscarriage (r = −0.46; P < 0.01; Pearson's correlation) and preterm birth (r = −0.39; P < 0.05; Pearson's correlation). AFP levels were moderately correlated with low neonatal birth weight (r = −0.33; P < 0.05; Pearson's correlation).

Threatened miscarriage occurred more frequently in the second trimester, particularly in Group II, compared to Group I and controls (Table 2, Figure 1). The incidence was highest in Group II, which included women with multiple or high-risk HPV types. In the first trimester, threatened miscarriage was registered in 8 of 88 women (9.1%) in Group I, 13 of 64 (20.3%) in Group II, and 0 of 54 (0%) in the control group (P = 0.012; χ^2 test). During the second trimester, the complication occurred in 14 of 88 (15.9%), 21 of 64 (32.8%), and 1 of 54 (1.9%), respectively (P < 0.001; χ^2 test). In the third trimester, threatened miscarriage was recorded in 3 of 88 (3.4%), 8 of 64 (12.5%), and 1 of 54 (1.9%) women (P = 0.041; χ^2 test). Overall, 45 of 152 HPV-positive women (29.6%) experienced at least one episode of threatened miscarriage during pregnancy, compared with 2 of 54 (3.7%) in the control group (P < 0.001; χ^2 test).

The analysis of delivery modes revealed that vaginal birth was associated with a higher rate of neonatal HPV DNA positivity compared with cesarean section (Table 3, Figure 2). However, neonatal HPV DNA was also detected in a small proportion of infants delivered by cesarean section.

Among HPV-positive mothers in Group I, 16 of 83 vaginal deliveries (19.3%) resulted in HPV DNA detection in newborns, whereas none of the 5 cesarean-delivered neonates (0%) tested positive (P = 0.048; χ^2 test). In Group II, neonatal HPV DNA was found in 32 of 50 vaginally delivered infants (64.0%) and in 1 of 14 cesarean-delivered infants (7.1%) (P < 0.001; χ^2 test). Overall, 49 of 152 neonates (32.2%) born to HPV-positive mothers had detectable HPV DNA in nasopharyngeal swabs taken 8–12 hours after birth, while none of the 54 infants (0%) in the control group were positive (P < 0.001; χ^2 test).

Table 2. Threatened miscarriage by trimester and study group.

Trimester	Group I (%)	Group II (%)	Control (%)
1st trimester	9.09	20.31	0.00
2nd trimester	15.91	32.81	1.85
3rd trimester	3.41	12.50	1.85

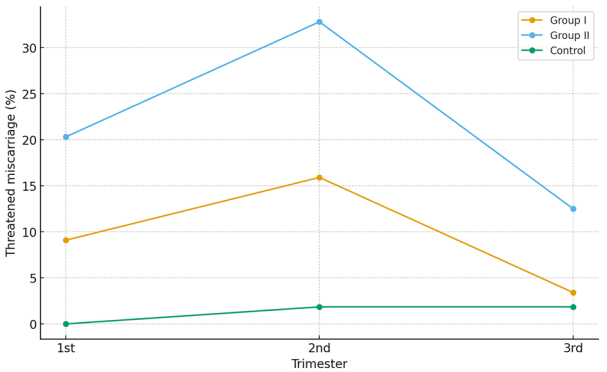

Figure 1. Incidence of threatened miscarriage across pregnancy trimesters in Group I, Group II, and the control group. Data are presented as percentages (χ^2 test).

Table 3. Neonatal HPV PCR positivity by delivery mode and maternal group.

Delivery mode	Group	N (deliveries)	Neonatal HPV PCR positive
Vaginal delivery	Group I	83	16 (19.3%)
Vaginal delivery	Group II	50	32 (64.0%)
Cesarean section	Group I	5	0 (0%)
Cesarean section	Group II	14	1(7.1%)

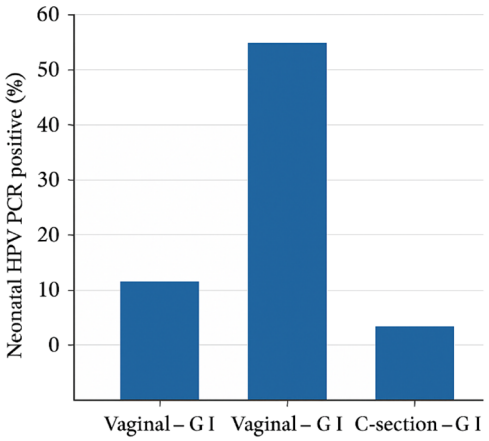

Figure 2. Neonatal HPV PCR positivity (%) by delivery mode and group.

Discussion

Our study demonstrates a clear association between maternal HPV infection and adverse obstetric and neonatal outcomes, including miscarriage, preterm birth, intrauterine infection, and neonatal complications. These findings are consistent with recent meta-analyses reporting an increased risk of preterm delivery and premature rupture of membranes among HPV-positive women (1–3). Notably, the concurrent reduction of β-hCG, placental lactogen, and α-fetoprotein is suggestive of a pattern of trophoblastic insufficiency; within this interpretative framework, lower α-fetoprotein may reflect diminished fetoplacental production or transfer rather than increased placental permeability. Mechanistically, HPV infection has been proposed to affect trophoblastic behavior and placental vascularization through modulation of angiogenic factors such as VEGF and PlGF, as well as activation of proinflammatory signaling pathways including NF-κB. Previous studies by Condrat et al. (5) and Li et al. (12) have reported that E6/E7 expression in experimental and placental model systems can downregulate p53-dependent apoptotic control and reduce β-hCG secretion (5,11). Elevated levels of IL-6 and TNF-α have also been implicated in impairment of endocrine and barrier functions of the placenta, potentially contributing to inflammation-associated hypoxia and metabolic stress (9,13). In the present study, the findings are consistent with a possible association between maternal HPV infection and altered placental endocrine function; however, a direct causal relationship cannot be established. In this context, the observed reductions in placental hormone levels may provide indirect clinical support for placental involvement in HPV-positive pregnancies and could contribute to the increased rates of miscarriage, preterm birth, and intrauterine growth restriction observed in our cohort. Nevertheless, these interpretations remain hypothetical, as placental tissue was not analyzed to verify viral presence at the molecular level. In line with these mechanistic considerations, HPV infection appears to be associated with signs of reduced placental endocrine activity, including decreases in AFP, β-hCG, and placental lactogen, which may reflect functional trophoblastic hypofunction rather than direct viral involvement or fetal structural abnormalities. The observed reduction in AFP can be more plausibly interpreted as a marker of impaired fetoplacental production or transfer in the context of HPV-associated placental dysfunction, rather than as evidence of congenital anomalies or direct viral damage to placental tissue. Accordingly, the hypothesis regarding endocrine effects of HPV remains biologically plausible but currently unconfirmed and requires further validation through molecular and histopathological analyses of placental tissue. Based on the current dataset and methodology, a causal relationship between HPV infection and decreased AFP levels

cannot be established; rather, the findings indicate an association that supports, but does not prove, this hypothesis. The detection of HPV DNA in 49 of 152 neonatal nasopharyngeal swabs (32.2%) indicates the occurrence of vertical HPV transmission, most likely during the intrapartum period, although transplacental transmission cannot be excluded. This proportion exceeds many previously reported ranges of 5–25% (9). Although most neonatal HPV infections are considered transient, persistent detection of viral DNA has been suggested to be associated with rare but clinically relevant conditions such as juvenile-onset recurrent respiratory papillomatosis. Taken together, these observations highlight the potential clinical relevance of considering maternal HPV status and placental endocrine markers during pregnancy, particularly in regions with limited access to routine screening programs (1,6,8,9). In the present study, higher rates of neonatal HPV DNA detection were observed following vaginal delivery compared with cesarean section, which is consistent with the hypothesis that intrapartum exposure may represent a major route of vertical transmission. However, the detection of HPV DNA in a small proportion of neonates delivered by cesarean section indicates that alternative routes, including transplacental or ascending infection, cannot be completely excluded. In addition to clinical complications, our analysis demonstrated lower levels of β-hCG, placental lactogen, and AFP in HPV-positive women compared with controls (Table 1). These hormonal alterations may reflect impaired placental function and appear to be associated with higher rates of threatened miscarriage and preterm birth in this group (9–11). Similar associations have been reported in Russian and Chinese cohorts, in which HPV-positive pregnancies were characterized by reduced placental hormone secretion and an increased frequency of adverse obstetric outcomes (13,14). In this context, the higher frequency of threatened miscarriage observed in HPV-positive women, particularly among those with multiple or high-risk genotypes, appears to be most pronounced during the second trimester. This period coincides with peak placental endocrine activity, suggesting that functional placental vulnerability during mid-gestation may contribute to the observed association between HPV infection and miscarriage risk. However, this interpretation remains speculative and should be considered in light of the observational design of the study. Collectively, the findings of this study indicate that maternal HPV infection is associated with adverse pregnancy outcomes and alterations in placental endocrine markers. However, all conclusions regarding placental endocrine impairment should be interpreted strictly as associations rather than evidence of direct causality. Further prospective studies incorporating molecular verification of HPV in placental tissue are required to clarify whether HPV plays a direct pathogenic role in placental dysfunction or represents an indirect marker of increased obstetric risk.

Limitations and future directions

This study has several limitations. HPV genotyping was not performed, which might have clarified correlations between viral subtype and outcome severity. Another important limitation is the lack of molecular analysis of placental tissue: neither PCR for HPV DNA nor evaluation of viral protein expression (E6/E7, p53, cytokines) was conducted. This prevents direct confirmation of viral presence in the placenta and limits mechanistic interpretation of the observed endocrine abnormalities, which therefore remain associative rather than causally proven. In addition, the present study did not include systematic postnatal follow-up beyond the neonatal period. Future research should incorporate molecular assessment of placental biomarkers (VEGF, HIF-1α, TNF-α) and long-term neonatal follow-up to evaluate potential sequelae of vertical transmission. Therefore, all conclusions regarding placental endocrine impairment should be interpreted as associations rather than evidence of direct causality.

Conclusions

HPV infection during pregnancy is associated with an increased risk of maternal complications, adverse perinatal outcomes, and vertical transmission. In addition to these clinical effects, our findings highlight the role of altered placental hormone secretion (reduced β-hCG, placental lactogen, and AFP)

as potential predictors of adverse outcomes. Early detection, regular monitoring, and preventive strategies, including HPV vaccination, are essential for improving obstetric prognosis. These findings provide region-specific evidence from Azerbaijan and support the inclusion of endocrine monitoring in future management strategies. Integrating endocrine marker screening (β-hCG, placental lactogen, AFP) into the obstetric care of HPV-positive women may improve early prediction and prevention of pregnancy complications. The link between HPV infection and placental endocrine alterations appears plausible but remains unconfirmed mechanistically. Because placental tissue was not evaluated for HPV DNA, the mechanistic link between maternal HPV infection and placental endocrine impairment should be considered hypothetical and requires further molecular investigation.

Ethical approval: All study procedures were reviewed and approved by the Ethics Committee of Azerbaijan Medical University (protocol No. 19, November 10, 2015). The protocol entitled *"Human Papillomavirus Infection in Pregnancy: Clinical and Perinatal Outcomes in a Regional Cohort and Modern Approaches to Management"* was considered compliant with bioethical standards, and the research was authorized for implementation.

Conflict of interest: Each author declares that he or she has no commercial associations (e.g., consultancies, stock ownership, equity interest, patent/licensing arrangement, etc.) that might pose a conflict of interest in connection with the submitted article.

Authors' contribution: LM: Conceptualization, study design, data collection, laboratory analyses, interpretation of results, and drafting of the manuscript. SS: Supervision of the research process, guidance in manuscript writing, and critical review of the final version. SI: Methodology & Editing. SG: Statistical analysis, data visualization, and assistance with data interpretation. All authors have read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Declaration on the use of AI: None.

Consent for publication: Written consent was obtained from the parents (father and mother) of the newborns. Information was provided regarding the research methods, the results obtained, and the use of the findings for scientific purposes.

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