Published in final edited form as:
Ophthalmic Epidemiol. 2009 ; 16(6): 337–345. doi:10.3109/09286580903144746.

Risk factors for neonatal conjunctivitis in babies of HIV-1 infected mothers

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Abstract

Purpose—To determine the prevalence and correlates of neonatal conjunctivitis in infants born to human immunodeficiency virus type 1 (HIV-1) infected mothers.

Methods—This was a nested case-control study within a perinatal HIV-1 cohort. HIV-1 seropositive mothers were enrolled during pregnancy and mother-infant pairs followed after delivery with assessment for neonatal conjunctivitis at 48 hours and up to 4 weeks after birth. Genital infections (chlamydia, gonorrhea, syphilis, trichomonas, bacterial vaginosis, and candida) were screened for at 32 weeks gestation. Mothers received treatment for genital infections diagnosed during pregnancy and short-course zidovudine. Newborns did not receive ocular prophylaxis at hospital deliveries. Multivariate logistic regression models were used to determine cofactors for neonatal conjunctivitis overall and stratified for infant HIV-1 status.

Results—Four hundred and fifty-two infants were assessed and 101 (22.3%) had neonatal conjunctivitis during the first month postpartum. In multivariate analyses using odds ratios (OR) and confidence intervals (CI), neonatal conjunctivitis was associated with neonatal sepsis (adjusted OR 21.95, 95% CI 1.76, 274.61), birth before arrival to hospital (adjusted OR 13.91, 95% CI 1.39, 138.78) and birth weight (median 3.4 versus 3.3 kilograms, p=0.016, OR 1.79, 95% CI 1.01, 3.15). Infant HIV-1 infection was not associated with conjunctivitis.

Conclusions—Despite detection and treatment of genital infections during pregnancy, neonatal conjunctivitis was frequently diagnosed in infants born to HIV-1 infected mothers suggesting a need for increased vigilance and prophylaxis for conjunctivitis in these infants. Neonatal sepsis, birth before arrival to hospital, and higher birthweight are factors that may predict higher risk of neonatal conjunctivitis in this population.

Keywords

Case-control; HIV; Maternal; Neonatal conjunctivitis; Risk factors

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Declaration of interest: The authors declare no conflict of interest.

INTRODUCTION

Neonatal conjunctivitis is a significant cause of childhood corneal blindness.[1] Neonatal conjunctivitis is caused by infectious or chemical etiologies including sexually transmitted infections (STIs) in pregnancy.[2,3] STIs associated with neonatal conjunctivitis include *N. gonorrhea, C. trachomatis* and Herpes simplex virus type 2.[4,5,6] Other infectious causes include *S. aureus, N. meningitidis* and Group B streptococcus.[7,8,9] However, microbial growth in culture from conjunctival smears may only be detected in about 50% of cases.[10] Prolonged labor and preterm rupture of membranes have been associated with neonatal conjunctivitis and are postulated to increase the risk of conjunctivitis via ascending genital infection.[4,11,12] Prematurity is associated with poorly developed ocular surface defense mechanisms and has also been associated with neonatal conjunctivitis.[13] Pathogens may infect the neonatal conjunctiva via different routes either as a result of transplacental passage of the pathogen to the fetus or during passage of the fetus through the birth canal.[14,15] In addition, contaminated fomites may be a source of pathogens while toxic chemicals introduced to the eye may erode the ocular surface allowing access for pathogens. Pathogens such as *N. gonorrhea, C. diphtheriae* and Shigella species produce toxins that can invade intact corneal epithelium.[16–19]

The epidemiology and risk factors for neonatal conjunctivitis in the setting of maternal human immuno-deficiency virus type 1 (HIV-1) have not been defined. HIV-1 may increase susceptibility to other STIs, and because HIV-1 is predominantly a sexually acquired infection, HIV-1 infected individuals may be expected to have more STIs than the general population.[20,21] Within settings of high HIV-1 seroprevalence it is important to know whether HIV-1 infection predisposes infants to neonatal conjunctivitis and whether practices should be modified to prevent neonatal conjunctivitis. Given the likely predisposition for HIV-1 infected mothers to be at risk for STIs, it is plausible that neonatal conjunctivitis may be more prevalent in this population. Ocular involvement occurs in 20–35% of HIV-1 infected infants with conjunctivitis being the most common ocular pathology.[22,23] This study was carried out to determine the prevalence and risk factors for neonatal conjunctivitis in infants born to HIV-1 infected mothers in Nairobi, Kenya.

MATERIALS AND METHODS

Clinical Methods

A case-control study nested in a perinatal HIV-1 cohort study was conducted at the Kenyatta National Hospital (KNH) in Nairobi, Kenya. Guidelines of the Declaration of Helsinki were followed. Ethical approval was obtained from the Ethical Review Committee at KNH and the University of Washington (UW) Institutional Review Board. Infants born to HIV-1 infected mothers between November 1999 and June 2003 were included in the analysis. Mothers were enrolled antenatally prior to 32 weeks gestation and followed through delivery and for 1 year postpartum. At 32 weeks gestation a pelvic examination was conducted and vaginal specimens obtained for bacterial vaginosis, *C. albicans*, and *T. vaginalis*. A cervical swab for *N. gonorrhea* and *C. trachomatis* was obtained and a blood sample drawn for syphilis serology at the same visit. Mothers with microbiologically detectable genital infections were treated according to Kenyan Ministry of Health guidelines. Specifically, women with syphilis received 2.4 megaunits of benzathine penicillin once weekly for 3 weeks, those with gonorrhea received ceftriaxone 125 mg IM as a single dose, women with chlamydia received erythromycin 500 mg six-hourly for 7 days. Women with trichomonas or candidiasis received clotrimazole pessaries and cream. Slides for bacterial vaginosis Nugent scores were batched and read after completion of the study and were unavailable to guide clinical treatment of bacterial vaginosis during pregnancy. Women received short-

course zidovudine for prevention of mother-to-child transmission of HIV-1.[24,25] Mothers were instructed to deliver at KNH. Infants were vaginally delivered unless there was an obstetric indication for cesarean section. Tetracycline eye ointment prophylaxis was not routinely administered to infants delivered at KNH during the study period. Infants received a complete physical examination within 48 hours of life and were seen in clinic at 2 weeks, 1 month, and then monthly during the first year of life. Structured questionnaires were used during follow-up. Infants who developed purulent or watery conjunctival discharge within one month of delivery were considered to have neonatal conjunctivitis and treated with topical and systemic antibiotics. Determination of neonatal conjunctivitis was made clinically by study physicians, five of whom were pediatricians (DN, CG, DW, EO, and PO).

Laboratory Methods

Maternal genital infections were diagnosed using the following methods; *N. gonorrhea* and *C. trachomatis* were diagnosed by polymerase chain reaction (PCR) nucleic acid amplification and hybridization assay for detection of *N. gonorrhea* and *C. trachomatis* primers (Amplicor® CT/NG test, Roche Molecular Systems Inc, Branchburg, NJ, USA), *C. albicans* by potassium hydroxide (KOH) preparation, *T. vaginalis* by in-pouch culture and bacterial vaginosis by Nugent score. Syphilis serology was determined by rapid plasma reagin (RPR) testing of sera, with confirmation of positive tests by Treponema pallidum hemagglutination assay (TPHA). T-cell subsets were enumerated using Facscan (Beckton Dickinson, Mountain View, California, USA). Infant plasma HIV-1 viral loads were determined using the Gen-Probe HIV-1 viral load assay (Gen-Probe Incorporated, San Diego, USA). Infant HIV-1 infection status and timing was determined by filter paper HIV-1 deoxyribonucleic acid (DNA) and/or plasma ribonucleic acid (RNA) as previously described.[26,27]

Statistical Methods

Babies who completed 4 weeks follow up after birth were included in the analysis for prevalence and risk factors. Potential risk factors were compared for infants with versus without neonatal conjunctivitis using non-parametric tests for continuous variables and Chi-square tests for dichotomous variables. Odds ratios with 95% confidence intervals (CI) were used to measure the effect of the exposure factors studied. Multivariable logistic regression analysis was carried out to determine the relative contributions of multiple factors to neonatal conjunctivitis. To build the regression model, the likelihood ratio test was used to assess whether a variable significantly improved the multivariable equation before it was included. The significance level used was 95%. Missing values were dropped in the multivariable analysis. Analyses were also stratified for infant HIV-1 status. Stata version 7.0 statistical program was used for analysis (Stata Corporation, College Station, TX, USA).

RESULTS

The perinatal HIV-1 cohort study enrolled 510 HIV-1 infected mothers. Delivery data were available for 476 (93.3%). The remaining 34 (6.7%) were lost to follow-up prior to delivery. Of the 476 infants with delivery data there were 468 (98.3%) live births, 7 (1.5%) stillbirths and 1 (0.2%) intrapartum death (Figure 1).

Characteristics of the mother-infant pairs enrolled in the cohort study are shown in Table 1. The median maternal age was 25 years ((interquartile range (IQR) 22–28)). Median maternal CD_4 count at 32 weeks gestation was 437 cells/mm^3 (IQR 306–618 cells/mm^3). The median $\log_{10}$ HIV-1 RNA level was 4.75 $\log_{10}$ copies/ml (IQR 4.20–5.28) at 32 weeks gestation and 4.08 $\log_{10}$ copies/ml (IQR 3.48–4.74) at delivery following short-course zidovudine. At 32

weeks gestation, 246 (50.9%) women had abnormal vaginal discharge, 10 (2.1%) had genital ulcers on pelvic examination and 299 (61.9%) women had genital infections with some having multiple infections; 164 (38.4%) had bacterial vaginosis, 147 (30.6%) had vulvovaginal candidiasis, 77 (16.1%) had trichomonas, 20 (4.2%) had chlamydia and 8 (1.7%) had gonorrhea. Of the 173 women tested for syphilis, 3 (1.7%) were positive.

Most (82.7%) women had vaginal deliveries. The median duration of labor was 9 hours (IQR 6–12) and 14 (2.8%) women had ruptured membranes by the time they presented for delivery. The median gestational age at delivery was 38 weeks (IQR 36–40) and 36 (7.1%) infants were born prematurely (before 37 completed weeks). The median birth weight was 3,100 gms (IQR 2850–3400). There were 101 (22.3%) cases of neonatal conjunctivitis, of which 37 (36.6%) cases were diagnosed within 48 hours of delivery and 64 (63.4%) were diagnosed after 48 hours but within the first month of life.

Risk Factors for Neonatal Conjunctivitis

In univariate analysis, the place of delivery and in particular birth before arrival to hospital [(odds ratio (OR) = 4.78, 95% CI 1.04, 21.96)], birth weight (OR= 1.76, 95% CI 1.08, 2.87), gestational age at delivery (OR=1.20, 95% CI 1.06, 1.36) and neonatal sepsis (OR=10.71, 95% CI 1.08, 106.14) were associated with neonatal conjunctivitis (Table 2). Infants with neonatal conjunctivitis did not differ from infants without neonatal conjunctivitis in terms of maternal CD4 count, maternal HIV-1 RNA viral copies, prenatal genital infections, mode of delivery, prematurity or infant HIV-1 infection status (Table 2).

On multivariate analysis, the following emerged as significant risk factors; neonatal sepsis [(adjusted (adj) OR 21.95, 95% CI 1.76, 274.61)], birth before arrival to hospital (adj OR 13.91, 95% CI 1.39, 138.78) and birth weight (adj OR=1.79, 95% CI 1.01, 3.15) (Table 3).

On stratified analyses by infant HIV-1 infection status at 1 month, none of the exposure factors was associated with conjunctivitis in HIV-1 infected infants, but several cofactors remained significantly associated with conjunctivitis in HIV-1 uninfected infants (birth weight, gestational age, with a trend for birth before arrival to hospital) (Table 4). There may not have been sufficient power to demonstrate effects in the smaller number of HIV-1 infected infants.

DISCUSSION

In this study we found a high prevalence of neonatal conjunctivitis among infants of HIV-1 infected mothers and identified some risk factors for neonatal conjunctivitis. Prevalence of neonatal conjunctivitis in this study was high (22.3%) and, surprisingly, was similar to an earlier study (23.2%) conducted by Laga and colleagues among HIV-1 uninfected mother-infant pairs in the same hospital over a decade previously in a cohort that neither received genital infection treatment during pregnancy nor ocular prophylaxis.[28] The prevalence of neonatal conjunctivitis that we observed was high despite provision of microbial testing for STIs with treatment during pregnancy. This prevalence was higher than most studies in developing country settings (3–16%).[2,29,30]

Genital infections were observed in over half of women at 32 weeks (61.9%), however we did not observe an association between any of the diagnosed treated genital infections and risk of subsequent neonatal conjunctivitis. This was likely due to successful treatment of the infections. Also, pathogens classically associated with neonatal conjunctivitis were relatively infrequent in this cohort during pregnancy—less than 2% of women had gonorrhea and about 4% had chlamydia. Among women with prenatally diagnosed gonorrhea, 14.3% had infants who developed neonatal conjunctivitis, for chlamydia 22.2%,

and trichomoniasis 18.1%. These prevalences did not differ significantly from women with no genital infection diagnosed, among whom 21% had infants with neonatal conjunctivitis. Of 101 cases of neonatal conjunctivitis, only 5 occurred in infants of women with either gonorrhea or chlamydia during pregnancy. Thus, except for bacterial vaginosis (BV), incomplete treatment or recurrence of genital infection does not adequately explain the high prevalence of neonatal conjunctivitis in this cohort.

In this study, women with microbiologically diagnosed BV did not receive prenatal BV treatment because BV scores were computed after completion of the study. Despite this, bacterial vaginosis was not a significant risk factor for neonatal conjunctivitis. Molecular studies of women with BV have identified a variety of organisms associated with BV.[31] Further studies are required to define the role of maternal BV in neonatal conjunctivitis. Treatment of BV in pregnancy is not consistently useful in reducing preterm birth or neonatal sepsis,[32] and HIV-1 infected women are more likely to have persistent bacterial vaginosis despite treatment.[33]

There is no consistently effective prophylactic treatment for neonatal conjunctivitis. Application of 1% tetracycline eye ointment, 0.5% erythromycin, or 1% silver nitrate and 1.25% or 2.5% povidone iodine either as a single or double application have been used with varying results[34,35,36] A Cochrane systematic review to evaluate the effectiveness of prophylaxis in reducing the incidence of neonatal conjunctivitis and to determine which medication is most effective is ongoing.[37]

Birth before arrival to hospital emerged as a potential risk factor for neonatal conjunctivitis in both univariate and multivariate analysis. Mothers in this situation delivered somewhere between their homes and the intended health facility. Transitioning between sites of delivery may expose infants to more bacterial pathogens such as staphylococcus.

Infant HIV-1 infection was not associated with increased risk of neonatal conjunctivitis in this cohort. This included early *in utero* HIV-1 infection and intra-partum HIV-1. It was plausible that similar cofactors could lead to conjunctivitis and HIV-1 (ruptured membranes, genital infections), however, this was not borne out in this study. Our observations suggest that infant HIV-1 is not a risk factor for neonatal conjunctivitis in HIV-1 exposed infants. In analyses stratified for HIV-1 status, risk factors remained associated with conjunctivitis among HIV-1 uninfected infants. The smaller sample size may have limited power to demonstrate significant associations in HIV-1 infected infants.

The associations we observed between birth weight and neonatal conjunctivitis (crude OR 1.76, 95% CI 1.08, 2.87) and between gestational age and neonatal conjunctivitis (crude OR 1.20, 95% CI 1.04, 1.39) were intriguing because they differ from previous studies. Prematurity and its associated low birth weight have been associated with the occurrence of neonatal conjunctivitis but on their own, they were not found significant in this study. In fact, we found the opposite—that larger infants and post-term infants were more likely to have conjunctivitis. Babies with neonatal conjunctivitis were significantly larger at birth than those without conjunctivitis (Table 2). It is possible that larger infants may have had more traumatic vaginal passage and subsequent conjunctival chemosis exposing the conjunctiva to infection.

Our study had several strengths and limitations. Strengths of the study included large sample size, prospectively collected information from pregnancy to delivery including microbiologically diagnosed genital infections, and comprehensive data on genital infections and infant infection status. Our study was limited by not having microbiologic studies of conjunctival specimens to define etiology and lack of follow up. In addition, the diagnosis of neonatal conjunctivitis was made by pediatricians rather than ophthalmologists. The clinical

criteria used for diagnosis was subjective and may have included cases of nasolacrimal duct obstruction rather than conjunctivitis.

In summary, this study suggests that neonatal conjunctivitis may be highly prevalent in infants born to HIV-1 infected women. Further bacteriological and virological studies in newborns' conjunctival flora are needed in order to better inform treatment and prophylaxis guidelines after delivery. There is need for improved implementation of ocular prophylaxis in public sector settings in the era of HIV-1. Our observations suggest the need to support enhanced interventions for ocular prophylaxis and close monitoring and treatment of neonates in high HIV-1 seroprevalence regions.

Acknowledgments

We would like to thank the women and children who participated in this study and the staff at Maternal Child Health Clinics and Kenyatta National Hospital who assisted in study implementation.

Supported by the US National Institutes of Child Health and Disease (NICHD) through grant #RO1 HD-23412. Phelgona Otieno, Carey Farquhar, Dalton Wamalwa, Grace Wariua, and Elizabeth Obimbo were scholars in the AIDS International training and Research Program, NIH Research Grant D43 TW000007, funded by the Fogarty International Center and the Office of Research on Women's Health.

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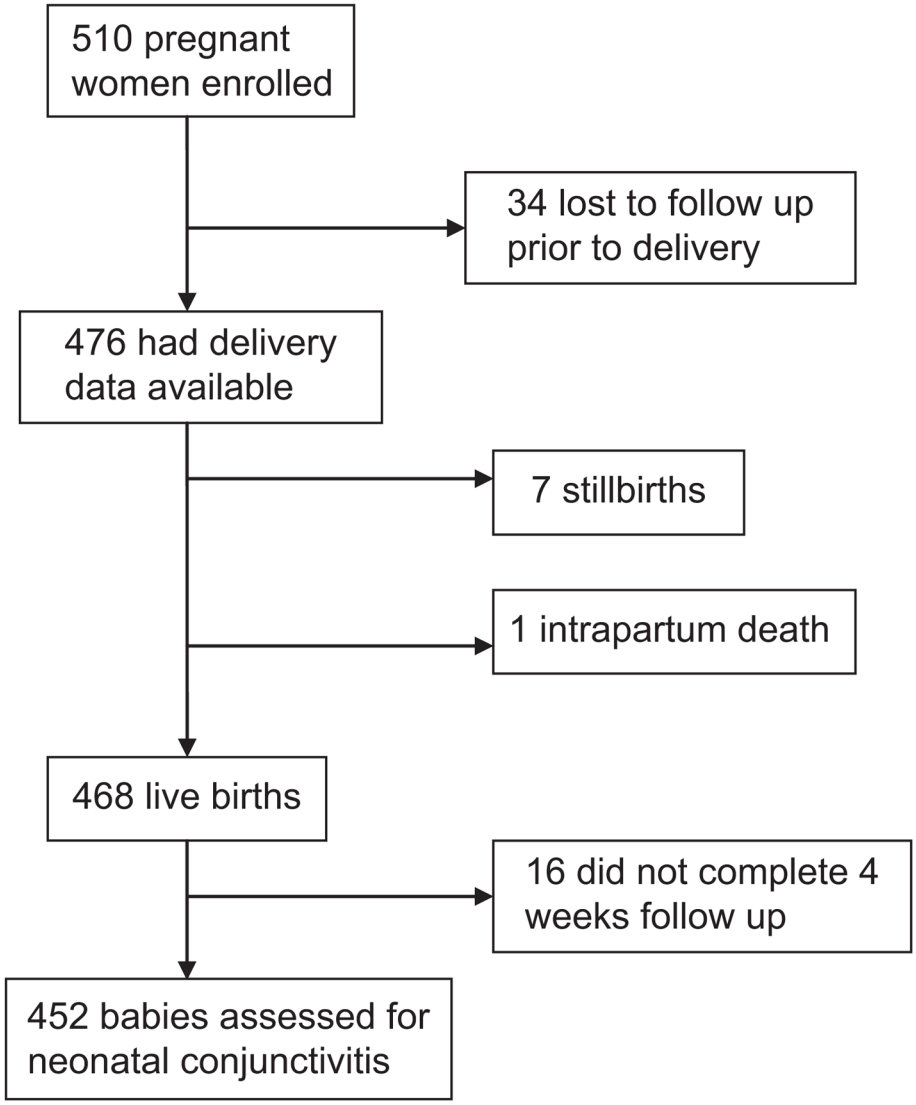

FIGURE 1.
Flow chart showing study participants.

TABLE 1

Characteristics of mothers and infants in the cohort

Characteristic	Median(IQR) or n (%)
At enrolment	
Maternal age (years)	25 (22–28)
Maternal education (years in school)	8 (7–12)
History of infections (N=510)	
STI	75 (14.7)
Syphilis	52 (10.2)
Gonorrhea	39 (7.6)
Genital ulcers	38 (7.5)
Shingles	21 (4.1)
At 32 weeks gestation	
Genital clinical examination (N=483)	
Vulvitis	11 (2.3)
Vulvar ulcers	15 (3.1)
Vulvar warts	24 (5.0)
Vaginal ulcers	2 (0.4)
Abnormal vaginal discharge	246 (50.9)
Vaginal warts	11 (2.3)
Cervical warts	7 (1.4)
Cervical ulcers	9 (1.9)
Genital infection screening by laboratory testing	
Bacterial vaginosis (N=427)	164 (38.4)
Candida (N=480)	147 (30.6)
Trichomonas (N=479)	77 (16.1)
Chlamydia (N=472)	20 (4.2)
Gonorrhea (N=481)	8 (1.7)
Syphilis (N=173)	3 (1.7)
Maternal CD4 count (cells/ml) (N=476)	437 (306–618)
<200	48 (10.1)
200–349	115 (24.2)
350–499	121 (25.4)
≥500	192 (40.3)
Maternal $\log_{10}$ HIV-RNA copies (N=468)	4.75 (4.20–5.28)
At Delivery	
Maternal $\log_{10}$ HIV-RNA copies (N=379)	4.08 (3.48–4.74)
Place of delivery(N=475)	
Kenyatta National Hospital	413 (86.9)
Other medical facility	23 (4.8)
Home	32 (6.7)
Birth before arrival to hospital (BBA)	7 (1.5)

Characteristic	Median(IQR) or n (%)
Delivered by SVD (N=468)	387 (82.7)
Breech presentation (N=441)	8 (1.8)
Duration of labour (hours) (N=447)	9 (6–12)
Duration from rupture of membranes to delivery (hours) (N=253)	1 (0–5)
Duration of second stage of labour (hours) (N=382)	10 (5–15)
Amniotic fluid stained with meconium (N=370)	87 (23.5)
Sex of baby (N=473)	
Male	244 (51.6)
Female	229 (48.4)
Birthweight (grams) (N=464)	3100 (2850–3400)
Gestational age by dates (weeks) (N=508)	38 (36–40)
Estimated maturity (weeks) (N=418)	39.9 (38.9–40.0)
Neonatal sepsis/meningitis (N=475)	4 (0.8)
Infant HIV-1 infected by DNA PCR (N=464)	65 (14)
Infant CD4 count (cells/ml) (N=197)	2236 (1664–2818)
Infant CD4% (N=197)	41 (34–47)
Neonatal conjunctivitis (N=452)	101 (22.3)

BBA–born before arrival to hospital; CD4–cluster of designation 4; DNA–deoxyribonucleic acid; IQR–interquartile range; N–number; PCR–polymerase chain reaction; RNA–ribonucleic acid; STI–sexually transmitted infections; SVD–spontaneous vertex delivery.

TABLE 2

Univariate analysis of potential risk factors in the 452 babies assessed for neonatal conjunctivitis

Exposure factor	With conjunctivitis n/N (%) Median (IQR)	Without conjunctivitis n/N (%) Median (IQR)	Crude OR (95% CI) or Mann- Whitney U test p-value
Clinical features at 32 weeks gestation			
History of STI	16/101 (15.8)	49/351 (14.0)	1.16 (0.63–2.14)
Abnormal vaginal discharge	52/98 (53.1)	179/350 (51.1)	1.08 (0.69–1.69)
Cervical ulcers	2/98 (2.0)	6/350 (1.7)	1.19 (0.24–6.01)
Maternal CD4 count (cells/ml)	683 (492–792)	463 (286–659)	0.94
Maternal HIV-RNA copies (log_{10} copies/ml)	3.87 (2.60–5.15)	4.75 (4.18–5.28)	0.80
Bacterial vaginosis	39/88 (44.3)	109/307 (35.5)	1.45 (0.89–2.34)
Candida	30/99 (30.3)	108/346 (31.2)	0.96 (0.59–1.56)
Trichomonas	13/98 (13.3)	59/346 (17.1)	0.74 (0.39–1.42)
Chlamydia	4/97 (4.1)	14/340 (4.1)	1.00 (0.32–3.12)
Gonorrhea	1/99 (1.0)	6/346 (1.7)	0.58 (0.07–4.86)
Syphillis	1/36 (2.8)	2/123 (1.6)	1.73 (0.15–19.63)
Features at birth and perinatal period			
Place of delivery; Birth before arrival to hospital (BBA)	4/101 (4.0)	3/351 (0.9)	4.78 (1.04–21.96)*
Gestational age by dates at delivery (weeks)	38.0 (37.3–39.8)	38.0 (36.3–40.0)	0.04*
Premature labor	3/98 (3.1)	18/338 (5.3)	0.56 (0.16–1.95)
Duration of labour (hours)	9.0 (6.5–12.8)	8.3 (5.3–11.8)	0.247
Time interval from rupture of membranes to delivery (hours)	1.5 (0–5.8)	1.5 (0–6.8)	0.18
Amniotic fluid stained with meconium	17/81 (21.0)	64/272 (23.5)	0.86 (0.47–1.58)
Birthweight (grams)	3425 (3125–3963)	3275 (2800–3488)	0.02*
Low birth weight (<2500gm)	5/101 (5.0)	24/341 (7.0)	0.69 (0.26–1.85)
Spontaneous vertex delivery	80/101 (79.2)	287/343 (83.7)	0.74 (0.43–1.30)
Breech presentation	3/94 (3.2)	4/325 (1.2)	2.65 (0.58–12.04)
Neonatal sepsis	3/101 (3.0)	1/351 (0.3)	10.71 (1.08–106.14)*
Maternal HIV-RNA copies at delivery (log_{10}copies/ml)	3.70 (3.23–5.00)	4.24 (3.69–4.65)	0.92
Infant HIV-1 infected (tested by DNA PCR)	12/100 (12.0)	52/350 (14.9)	0.78 (0.40–1.53)
Infant CD4 count (cells/ml)	2017 (1265–2875)	2453 (1989–2920)	0.72

* significant at the 95% confidence level.

BBA–born before arrival to hospital; CD4–cluster of designation 4; CI–confidence interval; DNA–deoxyribonucleic acid; gm–grams; HIV–human immunodeficiency virus; IQR–interquartile range; ml–milliliters; N–number; OR–odds ratio; PCR–polymerase chain reaction; RNA–ribonucleic acid; STI–sexually transmitted infections.

TABLE 3

Multivariable analysis of risk factors for neonatal conjunctivitis

Exposure factor	Crude OR (95% CI)	Adj OR (95% CI)**
Birth before arrival to hospital (BBA)	4.78 (1.04–21.96)	13.91 (1.39–138.78)*
Neonatal sepsis	10.71 (1.08–106.14)	21.95 (1.76–274.61)*
Birthweight	1.76 (1.08–2.87)	1.79 (1.01–3.15)*
Duration of labour (hours)	1.02 (0.98–1.05)	1.02 (0.98–1.07)
Premature labour at delivery	0.56 (0.16–1.95)	0.41 (0.08–2.11)
Breech presentation	2.65 (0.58–12.04)	5.97 (0.94–38.14)

* significant at the 95% confidence level.

** Adjusted for all other variables in the model.

Adj–adjusted; CI–confidence interval; OR–odds ratio;

TABLE 4

Stratified analysis by HIV-1 infection status at 1 month

	HIV-1 uninfected			HIV-1 infected by one month		
Exposure factor	**With conjunctivitis median (IQR) or number (%) N=85**	**Without conjunctivitis median (IQR) or number (%) N=306**	**Univariate analysis OR (95% CI), p-value**	**With conjunctivitis median (IQR) or number (%) N=15**	**Without conjunctivitis median (IQR) or number (%) N=57**	**Univariate analysis OR (95% CI), p-value**
Maternal CD4 at 32 weeks gestation (cells/mm^3)	487.5	483.7	0.9	381.2	424.0	0.5
Maternal HIV-1 RNA copies (log_{10}) at 32 weeks gestation	4.58	4.58	1.0	4.93	5.14	0.3
Maternal HIV-1 RNA copies (log_{10}) at delivery	3.98	4.08	0.5	3.60	4.17	0.5
Any genital infection at 32 weeks gestation	50/85 (58.8%)	179/306 (58.5%)	1.01 (0.62–1.65), 1.0	12/15 (80.0%)	40/57 (70.2%)	1.70 (0.42–6.81), 0.50
Birth before arrival to hospital (BBA)	3/85 (3.5%)	2/306 (0.7%)	5.86 (0.91–33.8), 0.09	0	0	NA
Vaginal delivery	65/85 (80%)	252/399 (84.3%)	0.75 (0.40–1.38), 0.35	11/15 (73.3%)	46/56 (82.3%)	0.60 (0.16–2.27), 0.47
Prolonged rupture of membranes (>24hrs)	1/84 (1.2%)	2/279 (0.7%)	1.67 (0.15–18.6), 0.55	0	2/51 (0.7%)	0.80 (0.71–0.91), 1.0
Gestational age at delivery (wks)	39.8	39.2	0.01	39.8	38.7	0.64
Birth weight (kgs)	3.2	3.1	0.04	3.19	2.96	0.13

BBA–born before arrival to hospital; CD4–cluster of designation 4; CI–confidence interval; HIV–human immunodeficiency virus; hrs–hours; IQR–interquartile range; kg–kilograms; mm^3–cubic millimeter; N–number; NA–not applicable; OR–odds ratio; RNA–ribonucleic acid.