Maternal Human Immunodeficiency Virus-1 Infection and Pregnancy Outcome

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Objective: To study the impact of maternal human immunodeficiency virus type 1 (HIV-1) infection on pregnancy outcome.

Methods: Between January 1989 and December 1991, 406 HIV-1-seropositive and 407 HIV-1-seronegative age- and parity-matched pregnant women from Nairobi, Kenya, all at less than 28 weeks' gestation, were recruited into a prospective study of HIV-1 infection in pregnant women and their offspring. Both groups were followed until 6 weeks postpartum.

Results: Three hundred fifteen HIV-1-seropositive women and 311 HIV-1-seronegative controls were followed until delivery. Seropositive women were younger at sexual debut and reported more lifetime partners and more sexually transmitted diseases (STDs) than the seronegative controls. The seropositive women had higher rates of genital ulcer disease (4.7 versus 2.0%; $P = .08$), genital warts (4.9 versus 2.0%; $P = .03$), and positive syphilis serology (7.9 versus 3.2%; $P < .001$), but there were no differences between the groups in isolation rates of *Neisseria gonorrhoeae* (6.8 versus 7.1%) and *Chlamydia trachomatis* (11.5 versus 9.0%). Maternal HIV-1 infection was associated with significantly lower birth weight (2913 versus 3072 g; $P = .0003$) and with prematurity (21.1 versus 9.4%; $P < .0001$), but not with small for gestational age size (4.2 versus 3.2%; $P = .7$). The stillbirth rate was higher in seropositive women, yet not statistically significant (3.8 versus 1.9%; $P = .2$). Women with a CD4 count lower than 30% had a higher risk of preterm delivery (26.3 versus 10.1%; $P < .001$). Postpartum endometritis was more common in HIV-1-infected women than in seronegative controls (10.3 versus 4.2%; $P = .01$) and was inversely correlated with the CD4 percentage. No histopathologic placental abnormalities attributable to HIV-1 were detected.

Conclusion: Maternal HIV-1 infection was significantly associated with prematurity and postpartum endometritis, but not with fetal growth retardation. There was a trend toward a higher stillbirth rate in HIV-1-seropositive mothers. *(Obstet Gynecol 1994;83:495–501)*

There is a controversy between studies from the developing world generally showing an association between maternal human immunodeficiency virus type 1 (HIV-1) infection and adverse pregnancy outcome,[1–3] and reports from the developed world that fail to demonstrate such an effect.[4–7] These discrepancies may result from methodologic and population differences. Studies from Europe and North America often contain small sample sizes and involve subjects who use intravenous drugs, known to be associated with both adverse pregnancy outcome and HIV infection. Studies from Africa usually include large sample sizes, but are sometimes loosely matched or uncontrolled for the disease stage or for other infections affecting birth weight. In addition, most African studies base the assessment of prematurity on birth weight and/or the reported gestational age in combination with a maturity score, resulting in some degree of misclassification of preterm and small for gestational age (SGA) infants.

We conducted a prospective study in HIV-1-infected pregnant women and a control group of comparable seronegative women, including obstetric ultrasound to date the pregnancy, data on malaria and sexually transmitted diseases (STDs), and clinical and immunologic disease stage. Patients were recruited from the same antenatal clinic in Nairobi where the HIV-1 seroprevalence was found to have doubled over the last 3 years (from 6.5% in 1989 to 13.0% in 1991).[8]

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Materials and Methods

The study was conducted between January 1989 and December 1991 at Langata clinic, a large health center operated by the Nairobi City Council. From Monday to Friday of each week, all new antenatal patients who presented at a gestational age of less than 28 weeks were screened for HIV-1 and syphilis antibodies after pretest counseling and informed consent.

Five thousand consecutive women were given pretest counseling and asked to participate in the study. One hundred seventeen women (2.3%) declined participation, most frequently because they wished to consult their husbands before agreeing to be tested.

The overall HIV-1 seroprevalence was 8.8% (432 of 4883), and the syphilis seroreactivity was 3.6% (173 of 4753). Almost 8% of women who were HIV-1 antibody-positive were also seroreactive for syphilis, compared to 3.2% of women who were HIV-1-seronegative (odds ratio 2.5, 95% confidence interval [CI] 1.7–3.8; $P < .001$). The HIV-1 seroprevalence rose from 6.5% in 1989 to almost 13.0% in 1991 ($P < .001$), and the syphilis seroreactivity from 2.9 to 5.3% ($P = .002$) over the same period.[8]

We enrolled HIV-1-seropositive women, as well as a similar number of HIV-1-uninfected antenatal controls matched for age and parity. At enrollment, data collection included demographic, medical, obstetric, and sexual histories; a physical examination; cervical swabs for *Neisseria gonorrhoeae* and *Chlamydia trachomatis*; a blood sample for malaria parasites and lymphocyte subsets; and an obstetric ultrasound (real-time linear scan; Toshiba SAL 30) to date the pregnancy. Routine antenatal care was provided and all women were informed and counseled with regard to their HIV serostatus. Twenty-eight weeks' gestation was chosen as the cutoff because most women presented for the first antenatal visit at a gestational age of 22–28 weeks. Further, the prediction of gestational age by sonographic measurements of the biparietal diameter is still accurate in the second trimester (±8 days), but this accuracy deteriorates in the third trimester.[9,10]

Patients were instructed to deliver in Pumwani Maternity Hospital, supervised by one of the research nurses. Pumwani Maternity Hospital is a referral hospital covering 12 smaller maternity units in Nairobi. Women attending the maternity hospital belong to the lower socioeconomic strata. The annual number of deliveries in Pumwani Maternity Hospital varies between 25,000–28,000 (over 70 deliveries daily). Follow-up visits were scheduled at 1 and 6 weeks postpartum. Physical examinations of mother and newborn were performed, cultures for STDs were repeated, and a serum sample was obtained for HIV-1 serology in the HIV-seronegative group and for syphilis serology in both groups. Postpartum endometritis was diagnosed if at least two of the following symptoms were present: fever of more than 38C, foul lochia, and uterine tenderness.

We used the World Health Organization (WHO) case definition of adult AIDS in Africa to diagnose AIDS in the mothers.[11] Caretakers were blinded to the patient's HIV serostatus. Mothers who had a medical problem (anemia, malaria, STDs, hypertension) were treated according to the Government of Kenya guidelines. Iron and vitamin supplements were given to all women enrolled in the study. Sexual partners of the women with treatable STDs also were treated when possible.

Maternal serum samples were tested by enzyme-linked immunosorbent assay (ELISA) for HIV-1 (Organon Teknika Boxtel, the Netherlands). Samples positive on repeat testing were analyzed by immunoglobulin G (IgG) Western blot (Diagnostic Biotechnology, Belgium). Sera were considered positive for HIV-1 antibody if on Western blot they showed at least one band representative of the HIV-1 core proteins and one band representative of the envelope glycoproteins. Serology for syphilis was performed using the Rapid Plasma Reagin card test (Wellcome, London, UK), and all positive results were retested with the *Treponema pallidum* hemagglutination test (Wellcome). Cervical cultures for *N gonorrhoeae* were performed on Thayer-Martin medium. *Chlamydia trachomatis* was diagnosed using the Wellcozyme ELISA test (Wellcome).

After June 1990, when a flow cytometer (Facs-scan; Becton Dickinson, Belgium) became available, maternal blood was obtained at enrollment for white blood cell count and lymphocyte subsets in all patients. Whole blood samples, drawn in heparin-containing tubes, were used to determine T-helper-inducer (CD4) cells by direct immunofluorescence using monoclonal antibodies (Becton Dickinson, CA) and with two-color flow cytometry (Becton Dickinson). Absolute numbers of CD4 were calculated using the white blood cell count and differential (Coulter Electronics, Inc., Hialeah, FL) and multiplying by the appropriate factor obtained on flow cytometry. For most analyses, we used the CD4 percentage; this seems to be more reliable because it is determined directly from flow cytometric measurements.[12] The diagnosis of malaria was based on the recognition of parasites in a blood film stained with Giemsa stain.

Placentas were collected at birth, stored in 10% formalin, and evaluated by routine hematoxylin and eosin staining. Chorioamnionitis, funisitis, and villitis were classified as light, moderate, or severe depending

Table 1. Sociodemographic Characteristics in Human Immunodeficiency Virus-1-Seropositive and -Seronegative Pregnant Women

	HIV-1 (+) (n = 406)	HIV-1 (−) (n = 407)	P	Odds ratio	95% CI
Mean age*	21.8 ± 3.9	22.1 ± 3.8	.3		
Single	12.8%	8.4%	.2	1.4	0.9–2.3
Housewife	82.8%	87.9%	.05	1.5	1.0–2.3
Polygamy	20.1%	8.6%	.002	1.1	1.1–1.2
From Western Kenya	59.8%	39.6%	<.0001	2.3	1.7–3.0
Illiterate	6.9%	3.7%	.06	1.9	1.0–3.9
History of blood transfusion	4.2%	2.5%	.2	1.7	0.7–4.1
History of oral contraceptives	13.5%	13.0%	.9		

HIV = human immunodeficiency virus; CI = confidence interval.
Data are presented as mean ± SD or %.
* The groups were matched for age.

on the number of polymorphonuclears per high-power field.

Odds ratios, relative risks, and the 95% confidence intervals (CI) were used to measure the strength of associations, and the t test to compare sample means. The χ^2 test for trends was used to study trends over time. The matching information was ignored during the analysis. The Mantel-Haenszel χ^2 test was used for stratified analysis.

Results

We enrolled 406 HIV-1-infected women and 407 age- and parity-matched HIV-1-seronegative controls from the same deprived socioeconomic class. Fifty-two percent of the HIV-1-seropositive women were asymptomatic, 43.6% had HIV-related signs and symptoms, and 4.2% had AIDS. Persistent generalized adenopathy and chronic diarrhea were the strongest indicators of HIV-1 infection. A history of herpes zoster was uncommon, and reported mainly by HIV-1-infected women. The average gestational age at enrollment was 24.2 weeks (range 14–30).

Table 1 presents data on selected variables for HIV-1-positive and -negative women. There were no significant differences in marital status, history of blood transfusion, and prior oral contraceptive use. Significantly more HIV-1-infected women originated from the Luo tribe in Western Kenya (59.8 versus 39.6%; $P <$ .0001) and were part of a polygamous relationship (20.1 versus 8.6%; P = .002). Because both HIV-1 and polygamy are common in Western Kenya, a potential association of HIV-1 and polygamy was analyzed after stratification of women by ethnic origin. Seventeen percent of HIV-1-seropositive women of Luo origin and 22% of HIV-1-infected women from other ethnic groups were part of a polygamous marriage, as compared to 9.1 and 7.6%, respectively, of HIV-1-seronegative women; this indicates an independent relation between ethnic group, polygamy, and HIV-1. Human immunodeficiency virus type 1-seropositive women were slightly younger at sexual debut, reported more sex partners, and more often had a history of STDs (25.4 versus 17.0%; P = .003) than the seronegative controls. Malaria parasitemia was more frequent in HIV-1-positive women than in HIV-seronegative controls (23.8 versus 14.9%; P = .09), but the difference was not significant.

Three hundred fifteen HIV-1-seropositive and 311 seronegative women were followed to delivery. The remaining 23% were lost to follow-up and could not be traced despite intensive efforts. The loss to follow-up rate was high but similar in both groups. There were no significant differences in sociodemographic or medical characteristics between defaulters and non-defaulters. Nine mothers had a twin pregnancy (seven in the HIV-seropositive group and two among the controls) and were excluded from the analysis of gestational age, aspect, and birth weight. A history of at least one death of a previous child was present in 27.9% of HIV-1-infected women, as compared to 20.1% in the control group (odds ratio 1.5, 95% CI 1.1–1.6; P = .04). Eighteen percent of HIV-1-seropositive patients and 22% of HIV-negative controls delivered at home or at one of the maternity units in town. For these cases, birth weight was included only if the mother presented with the infant within 48 hours postpartum.

Table 2 lists the obstetric characteristics for both groups. The mean birth weight was significantly lower in the HIV-1-infected group than in the control group (2913 versus 3072 g; $P <$ .001), and significantly more HIV-1-seropositive women delivered at a gestational age of less than 37 weeks (24.7 versus 14.9%, odds ratio 1.9, 95% CI 1.2–2.9; P = .003). Twenty-one percent of the neonates born to HIV-1-infected moth-

Table 2. Obstetric Characteristics in Human Immunodeficiency Virus-1-Seropositive and -Seronegative Women

	HIV-1 (+)	HIV-1 (−)	P	Odds ratio	95% CI
Mean parity*	1.2 ± 1.1	1.3 ± 1.2	.4		
History of abortion	20/281 (7.8%)	10/275 (3.6%)	.1	2.0	0.9–4.8
Low birth weight	36/247 (14.6%)	33/235 (14.0%)	.9		
Stillbirth	19/274 (6.9%)	11/270 (4.1%)	.2	1.8	0.8–4.0
Infant mortality	76/272 (27.9%)	54/268 (20.1%)	.04	1.5	1.1–2.3
Cesarean rate	6/315 (1.9%)	12/311 (3.8%)	.2		
Mean birth weight (g)†	2913 ± 539	3072 ± 488	<.001		
Birth weight <2500 g†	46/285 (19.6%)	24/276 (8.7%)	.01	1.4	1.1–1.6
Mean gestational age	37.8 ± 3.0	38.3 ± 2.4	.02		
Gestational age <37 wk	76/315 (24.1%)	46/311 (14.8%)	.003	1.9	1.2–2.9
Mean length of labor (h)	10.1 ± 6.9	9.4 ± 6.1	.2		
Mean length of ruptured membranes (h)	4.2 ± 12.6	2.6 ± 8.1	.07		
No. of stillbirths	12/315 (3.8%)	6/311 (1.9%)	.2	2.0	0.7–6.1
No. of preterm births	65/308 (21.1%)	29/309 (9.4%)	<.001	2.6	1.6–4.2
No. small for dates	13/308 (4.2%)	10/309 (3.2%)	.7		
Neonatal mortality	8/240 (3.3%)	5/234 (2.1%)	.6		

Abbreviations as in Table 1.
Data are presented as mean ± SD or *N* (%).
* The groups were matched for parity.
† Twins and home deliveries not examined within 48 hours after birth were excluded from the birth weight analysis.

ers were preterm, compared to 9.4% in the control group (odds ratio 2.6, 95% CI 1.6–4.2; $P < .001$), whereas 4.2 and 3.2%, respectively, were SGA (not significant). The stillbirth rate and neonatal mortality rate tended to be higher in the HIV-1-seropositive group, mainly because of prematurity. Prematurity was more common in mothers with AIDS or AIDS-related signs and symptoms than in the asymptomatic HIV-1-infected group and in seronegative controls (28.6, 23.1, 19.3, and 9.9%, respectively; $P < .001$). In addition, mothers with an antenatal CD4 percentage of less than 30 (44 of 277, 15.9%) were more at risk for a preterm delivery (26.3 versus 10.1% prematurity rate, odds ratio 3.2, 95% CI 1.6–6.5; $P < .001$), and this was true in both HIV-1-seropositive women (27.1 versus 16.3%; $P = .2$) and seronegative subjects (14.3 versus 8.1%; $P = .4$). The association between maternal HIV-1 infection and prematurity was independent of other STDs including syphilis and gonorrhea (Table 3). No HIV-related congenital abnormalities were found.

Four HIV-1-infected mothers died: two in labor, one within 24 hours postpartum, and one at home 4 days after giving birth. The two deaths during labor were classified as resulting from eclamptic fits, although the blood pressure had been normal throughout pregnancy. The woman who died 1 day post-delivery had a postpartum hemorrhage.

On histopathologic examination of the placenta, we found a trend toward more chorioamnionitis in preterm than in term deliveries (36.0 versus 26.2%; $P = .2$) for both the HIV-1-seropositive and -negative groups. The rates of placental inflammation (chorioamnionitis, villitis, funisitis) were high, but similar in both groups (Table 4). There was no difference in placental inflammation between patients with low and high CD4 counts or CD4 percentage.

Two hundred fifty-three HIV-1-seropositive women and 265 controls attended the postpartum clinic. There was no difference in maternal infection rates with gonorrhea and chlamydia between the groups (Table 5). More women in the HIV-1-positive group had genital warts and genital ulcerations, and seven of 192 (3.6%) initially seronegative women seroconverted within 6 weeks postpartum. Pregnancy outcome in these patients was similar to the HIV-1-seronegative group. The incidence of syphilis during pregnancy or the immediate postpartum period tended to be higher in HIV-1-infected women than in seronegative controls

Table 3. Prematurity by Human Immunodeficiency Virus-1 Infection Stratified by Syphilis Serology and Gonococcal Infection

	HIV-1 (+)	HIV-1 (−)	P	Odds ratio*	95% CI
Syphilis antibodies (+)	5/20 (35.0%)	0/15			
Syphilis antibodies (−)	60/252 (23.8%)	29/260 (11.2%)	<.001	2.7	1.7–4.3
Gonorrhea postpartum +	6/18 (33.3%)	1/11 (9.1%)			
Gonorrhea postpartum −	29/129 (22.5%)	15/124 (12.1%)	.017	2.3	1.2–4.5

Abbreviations as in Table 1.
* Mantel-Haenszel odds ratio.

Table 4. Placental Characteristics in Human Immunodeficiency Virus-1-Seropositive and -Seronegative Women

	HIV-1 (+)	HIV-1 (−)	*P*
Chorioamnionitis	39/137 (28.5%)	42/140 (30.0%)	NS
Funisitis	16/139 (11.5%)	20/140 (14.3%)	NS
Villitis	4/139 (2.9%)	4/143 (2.8%)	NS

HIV = human immunodeficiency virus; NS = not significant.

(4.5 versus 1.3%; $P = .2$). However, only one out of nine women who seroconverted for syphilis delivered a preterm infant, and no stillbirths were noted.

Postpartum endometritis was more common in HIV-1-infected women than in seronegative controls (10.3 versus 4.2%, odds ratio 2.6, 95% CI 1.2–5.8; $P = .01$) (Table 5), and was associated with the isolation of *N gonorrhoeae* (28.6 versus 8.6%, odds ratio 4.2, 95% CI 2.2–6.9; $P = .01$). The isolation rate for gonorrhea in the postpartum period was similar in both groups (12.1 and 8.2%, respectively). Postpartum endometritis was inversely correlated with CD4 percentage (25.0, 14.3, 10.2, and 2.2% in CD4 categories of less than 20, 20–29, 30–39, and greater than 39%, respectively; $P = .0004$).

Geometric mean CD4 counts and mean CD4 percentages in patients with AIDS ($n = 9$), HIV-related symptoms (91), asymptomatic women (109), and HIV-seronegative controls (212) were 287, 421, 504, and 735 $\times 10^6$ cells/L and 19, 26, 27, and 40%, respectively. Sixty-six percent of HIV-1-seropositive women and 5% of seronegative controls had a CD4 percentage of less than 30%, and 48 and 7%, respectively, had a CD4 count of less than 400 $\times 10^6$ cells/L.

Discussion

Our data indicate that maternal HIV-1 serostatus in a population of African non-drug-using women has a serious impact on pregnancy outcome, particularly on prematurity, perinatal mortality, and maternal morbidity. Our findings are in agreement with those of Ryder et al,[1] who found that children born to HIV-seropositive women were on average 219 g lighter than children born to HIV-uninfected women in Kinshasa, in a population where 18% of the patients had AIDS. In our group, 4% of women had AIDS according to the WHO definition,[11] and 43% had HIV-related signs or symptoms. Sixty-six percent of HIV-seropositive women had a CD4 count lower than 30%, which increased by three times the risk of prematurity. These results differ from most studies from the developed world, which include mainly drug users, and from some African studies. Prospective cohort studies in Congo[13] and Rwanda,[14] with mostly asymptomatic women, found no differences in prematurity, low birth weight, and perinatal mortality rates. Possible explanations for these conflicting observations may include the stage of immunodeficiency, as our study and the Kinshasa study consisted of a relatively high proportion of women with clinical or immunologic signs of disease progression, compared to most other reports. Other factors may be variations in the virulence of HIV strains and differences in other causes of low birth weight, including concomitant infections, nutrition, or tobacco or alcohol use. In addition, the definition of prematurity is important: Most studies from developing countries use the cutoff level of a birth weight of 2500 g or less, whereas North American and European reports rely on gestational age. Our study is the first African report on HIV-1 and pregnancy outcome including ultrasonic assessment of gestational age, allowing accurate calculation of the gestational age and thus the prematurity rate. Over 90% of the ultrasound examinations in this study were done in the second trimester, when the accuracy of gestational age prediction is ±8 days; this accuracy deteriorates to ±22 days in the third trimester.[9] These data also confirm the results of a previous case-control study from Nairobi, which found an increased risk of prematurity in HIV-1-infected mothers.[3] However, the previously reported association between HIV-1 and fetal growth retardation could not be confirmed. This might be explained by the fact that in the case-control study carried out in

Table 5. Reproductive Tract Infections in Human Immunodeficiency Virus-1-Seropositive and -Seronegative Pregnant Women

	HIV-1 (+)	HIV-1 (−)	*P*	Odds ratio	% CI
Genital ulcer disease	19/406 (4.7%)	9/407 (2.2%)	.08	2.2	0.9–5.2
Genital warts	20/406 (4.9%)	8/407 (2.0%)	.03	2.6	1.1–6.4
Gonorrhea antepartum	26/381 (6.8%)	27/382 (7.1%)	1.0		
Gonorrhea postpartum	20/165 (12.1%)	12/146 (8.2%)	.3		
Chlamydia antepartum	36/314 (11.5%)	25/279 (9.0%)	.4		
Chlamydia postpartum	15/117 (12.8%)	16/103 (15.5%)	.7		
Postpartum endometritis	26/253 (10.3%)	11/265 (4.2%)	.01	2.6	1.2–5.8

Abbreviations as in Table 1.

1988, birth weight of less than 2500 g was used rather than gestational age, resulting in some degree of misclassification, whereas our study used obstetric ultrasound for a more reliable estimation of gestation.

A history of spontaneous abortion or infant mortality was higher in the HIV-infected group. These findings agree with data from Rwanda and Malawi, where childhood mortality and abortion were independently associated with HIV-1 seropositivity.[14,15] The neonatal mortality rate was slightly higher in the HIV-infected group, mainly because of the higher prematurity rate.

Most of the risk factors for HIV-1 infection in our population were related to sexual risk behavior, including age at sexual debut, polygamy, number of sex partners, and a history of STDs. Although most women reported multiple sex partners in their lifetime, the majority had had a stable relationship for the last 2 years. However, information on the sexual behavior of their partner was not available. Ethnic origin is important, as significantly more HIV-1-infected women belonged to the Luo tribe, which is the only ethnic group in Kenya that does not practice male circumcision. Lack of circumcision in men has been suggested as an important risk factor for HIV seroconversion.[16,17]

The incidence rates of STDs in our study group were high, particularly given the time frames of late pregnancy and postpartum, when most women report a decrease in sexual activity. This decrease might lead to a higher number of extramarital partners for the husband, increasing the risk for acquiring an STD. These findings underscore the WHO recommendations to repeat the syphilis screening test in the third trimester in populations with a high prevalence of syphilis.[18] The higher rate of postpartum endometritis in HIV-1-seropositive women despite similar prevalences of STDs in both groups can be explained by HIV-induced immunosuppression, resulting in a higher risk of ascending infections. This is illustrated by the dose-response association between postpartum endometritis and decreasing levels of CD4 percentage.

The maternal mortality rate of 16 per 1000 in the HIV-infected group was significantly higher than the 0.9 per 1000 rate in Pumwani Maternity Hospital. Two of the four women who died were asymptomatic and two had HIV-related symptoms. One of the asymptomatic women and one woman with persistent generalized lymphadenopathy and diarrhea died after an episode of "eclamptic fits," although their blood pressure had been normal throughout pregnancy. A postmortem was not performed; hence these data are inconclusive as to the cause of death in these women.

Biologic mechanisms for HIV as a cause of adverse pregnancy outcome are not clear. The mechanisms regulating the onset of labor have been the subject of intensive investigations over the past decade. Fetal plasma cortisol levels, maternal progesterone and estrogen concentrations, and prostaglandins and their metabolites are all involved in human parturition. Most of the major pregnancy-associated factors, such as placental-ovarian steroids and protein hormones, that have been implicated as immunomodulators are produced by the trophoblast. Trophoblast cells are also phagocytic and capable of engulfing pathogenic microorganisms, a property expressed by placental macrophages as well.[19] The HIV-1 antigen has been identified in maternal leukocytes and trophoblast derivates.[20,21] Theoretically, fetal or placental infection could interfere with the production of steroids and hormones and hence play a triggering role in the onset of labor, but so far no evidence has been provided for such an interaction.

The disease stage of the mother could play a role in HIV-related poor pregnancy outcome. In our study, AIDS-related signs and symptoms and a low antenatal CD4 percentage were risk factors for preterm birth and postpartum endometritis. A possible explanation might be the higher susceptibility to infections in immune-suppressed individuals or the overall impaired health condition in patients with HIV disease.

In contrast, no association was found between HIV-1 infection and placental inflammation, as suggested previously. One hypothesis of a possible biologic mechanism to explain HIV-related prematurity was the possible cumulative immunosuppressive effects of HIV infection and pregnancy, facilitating ascending infections and thus causing chorioamnionitis leading to prematurity or fetal death. Histologic chorioamnionitis by itself appears to be an independent risk factor for prematurity.[22] Our data demonstrated a trend toward more chorioamnionitis in preterm births from HIV-1-seropositive women than from seronegative controls. However, we did not show any significant differences in the rates of chorioamnionitis, villitis, or funisitis between HIV-1-seropositive and -seronegative women. Moreover, maternal immunosuppression did not seem to increase the degree of placental inflammatory changes. Thus, our findings do not support the role of chorioamnionitis in HIV-related prematurity.

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