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Growth Patterns in the First Year of Life Differ in Infants Born to Perinatally vs. Non-Perinatally HIV-infected Women

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Abstract

Objective—To compare growth patterns in the first year of life between children born to perinatally HIV-infected (PHIV) vs. non-perinatally HIV-infected (NPHIV) women in the U.S.

Design—Retrospective cohort study of HIV-infected pregnant women who received care and delivered a liveborn at two urban tertiary centers from January 2004 - March 2012.

Methods—We collected data via chart review on demographics, behavioral risk factors, HIV clinical markers, combination antiretroviral therapy (cART), mode of HIV acquisition, pregnancy outcomes, and infant anthropometrics on study subjects. Mixed effects models were used to assess the association between maternal mode of HIV acquisition and weight-for-age (WAZ), length-for-age (LAZ), and weight-for-length (WLZ) *z*-scores.

Results—Of 152 pregnancies evaluated, 32 and 120 infants were born to 25 PHIV and 99 NPHIV women respectively. Infants of PHIV women exhibited lower mean WAZ and LAZ throughout the first year of life in unadjusted analyses. After adjusting for potential confounders, the relationship between PHIV & LAZ persisted (β=−0.54, p=0.026). Small for gestational age for each birth anthropometric parameter [birth length, birth weight, and both birth length and weight] was associated with decreased LAZ (β=−0.48, p=0.007), WAZ (β=−0.99, p<0.001) and WLZ (β=

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−0.36, p=0.027) respectively. A delivery HIV RNA level <400 copies/mL was associated with increased WAZ & WLZ (β=0.43, p=0.015; β=0.38, p=0.021).

Conclusions—Infants of PHIV women may remain at persistently decreased lengths throughout the first year of life. Further studies aimed at understanding intrauterine and environmental factors in PHIV women are warranted.

Introduction

With the success of combination antiretroviral therapy (cART), an increasing number of women infected with HIV since birth are reaching their reproductive years. [1-3] As these long-term survivors are achieving pregnancies, maternal immunological alterations associated with chronic HIV infection and inflammation, [4, 5] as well as maternal behavioral, [6, 7] neurocognitive, [8] and metabolic complications [9] from long-term HIV and antiretroviral (ARV) exposure present a growing concern for a potential impact on the developing fetus/infant. To date, limited information exists on neonatal outcomes of infants born to perinatally HIV-infected (PHIV) pregnant women [10-15]. In contrast to non-perinatally HIV-infected (NPHIV) pregnant women, PHIV women have been observed to have poorer immunologic status throughout pregnancy,[16-18] lower rates of viral suppression at delivery,[13, 14, 17] and higher rates of low birth weight (LBW) (<2500 g) [13] and small-for-gestational age (SGA)[10] outcomes in their infants. Given the recognized association of compromised growth during infancy with adverse long-term health outcomes [19-21] our analysis focuses on a comparison of growth between offspring of PHIV and NPHIV women during the first year of life.

Methods

Study Population

Mount Sinai Hospital (MSH) and Johns Hopkins University (JHU) provide comprehensive care to HIV-infected pregnant women and their offspring in an urban tertiary care environment. This analysis includes all HIV-infected pregnant women/children who received obstetrical care and pediatric follow-up at these institutions from January 2004-March 2012. Pregnancies with multiple gestations [22] or ending in spontaneous/therapeutic abortions or intra-uterine fetal demise (IUFD) were excluded from primary analysis. In addition, infants <28 weeks gestational age (GA), with documented HIV infection, deceased before 1 year, or not followed at the clinics above so as to result in > 3 anthropometric measurements each separated by > 6 weeks in the first year of life were also excluded. The majority of data was obtained at birth, 1-2 weeks, 4 weeks, and 2, 4, 6, 9 and 12 months according to well child visit schedules. The study was approved by the Institutional Review Boards of MSH and JHU.

Primary Outcome

We evaluated growth in the first year of life using routinely collected weight and length measurements. Weight-for-age (WAZ), length-for-age (LAZ), and weight-for-length (WLZ) z-scores were calculated based on World Health Organization (WHO) child growth standards.[23]

Predictor Measurements

Our main exposure of interest was the documented mode of maternal HIV acquisition; women were categorized as PHIV or non-PHIV (NPHIV) according to medical records or patient report. Other potential confounders for infant growth including maternal age, pre-pregnancy body mass index (BMI), race [24], and substance use during pregnancy were determined by medical record review. In addition we controlled for all parameters of infant SGA [SGA by birth weight (BW), SGA by birth length (BL) and SGA by BW and BL for each of the mixed models (WAZ, LAZ and WFLZ respectively) as these are known to be associated with subsequent growth.[25] Substance use was defined as any tobacco, alcohol, or illicit drug use. We collected CD4 cell counts including pregnancy nadirs, HIV RNA levels (using <400 copies/mL to define viral suppression,) and cART recorded throughout pregnancy and at delivery. Second-line cART regimens were defined as containing enfuvirtide, etravirine, darunavir, or raltegravir.

Statistical Analysis

Characteristics of PHIV and NPHIV women at first pregnancy were compared using Wilcoxon test, Chi-square, or Fisher's exact test as appropriate. Loess plots [26] were fit to compare infant WAZ, LAZ, and WLZ over time between groups. Mixed effects models were used to assess the association between maternal mode of HIV acquisition and each of the three primary growth outcomes. Models included random effects for intercept, slope, and the mother (to account for repeat pregnancies) with an unstructured covariance matrix. Additional analyses were conducted to compare baseline characteristics of infants with incomplete growth data due to lack of clinical follow-up and those with complete data. We did not impute missing data because using our exclusion criteria for those lost to follow-up, <5% had missing (at random) data. Statistical analyses were performed using SAS® 9.3 (SAS Institute, Cary, NC.)

Results

Between January 2004 and March 2012, 170 pregnant women (38 PHIV, 132 NPHIV) received care at the institutions above, resulting in 223 pregnancies. After excluding 18 spontaneous/induced abortions, two pregnancies with IUFD, three twin gestations, five extreme premature, two HIV-infected and three deceased infants, and 38 infants not followed at MSH or JHU, 152 pregnancies remained for analysis: 32 and 120 infants born to 25 PHIV and 99 NPHIV women respectively. Baseline characteristics of women at first pregnancy and of all infants are shown. (Table 1) PHIV women were younger ($p<0.001$), had lower gravidity ($p<0.001$), had lower CD4 nadirs during pregnancy ($p=0.004$), were less likely to demonstrate viral suppression at delivery ($p=0.036$), and more likely to be on second-line cART ($p=0.005$) than the NPHIV women. Similar rates of PI-based cART were seen in PHIV and NPHIV women (92 vs. 91%). Substance use patterns differed between groups as no PHIV women reported heroin, cocaine or alcohol use during pregnancy, but these differences did not reach statistical significance. Two PHIV and five NPHIV women were HIV/Hepatitis C co-infected. Three NPHIV women were positive for hepatitis B surface antigen. Infants born to PHIV women were less likely to be female ($p=0.03$) and had lower mean BWs ($p=0.04$) as well as birth WAZ ($p=0.032$). Median birth GA, rates of

prematurity and SGA BW [27] were similar between groups. Comparison of the study sample infants to those who were not followed by MSH or JHU (n=38) did not reveal any differences in maternal characteristics (reported substance abuse, baseline CD4, and delivery HIV RNA level) or infant outcomes [GA, BW, and birth length (BL)].

Overall, by 12 months, 9.2% (14/152) of infants remained underweight (WAZ < −2), and 13.2% (20/152) remained short statured (LAZ < −2). Infants of PHIV women exhibited lower mean WAZ and LAZ throughout the first year of life in unadjusted analyses. (Figure 1) After adjusting for maternal age, pre-pregnancy BMI, nadir CD4 cell count during pregnancy, HIV RNA level at delivery, use of second-line cART, and infant SGA at birth, the relationship between PHIV & LAZ persisted (β=−0.54, p=0.026). SGA for each birth anthropometric parameter [BL, BW, and BW and BL] was associated with decreased LAZ (β=−0.48, p=0.007), WAZ (β=−0.99, p<0.001) and WLZ (β=−0.36, p=0.027) respectively. A delivery HIV RNA level <400 copies/mL was associated with increased WAZ & WLZ (β=0.43, p=0.015; β=0.38, p=0.021). We did evaluate tenofovir use instead of second-line cART use in multivariate modeling, but found no effect of tenofovir on our outcomes; nor did the effect of PHIV on LAZ change (data not shown.)

Discussion

In this study of HIV-infected pregnant women, we found that HIV-exposed uninfected (HEU) infants born to PHIV women exhibited decreased WAZ and LAZ throughout the first year of life compared to HEU infants born to NPHIV women in unadjusted analysis. HEU infants born to PHIV women were found to be at risk for decreased length in the first year, while maternal viral suppression was protective for growth (weight and length), even after adjusting for confounders.

We previously described the association between maternal PHIV and SGA birth outcomes [10] and expand upon this analysis to assess infant growth in this larger study population. To our knowledge, no studies to date have evaluated growth in HEU infants born to PHIV vs. NPHIV women, though several have evaluated differences in maternal factors [14, 17, 18, 28, 29] and pregnancy outcomes [12-17, 30, 31]. Evidence shows that PHIV pregnant women are more immunosuppressed than their NPHIV counterparts. Several studies reported similarly low CD4 cell counts both before (mean initial CD4 cell count 144-314 cells/mm^3) [13, 17, 18] and during the pregnancy (136-394 cells/mm^3) [14, 15, 17, 18, 30, 32] in PHIV women as seen in our cohort. Our finding of decreased rates of maternal virologic suppression at delivery in PHIV women is also consistent with other reports. [13-15, 17, 18] Not unlike our results, a number of studies have reported increased rates of LBW [13, 15] and similar median BWs (2667-2688g) of children born to PHIV women. [14, 17] The overall rate of preterm birth in our cohort (22.4%) was comparable to rates found in the U.S. among HIV-infected pregnant women (18-22%) [33, 34] as well as reported rates in PHIV women (13.3 – 36%) [12-14, 16, 17].

While infant growth is subject to much confounding, we did find that being born SGA is a predictor for decreased growth in the first year of life, consistent with current literature. [35-37] The protective effect of maternal viral suppression at delivery on infant WAZ and

WLZ throughout the first year of life may simply represent the fact that women who adhere to their cART during pregnancy are more likely to also attend to their own health as well as the health care of their infants. It is not entirely clear why infants born to PHIV women had decreased LAZ compared to those born to NPHIV throughout the first year of life, even after controlling for known risk factors. Maternal antenatal factors such as anthropometrics, CD4 nadir during pregnancy, and cART were not associated with poor postnatal growth, suggesting perhaps that other elements of the prenatal environment in PHIV women may affect intrauterine and early postnatal growth. The protracted state of impaired immunologic function in PHIV women may alter the prenatal environment of their offspring such that fetal programming in this unique *in utero* milieu affects metabolic pathways and postnatal growth and development. [38] Alternatively, external factors such as differences in early postpartum maternal socioeconomic status and nutritional security may be the reason for our findings. We were unable to thoroughly assess these given the retrospective nature of our study. However, the PHIV and NPHIV women were from the same clinical catchment areas where similar racial and socioeconomic backgrounds exist.

The long term implications for poor early postnatal gains in length, particularly in HEU infants born to PHIV remain unclear. However, studies in HIV-unexposed infants have demonstrated that poor early postnatal growth is associated with short adult stature, [19] diminished cognitive abilities, [20, 21, 39] and a host of diminished socioeconomic gains including lower educational attainment, employment, and wages. [39-41] Intrauterine growth and that attained by 12 months has been shown to be the critical period during which interventions can be made to avoid long term complications.[19, 39, 41]

In addition to our inability to properly assess maternal socioeconomic status mentioned above, our study is also limited by the small sample size. Since we determined the mode of HIV acquisition in some via patient report, there is the possibility of misclassification bias. Our study lacked power to assess effects of specific ARVs administered *in utero* on postnatal infant growth. Lastly, we were not able to assess diet or feeding habits in the first months of life.

In conclusion, because infants of PHIV women may remain at persistently decreased lengths in the first year of life, careful monitoring of these infants is essential. Further studies aimed at understanding both intrauterine and external environmental factors in PHIV women are warranted.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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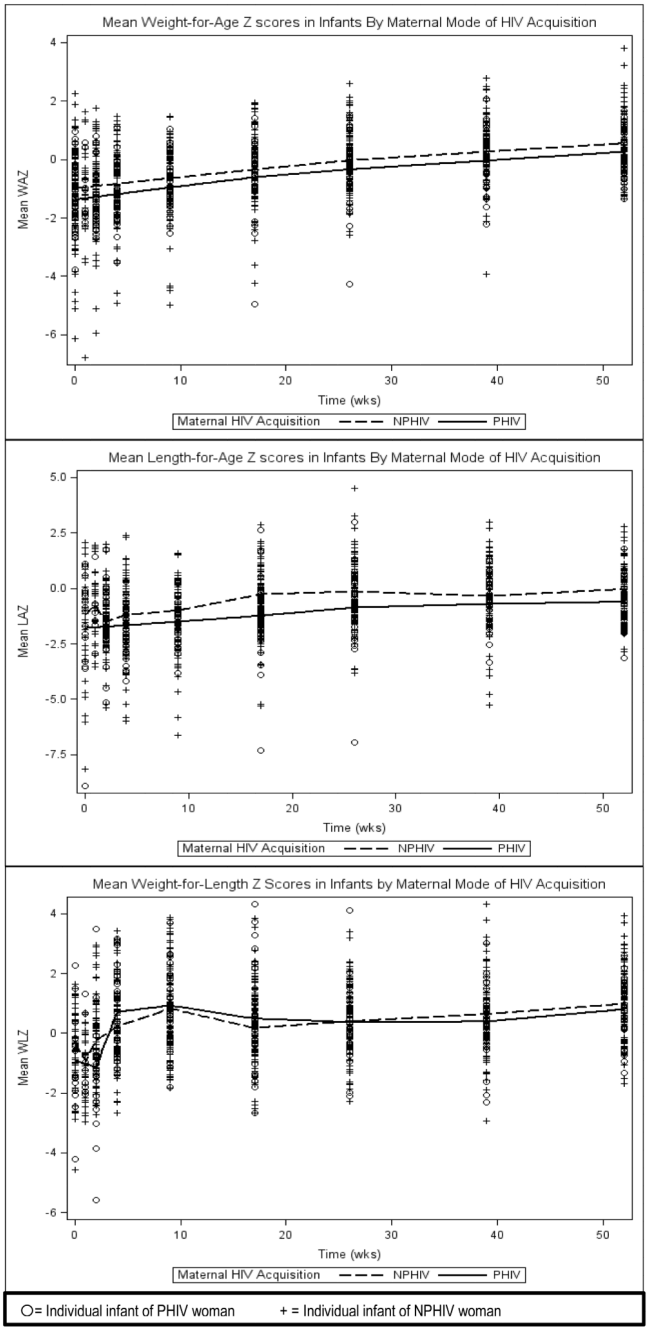

Figure 1. Loess Plots of Mean Weight for Age (WAZ), Length for Age (LAZ), and Weight for Length (WLZ) *Z*-scores by Maternal Mode of HIV Acquisition

Table 1

Linear Mixed Effects Model of Maternal/Infant Factors Associated with *Z*-scores of Weight, Length, and Weight-for-Length in HIV-Exposed Uninfected Infants

	Weight (WAZ)		Length (LAZ)		Weight-for-Length (WFLZ	
	Coefficient	***p* value**	**Coefficient**	***p* value**	**Coefficient**	***p* value**
Maternal HIV Acquisition						
PHIV	−0.29	*0.137*	−0.54	*0.026*	0.09	*0.597*
NPHIV	0		0		0	
Maternal age, per year	−0.02	*0.090*	−0.01	*0.611*	−0.01	*0.391*
Pre-pregnancy BMI of mother	0.00	*0.913*	0.00	*0.678*	0.00	*0.622*
Nadir CD4 cells in pregnancy						
≤ 200 cells/mm^3	0.16	*0.42*	0.44	*0.085*	−0.29	*0.114*
> 200 cells/mm^3	0		0		0	
HIV RNA level at delivery						
< 400 copies/mL	0.43	*0.015*	0.17	*0.443*	0.38	*0.021*
≥ 400 copies/mL	0		0		0	
Second line cART use in pregnancy						
Not on second line cART	0.03	*0.912*	0.24	*0.483*	−0.31	*0.216*
On second line cART	0		0		0	
Infant SGA for BW			---	---	----	----
SGA for BW	−0.99	*<0.001*				
Not SGA for BW	0					
Infant SGA for BL	---	---			----	----
SGA for BL			−0.48	*0.007*		
Not SGA for BL			0			
Infant SGA for BW and BL	---	---	---	---		
SGA for BW and BL					−0.36	*0.027*
Not SGA for BW and BL					0	

WAZ=Weight for Age Z score, LAZ=Length for Age Z score, WFLZ=Weight for Length Z score, PHIV=Perinatally HIV-infected, NPHIV=Non-Perinatally HIV-infected, BMI=Body Mass Index, cART=Combination Antiretroviral Therapy, SGA=Small for Gestational Age, BW=Birth Weight, BL=Birth Length