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Food Insecurity, CD4 Counts, and Incomplete Viral Suppression Among HIV+ Patients from Texas Children's Hospital: A Pilot Study

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Abstract

Determine the relationship between food insecurity and CD4 counts and viral suppression among pediatric HIV-positive patients. Food insecurity was assessed by validated survey. CD4 counts and viral load were abstracted from patients' charts. We used linear regression for the dependent variable of the natural log of CD4 counts and logistic regression for viral suppression, with backward deletion of covariates with p > 0.1. Food insecurity ($\beta = -0.23, 95$ % CI [-0.40, -0.01])

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was associated with lower CD4 counts and higher odds of incomplete viral suppression (OR = 4.07, 95 % CI [1.02, 13.92]). Food insecurity may adversely impact pediatric HIV outcomes.

Keywords

Pediatric; HIV; Food security; CD4 count; Viral load

Introduction

HIV infection is an important US public health problem [1]. Identifying risk factors for poor prognosis and optimizing HIV prevention and treatment are public health goals. Food insecurity is an understudied yet important likely cause and consequence of HIV infection. The US Department of Agriculture defines food insecurity as "limited or uncertain availability of nutritionally adequate and safe foods or limited or uncertain ability to acquire acceptable foods in socially acceptable ways" [2]. In the US, the prevalence of household food insecurity remains at record high levels (14.5 %) [2]. Food insecurity adversely impacts a wide range of child health outcomes including obesity, child development, undernutrition/ nutrient deficiencies, and overall health status [3]. Food insecurity was associated with lower CD4 counts and incomplete viral suppression among HIV-positive adults in the US and Canada [4-7]. For example, food insecure HIV + adults in Atlanta had lower CD4 counts and higher viral loads compared to their food secure peers [4]. Food insecurity was longitudinally associated with lower CD4 counts among HIV + adults in the Boston and Providence areas [7]. Food insecurity likely impacts clinical prognosis and indicators of HIV infection, including CD4 count and viral load, through macronutrient and micronutrient deficiencies that lead to weight loss, malabsorption, diarrhea, malnutrition, and suboptimal immune response and absorption of antiretroviral medications [8]. Previous studies have not determined the relationship between food insecurity and HIV outcomes among pediatric patients and young adults [8]. Since in 2010, 25.5 % of households with children in Harris County, Texas, which includes the City of Houston, were food insecure [9], we have the opportunity to fill this important research gap by examining the relationship among food insecurity, CD4 count, and viral suppression among HIV-positive patients receiving care at Texas Children's Hospital (TCH) in Houston. We hypothesized that food insecurity was inversely associated with (1) CD4 count and (2) viral suppression.

Methods

The design of this pilot study was cross-sectional. Participants were recruited during routine clinic visits from the TCH HIV specialty clinic, which serves the Houston/Harris County area. For the present study, recruitment duration was December 2010–August 2011 and occurred via study flyers and research coordinators. Inclusion criteria included age of 18 months or older, diagnosed with perinatally acquired HIV infection, able to complete forms in English, and current patients of the clinic. Patients noted during their clinic visit to have an acute illness in the past 2 weeks were excluded from enrolling at that particular clinic visit, but could be enrolled if eligible at future visits. This exclusion eliminated the potential impact of acute illness on the measurement of food insecurity and outcome variables. There was no upper age limit for this study, in order to reflect the clinic population served, although the patients seen in the clinic were less than 25 years of age. A total of 90 patients were deemed eligible for this study from the TCH HIV clinic, based on birth records and laboratory confirmation of HIV-infection on the basis of two positive HIV DNA polymerase chain reaction (PCR) assays. The institutional review board of Baylor College of Medicine and Affiliated Institutions approved this study.

Sample size was calculated, a priori, based on the maximum expected 90 participants, which would provide 80 % power to detect 75 log-transformed cells/mm³ difference between food insecure and secure groups (a moderate effect size, f = 0.30), assuming $\alpha = 0.05$ (two tailed), one-way ANOVA, and a 0.95 pooled standard deviation of log-transformed CD4 + count (cells/mm³) [10].

Participants or their proxies (usually a parent) completed a demographic questionnaire to obtain child's age, gender, race/ethnicity, and their parents' education. Current health insurance status, as a proxy for socioeconomic status, was obtained from each patient's electronic medical record and coded as none, Medicaid/Medicare, or private. These demographic characteristics have been associated with food insecurity and/or HIV infection or HIV risk-related behaviors [1, 3, 11], and thus were considered important covariates to reduce potential confounding. Current antiretroviral (ARV) therapy was obtained from the medical record and coded as (1) yes (reference category), (2) no-slow progressor, i.e. a clinical history of slow disease progression, or (3) no-non-adherence, i.e. non-adherent to the ARV regimen. Household food insecurity over the past 12-months was assessed using the US Department of Agriculture's 18-item Household Food Security Survey Module (HFSSM) [2]. The HFSSM is one of the most widely used food security scales and provides national US estimates in the Current Population Survey of the US Census Bureau and the National Health and Nutrition Examination Survey of the Centers for Disease Control and Prevention. Validation of the scale has been previously reviewed [12]. Sample questions included: (1) "In the last 12 months, did you ever eat less than you felt you should because there wasn't enough money for food?" and (2) "In the last 12 months, did you ever not eat for a whole day because there wasn't enough money for food?". Using a standard protocol, research staff obtained duplicate measures of participants' height using a portable stadiometer (Seca model 214, Birmingham, UK) and weight using a digital scale (Tanita model BWB-800S, Arlington Heights, IL). Participants' BMI (kg/m²) was calculated, and for participants <20 years of age, BMI z-scores were determined from Centers for Disease Control and Prevention growth charts. Because growth charts do not include BMI z-scores for subjects 20 years of age (n = 7 in this sample), for the purposes of calculating BMI zscore for these older subjects, we applied the 19 year old BMI z-scores to subjects 20 years of age. BMI z-score was included as a covariate, since CD4 counts and food security have been associated with adiposity [3, 8]. CD4 counts were determined using flow cytometry and HIV RNA viral suppression was determined by quantitative RNA PCR assay by the TCH clinical virology laboratory on samples from the day of the clinic visit, or, if unavailable, then up to 3-months later. The average number of days from the day of the clinic visit to collection of CD4 counts or viral load was 9.2 and 12.1 days, respectively.

Due to a number of participants with food insecurity scores of zero, the raw scores were dichotomized to food secure (reference category) or insecure categories based on previous recommendations [13]. HIV RNA suppression was defined as participants with undetectable viral loads (<144 copies/mL), i.e. this variable was dichotomized to completely suppressed (reference category) or incompletely suppressed (144 copies/mL). CD4 counts were log-transformed due to a skewed distribution. Race/ethnicity was dichotomized into African–American and non-African–American groups, since 65 % identified as African–American and the other groups were small.

Participant characteristics, mean \pm (standard deviation) or number (percentage), for the total sample and stratified by food security status were calculated. Differences in characteristics by food security status were examined using Student's *t* test for continuous variables and Pearson's Chi squared or Fisher's exact tests for categorical variables. To test Hypothesis 1, linear regression was used. The main dependent variable was the natural log of CD4 counts and the main independent variable was food insecurity status. Covariates included age,

gender, race/ethnicity, BMI z-score, health insurance status, ARV therapy status, and parent education. To test Hypothesis 2, logistic regression was used. The main dependent variable was HIV RNA suppression. The main independent variable, food insecurity, and covariates were the same as for the linear regression model. For both the linear and logistic regression models, stepwise procedures with backward elimination of covariates with p > 0.1 were used to identify significant predictors. Participants with missing variables were dropped from analyses on a case-by-case basis. The back-transformed β coefficients are presented for the linear regression model. Analyses were conducted using SAS 9.2 (Cary, North Carolina). A significance level of p < 0.05 was chosen for the final models.

Results

Of the 90 patients eligible for participation from the clinic, 62 were enrolled in the study (68.9 %). Of the 28 not enrolled, the most common reason was due to missing their scheduled appointment or lack of research coordinator coverage. For the sample (Table 1), n = 62, average age was 14.0 ± 5.2 years, 56.5 % female, 66.1 % African–American, 16.1 % with no insurance, 66.1 % with Medicaid/Medicare, 17.7 % with private insurance, and 50.8 % of parents had a high school education or lower. Participants did not differ on these characteristics when stratified by food security status (Table 1, all p > 0.05). The prevalence of food insecurity was 37.1 %. Mean CD4 count was 808.6 ± 488.5 cells/mm³ and 56.7 % had complete viral suppression. 90.3 % were currently on ARV therapy with two patients (3.2 %) off ARVs due to non-adherence and four patients (6.5 %) off ARVs due to slow disease progression.

For Hypothesis 1, compared to those who were from food secure households, those from food insecure households had lower CD4 counts ($\beta = -0.23$, 95 % CI [-0.40, -0.01], p = 0.0453, n = 9). In other words, participants from food secure households had mean adjusted CD4 counts of 788 ± 63 which was significantly different compared to their food insecure peers who had mean adjusted CD4 counts of 614 ± 68. Both race/ethnicity ($\beta = -0.26$, 95 % CI [-0.43, -0.04], p = 0.0263) and child age ($\beta = -0.05$, 95 % CI [-0.08, -0.03], p = 0.002) were significant and retained in the linear regression model such that African–Americans had lower CD4 counts than their peers and child age was inversely associated with CD4 counts. The linear regression model for Hypothesis 1 accounted for 42 % of the variability in CD4 counts. For Hypothesis 2, compared to those who were from food secure households, those from food insecure households had higher odds of incomplete viral suppression (OR = 4.07, 95 % CI [1.19, 13.92], p = 0.025, n = 59). Gender was retained in the logistic regression model such that males had higher odds of incomplete viral suppression (OR = 3.65, 95 % CI [1.09, 12.30], p = 0.037). The logistic regression model for Hypothesis 2 accounted for 17 % of the variability in viral suppression.

Discussion

This study documents the high prevalence of household food insecurity, the inverse association between food insecurity and CD4 counts, and the positive association between food insecurity and incomplete viral suppression among a mainly pediatric sample of HIV-positive patients. The prevalence of household food insecurity (37.1 %) for this sample was more than 2½ times the prevalence in the US nationally but lower than the prevalence reported among selected samples of adult HIV-positive individuals in the US and Canada by previous groups: 48 % in British Columbia [6], 49 % in San Francisco [5], 52 % in Atlanta [4], and 63 % in Boston/Providence [7]. Locally, this sample's prevalence of food insecurity is almost 1½ times the prevalence for households with children in Houston/Harris County [9]. Thus, households with HIV + children and young adults are at increased risk for

food insecurity compared to local and national estimates and merit interventions to address food insecurity and its complications.

Food insecure participants in the present study had 174 fewer CD4 cells/mL than their food secure peers. This difference is similar to the difference in CD4 counts (100 CD4 cells/mL) between food secure and insecure adults over 5 years [7]. These results imply that addressing food insecurity may result in clinically relevant improvements to CD4 counts among HIV + patients, which will require confirmation with longitudinal cohort studies and randomized controlled trials. These results also highlight the importance of adequate dietary intake on HIV outcomes. Moreover, food insecurity was significantly associated with incomplete viral suppression, as reported elsewhere among adults [6, 8]. In the present study, there was a greater than fourfold higher odds of incomplete viral suppression among patients from food insecure versus food secure households. Food insecurity is thought to influence HIV disease progression through several mechanisms. First, food insecurity has been postulated to lead to undernutrition and micronutrient deficiencies which impairs immune response and predicts disease progression [8], even among patients taking highly active ARV therapy [7]. Second, food insecurity may impair absorption of ARV medications and influence medication adherence [8], which contributes to treatment failure and disease progression. HIV disease progress in turn contributes to worsened food insecurity by redirecting income, assets, and time towards HIV care, and thus a vicious cycle results [14].

Strengths of this study include the pediatric and young adult sample, a widely used and well validated measure of food insecurity, and clinically relevant outcomes. Limitations include generalizability, the cross-sectional design, small sample size, the lack of biomarkers for insufficient dietary intake and undernutrition, and the lack of both an indicator of ARV therapy adherence and length of time on ARV therapy. Clearly, further studies that address these limitations are necessary to confirm the relationship between food insecurity and HIV clinical outcomes and to identify the mechanisms for these relationships.

Conclusions

Households of pediatric HIV + individuals experience significant food insecurity. This experience may compromise their CD4 counts and likelihood of HIV viral suppression. Routine measurement of food insecurity is needed in clinics caring for HIV + pediatric patients. Furthermore, policies and programs directed at alleviating pediatric food insecurity may improve clinical HIV outcomes.

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Abbreviations

ANOVA	Analysis of variance
ARV	Antiretroviral

CD	Cluster of differentiation
HIV	Human immunodeficiency virus
PCR	Polymerase chain reaction
ТСН	Texas Children's Hospital
US(A)	United States (of America)

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Table 1

Descriptive statistics for the study sample (n = 62) and main results from the multivariate regression models

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4 (40.0)	$6\ (60.0)$	10 (16.1)
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15 (36.6)	26 (63.4)	41 (66.1)
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	Food insecure	Food secure	Total
Food secure	Reference	Reference	Reference
Food insecure	-0.23	[-0.40, -0.01]	0.0453
Logistic regression for incomplete viral suppression d	Odds Ratio	[95 % CI]	d
Food secure	Reference	Reference	Reference
Food insecure	4.07	[1.02, 13.92]	0.025

> using Student's t test for continuous variables and Pearson's Chi squared or Š, 2 2 5, Fisher's exact tests for categorical variables

 a Data presented as mean (standard deviation)

 $\overset{h}{D}$ Data presented as n (row % for food insecure versus secure, or column % for Total)

cControlling for age and race/ethnicity

d Controlling for gender