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The interaction between detectable plasma viral load and increased body mass index on hypertension among persons living with HIV

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Abstract

Increased body mass index (BMI) and HIV are each associated with hypertension. This study tested interactions between BMI and detectable plasma viral load (pVL) on hypertension among 659 persons living with HIV (PLWH). All participants were categorized into four subgroups based on BMI (<25 and ≥25 kg/m^2) and pVL (<200 and ≥200 copies/ml). Multiplicative interaction was assessed using logistic regression; addictive interaction was assessed using three measures: Relative Excess Risk due to Interaction (RERI), Attributable Proportion (AP), and Synergy index (S). Compared to the participants with normal BMI and undetectable pVL, those who had increased BMI with an undetectable pVL had an elevated risk of hypertension with OR [95%CI] = 1.80 [1.02, 3.20]; the risk was further increased for those who had increased BMI with detectable pVL with OR [95%CI] = 3.54 [1.71, 7.31]. The multiplicative interaction was significant (p = 0.01). Results from additive interaction indicated RERI [95%CI] =1.89 [0.76, 4.79] and AP [95%CI] = 0.64 [0.32, 0.95]. The interaction effects of increased BMI and detectable pVL on hypertension on both multiplicative and additive scales suggested that PLWH with increased BMI and detectable pVL should be intensively managed and monitored for hypertension prevention and treatment.

Keywords

HIV; hypertension; BMI; detectable plasma viral load; interaction

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Disclosure statement

No potential conflict of interest was reported by the authors.

Introduction

Increased access to antiretroviral therapy (ART) has sub-stantially changed the HIV infection from a fatal diagnosis to a chronic condition. Consequently, age-related chronic diseases, such as hypertension, have become significant, with a prolonged life expectancy of persons living with HIV (PLWH).

Hypertension among PLWH is associated with multiple risk factors, including traditional cardiovascular risk factors, HIV infection and side effects of some ART agents. Increased body mass index (BMI) is a modifiable risk for hypertension. PLWH are at higher risk of being overweight or obese than the general population, and the risk further increases after the initiation of ART (Ezechi, Musa, Otobo, Idigbe, & Ezechi, 2016; Thompson-Paul et al., 2015). Reported studies also suggest that increased BMI interferes with ART, leading to poor adherence, virological failure, and other negative health outcomes (Crum-Cianflone et al., 2010).

HIV infection itself is related to important pathophysiological processes of hypertension, such as immune activation and chronic low-grade inflammation (Bourgi, Wanjalla, & Koethe, 2018). The pro-inflammatory effects of HIV infection can induce endothelial damage, vasoconstriction, reduced endothelium-dependent relaxation, and vascular stiffness (Bourgi et al., 2018; Calo, Caielli, Maiolino, & Rossi, 2013; Dube et al., 2008; Maia-Leite et al., 2016; Stein & Hsue, 2012; Xu, Chen, & Wang, 2017). The same mechanisms of inflammation and immune activation for HIV infection can also explain the BMI-hypertension relationship (De Marco, Aroor, & Sowers, 2014; Okello et al., 2015). These two lines of research findings strongly suggest that increased BMI and detectable HIV plasma viral load (pVL) may interact with each other, increasing the risk of hypertension among PLWH. The primary purpose of this study is to test this interactive effect.

Methods

Study setting and participants

We analyzed data of 659 participants who were enrolled in the Florida Cohort Study (http://sharc-research.org/) (Cook et al., 2017; Sharpe et al., 2017). Participants with complete data on hypertension diagnosis, height, and weight were included. Compared with participants included, those excluded were older, less likely to be current smokers, and more likely to be Hispanics, depressive, with longer durations of HIV diagnosis and higher CD4+ T-cell counts ($p < 0.05$ for all). Data about demographics and behavioral factors were collected using a survey questionnaire. The survey data were linked to Enhanced HIV/AIDS Reporting System (eHARS) to obtain data on HIV pVL, CD4+ T-cell counts, and nadir CD4+ T-cell counts, and linked to medical records to extract data regarding height, weight, and diagnosis of hypertension, depression, renal disease, and diabetes. This study was approved by the institutional review boards (IRB) at the University of Florida, Florida International University, and the Florida Department of Health.

Measurements

Diagnosis of hypertension based on the International Classification of Diseases-10 (ICD-10) and/or −9 (ICD-9) was directly extracted from the linked medical records. BMI was calculated with data on height and weight also extracted from the linked medical using the standard method, dividing the weight (kg) by squared height (m).

Data for the most recent measures of pVL and CD4+ T-cell count and nadir CD4+ T-cell count were derived from eHARS. Depression, renal disease, and diabetes were defined based upon ICD-10 and/or ICD-9 from the medical records. Other covariates were collected through questionnaire (age, gender, race/ethnicity, cigarette smoking, heavy drinking, substance use, antidepressant drugs) or directly extracted from medical records (duration of HIV diagnosis and status of antiretroviral therapy).

Statistical analysis

Bivariate analyses were performed and potential risk factors (with a difference at $p < 0.10$ level) between participants with and without hypertension were included in multivariate analyses. To detect interactions, participants were categorized into four subgroups with BMI (normal: BMI < 25 kg/m^2; increased: BMI ≥ 25 kg/m^2) and pVL (detectable: VL ≥ 200 copies/ml; undetectable: VL < 200 copies/ml). Multiplicative interaction was examined using logistic regression by adding a product term of categorized BMI and pVL; the additive interaction was evaluated using three key measures: Relative Excess Risk due to Interaction (RERI), Attributable Proportion (AP) and Synergy index (S) (Andersson, Alfredsson, Kallberg, Zdravkovic, & Ahlbom, 2005; Kalilani & Atashili, 2006; Knol et al., 2011; Lou et al., 2018; Olofindayo et al., 2015). Potential risk factors were compared across the four groups using the Chi-square test, ANOVA, or Wilcoxon rank-sum test. All the analyses were adjusted using inverse probability weighting methods to handle missing data (Seaman & White, 2013). The significance level was set at $p < 0.05$ (two-sided). Statistical analyses were performed using the software SAS version 9.4.

Results

Participant characteristics

Participant characteristics are summarized in Table 1. The median age (IQR) of the participants was 45.8 (11.5), with 64.6% being males, 59.8% blacks. Of these participants, 40.4% were hypertensive, 62.3% had increased BMI, and 26.3% had detectable pVL. Table 2 contrasts the characteristics of participants across the four subgroups. Compared to those with normal BMI and undetectable pVL, PLWH with increased BMI and detectable pVL was associated with younger age, diabetes, not on ART, longer duration of HIV diagnosis and lower CD4+ counts ($p < 0.05$ for all).

Interactions between BMI and pVL

Results from the logistic regression assessing multiplicative interaction are presented in Table 3. Compared to the participants with a normal BMI and undetectable pVL, the risk of hypertension increased for those whose BMI was higher than normal with OR [95%CI] =

1.80 [1.02, 3.20]. The risk further increased for the participants with increased BMI and detectable pVL with OR [95%CI] = 3.54 [1.71, 7.31] ($p\text{-}value_{interaction} = 0.01$).

Results from the addictive interaction analyses are summarized in Table 4. The RERI [95%CI] of hypertension for participants with an increased BMI and detectable pVL was 1.89 [0.76, 4.79] and the AP due to the interaction was 0.64 [0.32, 0.95] ($p < 0.05$ for both).

Discussion

In this study, we examined the interaction between increased BMI and detectable pVL on hypertension. Of the study sample, 40.4% were hypertensive, 62.3% with BMI higher than normal, and 26.3% with a detectable pVL. We found a significant interaction between increased BMI and detectable pVL on hypertension and the effect was present based on both the multiplicative and the additive scales. The hypertension risk was 1.8 times for PLWH with an increased BMI alone relative to those with a normal BMI; the risk increased to 3.54 time for those with an increased BMI plus a detectable pVL. Overall 64% of the hypertension risk was due to the interaction.

Findings of this study confirm the significance of body weight control as a measure of hypertension prevention among PLWH. Increased BMI is a well-established predictor of hypertension (De Marco et al., 2014; Malaza, Mossong, Barnighausen, & Newell, 2012; Willig et al., 2015). In addition to some modifiable and non-modifiable risk factors such as age, sedentary time, physical inactivity, dietary practices and socioeconomic level (Ezechi et al., 2016; Semu et al., 2016), one challenge for weight control for PLWH is the use of ART regimens that may lead to weight gain (Kaplan et al., 2007; McComsey et al., 2011; Tate et al., 2012). Research has shown that ABC-3TC- and TDF-FTC-based regimens were significantly associated with limb and visceral fat (Kaplan et al., 2007; McComsey et al., 2011; Tate et al., 2012; Yuh et al., 2015).

The synergetic effect of detectable pVL with increased BMI detected in our study suggests great potentials to reduce the risk of hypertension up to 64% by weight control and effective ART to bring pVL to an undetectable level. The association between increased BMI and hypertension was much stronger among PLWH with a detectable pVL than those with an undetectable pVL. Given increased BMI contributes to hypertension and the effects are dependent of pVL, interventions to address increased BMI should become more routine in HIV clinical and public health settings for hypertension prevention, particularly among those with detectable pVL. Our study demonstrated a high variability of potential risk factors across the 4 groups. Therefore, multiple risk factor management is essential for hypertension prevention among PLWH.

Mechanisms underlying interactions between increased BMI and undetectable pVL on the risk of hypertension is complex. Findings of this study provided empirical evidence supporting the mechanism that may be commonly derived from HIV infection and increased BMI, including of chronic inflammation, immune activation, endothelial dysfunction, and arterial stiffness (Baekken, Os, Sandvik, & Oektedalen, 2008; Koethe et al., 2013; Okello et

al., 2015; Re, 2009; Rodriguez-Hernandez, Simental-Mendia, Rodriguez-Ramirez, & Reyes-Romero, 2013; Rogalska-Plonska et al., 2017; Society, 2004).

It is worth mentioning that the proportion of hypertension observed in our sample was higher than that for the general population (CDC, 2017; Mills et al., 2016) and PLWH subpopulations from other studies (Antonello et al., 2015; De Socio et al., 2014; Miguez-Burbano et al., 2014; Myerson et al., 2014; Wiernik et al., 2013); the proportion of participants with increased BMI in our sample was also higher than reported from studies among other PLWH subpopulations (Ding et al., 2017; Mashinya, Alberts, Colebun-ders, & Van Geertruyden, 2016; Ogunmola, Oladosu, & Olamoyegun, 2014; Thompson-Paul et al., 2015). One possible explanation could be due to the higher proportion of black participants in our study with a Florida sample than in others.

There are limitations to this study. No causal relationship is established due to the cross-sectional nature of this study. Some unmeasured confounders did not include, such as lipoprotein, cholesterol, diet, and linkage to HIV care. Other bodyweight measures were not assessed, such as waist circumference, hip circumference and waist-to-hip ratio (Ding et al., 2017). The impact of different ART regimens on BMI and hypertension could not be assessed because of the small sample size. Further research is necessary to explore more. Finally, participants in this study were recruited from one state. Caution is needed when the study findings are generalized to PLWH in other places.

Despite the limitations, our study provides empirical data supporting the interaction between increased BMI and detectable pVL on the risk of hypertension among PLWH in Florida. This finding suggests the significance of an intervention combining weight and viral load control for hypertension prevention among PLWH. Future research should seek to identify and address mechanisms underlying the interactive effects.

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Table 1.

Participant characteristics and bivariate analysis between hypertensive and normotensive PLWH, the Florida Cohort, October 2014 - December 2018.

Variables	Total	Hypertensive	Normotensive	*p-value*
Total, *n* (%)	659	266 (40.4)	393 (69.6)	
Age, *n* (%)				<0.01
18–34	124 (19.3)	23 (8.9)	101 (26.3)	
35–44	134 (20.9)	44 (17.0)	90 (23.4)	
45–54	242 (37.7)	107 (44.5)	135 (35.2)	
≥55	142 (22.1)	84 (32.6)	58 (15.1)	
Median (IQR)	48 (38,54)	51(45, 57)	46 (35, 52)	<0.01
Male, *n* (%)	415 (64.6)	144 (55.8)	271 (70.6)	<0.01
Race/ethnicity, *n* (%)				<0.01
Hispanics	95 (14.8)	37 (14.3)	58 (15.1)	
Whites	140 (21.8)	42 (16.3)	98 (25.5)	
Blacks	384 (59.8)	176 (68.2)	208 (54.2)	
Other	23 (3.6)	3 (1.2)	20 (5.2)	
BMI (kg/m^2), *n* (%)				<0.01
Underweight	20 (3.0)	9 (3.4)	11 (2.8)	
Normal	229 (34.8)	58 (21.8)	171 (43.5)	
Overweight	206 (31.3)	87 (32.7)	119 (32.8)	
Obesity	204 (31.0)	112 (42.1)	92 (23.4)	
Median (IQR)	26.9 (23.2, 31.4)	28.5 (25.0, 33.6)	25.5 (22.3, 29.9)	<0.01
Cigarette smoking, *n* (%)				0.28
Never	180 (28.8)	75 (30.0)	105 (28.0)	
Current	330 (52.8)	123 (49.2)	207 (55.2)	
Former	115 (18.4)	52 (20.8)	63 (16.8)	
Alcohol use, *n* (%)				0.54
Never drank any alcohol	53 (8.8)	24 (10.0)	29 (8.1)	
No drinks in the past year	129 (21.5)	54 (22.6)	75 (20.7)	
No heavy drinking in the past year	365 (60.7)	137 (57.3)	228 (62.9)	
Heavy drinking in the past year	54 (9.0)	24 (10.1)	30 (8.3)	

Variables	Total	Hypertensive	Normotensive	*p-value*
Antidepressant drugs in the past 6 months, *n* (%)				0.01
No	390 (62.7)	139 (55.8)	251 (67.3)	
Yes and currently taking it	183 (29.4)	85 (34.1)	98 (26.3)	
Yes but not taking it now	49 (7.9)	25 (10.1)	24 (6.4)	
Any substance use in the past year, *n* (%)	226 (40.7)	104 (47.5)	122 (36.2)	<0.01
Diabetes, *n* (%)	79 (12.0)	59 (22.2)	20 (5.1)	<0.01
Renal Disease, *n* (%)	75 (12.3)	48 (19.7)	27 (7.4)	<0.01
Depression, *n* (%)	189 (28.7)	95 (35.7)	94 (23.9)	<0.01
Currently on ART, *n* (%)	539 (81.8)	216 (81.2)	323 (82.2)	0.75
Nadir CD4 + T cell counts, cells/mm^3, median (IQR)	334 (146, 558)	349 (160, 570)	312 (141, 544)	0.26
Plasma viral load ≥ 200 copies/ml, *n* (%)	173 (26.3)	62 (23.3)	111 (28.2)	0.16
CD4 + T cell counts ≥ 350 cells/mm^3, *n* (%)	464 (71.3)	201 (75.9)	263 (68.1)	0.03
Years of HIV Diagnosis, median (IQR)	10 (4, 16)	13 (7, 18)	8 (3, 15)	<0.01

Note: ART: Antiretroviral Therapy; BMI: Body Mass Index; CI: Confidence Interval; IQR: Interquartile Range.

Table 2.

Distribution of potential risk factors among participants by BMI and pVL, the Florida Cohort, October 2014 - December 2018.

Variables	BMI < 25 pVL < 200	BMI < 25 pVL ≥ 200	BMI ≥ 25 pVL < 200	BMI ≥ 25 pVL ≥ 200	*p-value*
No. of participants	172	77	314	96	
Age, years, mean (SD)	49 (35, 55)	45 (31, 51)	49 (43, 54)$^{\#}$	44 (33, 50) $^{*\$}$	<0.01
Female, *n* (%)	38 (22.6)	13 (17.1)	137 (45.1) $^{*\#}$	39 (41.5) $^{*\#}$	<0.01
Hispanics, *n* (%)	24 (13.9)	9 (11.7)	47 (15.0)	15 (15.6)	0.88
Diabetes, *n* (%)	12 (7.0)	2 (2.6)	49 (15.6) $^{*\#}$	16 (16.7) $^{*\#}$	<0.01
Depression, *n* (%)	54 (31.3)	18 (23.4)	90 (28.7)	27 (28.1)	0.64
Current use of antidepressant drugs, *n* (%)	49 (30.1)	20 (26.3)	130 (44.7) $^{*\#}$	33 (35.9)$^{\$}$	<0.01
Renal disease, *n* (%)	21 (12.9)	6 (7.0)	34 (11.7)	15 (16.8)	0.30
Current smoking, *n* (%)	98 (60.9)	54 (73.0) *	131 (44.9) $^{*\#}$	48 (53.3)$^{\#}$	<0.01
Current drinking, *n* (%)	119 (71.7)	63 (87.5) *	199 (67.4)$^{\#}$	63 (67.7)$^{\#}$	0.01
Current any substance use, *n* (%)	88 (61.9)	62 (88.6) *	128 (38.8) $^{*\#}$	52 (59.8)$^{\#}$	<0.01
On ART, *n* (%)	153 (88.9)	42 (54.6) *	279 (88.9)$^{\#}$	65 (67.7) $^{*\$}$	<0.01
Nadir CD4 count, median (IQR)	383 (191, 574)	336 (157, 508)	348 (155, 620)	266 (116, 497)$^{\$}$	0.02
CD4 count ≥ 350 cells/mm^3, *n* (%)	119 (70.0)	34 (44.7) *	252 (81.3) $^{*\#}$	59 (62.1) $^{*\#\$}$	<0.01
Duration of HIV diagnosis, median (IQR)	10 (4,15)	8 (1,2)	11 (5,17)$^{\#}$	15 (9,20) $^{*\$}$	0.03

Note:

*Compared to participants with BMI < 25 kg/m^2 and HIV VL < 200 copies/ml, p <0.05;

$^{\#}$Compared to participants with BMI < 25 kg/m^2 and HIV VL ≥ 200 copies/ml, p <0.05;

$^{\$}$Compared to participants with BMI ≥ 25 kg/m^2 and HIV VL < 200 copies/ml, p <0.05; BMI: Body Mass Index (kg/m^2); ART: Antiretroviral Therapy; BMI: Body Mass Index; IQR: Interquartile Range; pVL: plasma Viral Load.

Table 3.

Interactive effect of increased BMI and detectable pVL on hypertension among PLWH, the Florida Cohort, October 2014 - December 2018.

BMI (kg/m^2)	PVL (copies/ml)	Hypertension % (n/N)	OR [95% CI]
<25	<200	31.9 (55/172)	1.00
<25	≥200	15.6 (12/77)	0.62 [0.25, 1.51]
≥25	<200	44.8 (149/314)	1.80 [1.02, 3.20]
≥25	≥200	52.1 (50/96)	3.54 [1.71, 7.31]
$p\text{-value}_{interaction}$			0.01

Note: BMI: Body Mass Index; CI: Confidence Interval; OR: Odds Ratio; pVL: plasma Viral Load; Models were adjusted for age, gender, race/ethnicity, diabetes, depression, antidepressant drugs, renal disease, CD4+ T cell counts, and duration of HIV diagnosis.

Table 4.

Additive interaction of increased BMI and detectable pVL on hypertension among PLWH, the Florida Cohort, October 2014 - December 2018.

Measures*	Estimate	Lower	Upper
Relative Excess Risk due to Interaction (RERI)	1.89	0.76	4.79
Attributable Proportion due to Interaction (AP)	0.64	0.32	0.95
Synergy index (S)	7.65	0.29	204.50

Note:

*If there was no biological interaction the 95% CI of RERI and AP would include 0 and the 95% CI of S would include 1. Models were adjusted for age, gender, race/ethnicity, diabetes, depression, antidepressant drugs, renal disease, CD4+ T cell counts, and duration of HIV diagnosis.