Published in final edited form as:
AIDS. 2009 November 13; 23(17): 2317–2322. doi:10.1097/QAD.0b013e328332204e.

Role of Metabolic Syndrome Components in HIV Associated Sensory Neuropathy

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Abstract

Objectives—Sensory neuropathy (SN) is a common peripheral nerve complication of HIV infection and highly active antiretroviral therapy. Metabolic syndrome (MetS), a cluster of risk factors for atherosclerosis and microvascular disease, is associated with SN in HIV-uninfected (HIV −) persons. We examined if MetS or its components predispose individuals to HIV-SN.

Design—From a prospective multicenter cohort of 1,556 HIV+ subjects, a subgroup (n=130) with fasting laboratory tests and SN assessment was selected.

Methods—SN was defined by symmetrically decreased reflexes or sensation loss in the legs. MetS was defined by presence of ≥3 risk factors: mean arterial pressure (MAP) ≥100 mm Hg; triglycerides (TRG) ≥150 mg/dl and high-density lipoprotein cholesterol (HDL-C) <40 mg/dL for males, <50 mg/dL for females; body mass index (BMI) >25 kg/m^2; plasma glucose (GLU) ≥100 mg/dl and self-reported diabetes (DM II). Multivariate logistic regression examined the association between HIV-SN and MetS.

Results—After controlling for HIV-SN risk factors- age, CD4 current, length of HIV infection, use of dideoxynucleoside reverse transcriptase inhibitors and protease inhibitors; MetS was not associated with HIV-SN (p=0.72). However, when each MetS component was assessed, elevated TRG was a significant risk factor for HIV-SN. From the larger cohort, both DM II (OR=1.4, p<0.01) and elevated TRG (OR=1.4, p=0.01) were risk factors for HIV-SN.

Conclusion—The risk of HIV-SN was increased for DM II and elevated TRG, but not other MetS components. Both increase the risk of SN in HIV- populations, but the mechanism(s) remains unclear.

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Author contributions: Study design: BA, DR, JM-B, RJE, JAM
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Drafting of paper: BA, DR, RJE, DMS, DBC, JCM, IG, JAM
Revisions of paper: BA, FV, DR, RJE, DMS, DBC, JCM, IG, JAM

Keywords

HIV; sensory neuropathy; metabolic syndrome; highly active antiretroviral therapy

Introduction

The term metabolic syndrome (MetS) reflects the concurrence of risk factors for atherosclerosis and microvascular disease and includes obesity, glucose intolerance, hypertension, and dyslipidemias. The prevalence of MetS in HIV infected (HIV+) subjects ranges from 25–96% depending on definition used [1,2].

HIV-associated sensory neuropathy (HIV-SN) is caused by either HIV infection or several highly active antiretroviral therapy (HAART) medications, including dideoxynucleoside reverse transcriptase inhibitors (d-drugs), and is among the most common neurological complications of HIV [3]. HIV-SN subjects present with numbness, paresthesias, and/or pain causing decreased mobility [4]. Prior to HAART the risk and severity of HIV-SN correlated with advanced immunosupression (lower CD4 cell count) and increased plasma HIV viral loads [5]. HAART has improved immune function, suppressed HIV replication, and decreased the incidence, but not the prevalence of HIV-SN [6–8]. Continued persistence suggests other contributing etiologies [4].

In HIV-uninfected (HIV−) individuals, SN is associated with MetS components [9,10]. We evaluated if MetS, or its components, predisposes individuals to HIV-SN within a multicenter, prospective, cross-sectional study investigating the effects of antiretroviral (ARV) therapy in the nervous system: CNS HIV Antiretroviral Therapy Effects Research (CHARTER).

Methods

Study Design

HIV+ subjects for CHARTER were recruited from six North American academic HIV+ clinics. The Institutional Review Boards at each site approved all research. All subjects completed a cross-sectional evaluation consisting of comprehensive neuromedical assessments, phlebotomy, and lumbar puncture. From this cohort, metabolic substudy subjects were selected.

Subjects

At the time of analysis 1,556 HIV+ subjects were enrolled and examined cross-sectionally within CHARTER from June 2006 to September 2007. Informed consent was received from each participant prior to enrollment. Those meeting criteria for the metabolic subgroup (n=130) had a fasting blood draw and neuromedical examination.

Characterization of HIV infection

Blood CD4 cell counts were measured by flow cytometry. HIV RNA levels were quantified in plasma and cerebrospinal fluid (CSF) by reverse transcriptase-polymerase chain reaction (Amplicor®, Roche Diagnostic Systems, Indianapolis, IN) using an ultrasensitive assay (lower quantification limit < 50 copies/mL). HIV RNA levels were $\log_{10}$ transformed and analyzed continuously and categorically, based on whether or not the value was below the limit of the assay (undetectable vs. detectable).

Details of past and current antiretroviral usage were captured by combined self-report and interview questionnaires. Data collected included usage dates, dose, and schedule for each

antiretroviral drug. Antiretroviral usage was categorized as currently on, past use, or never used with particular emphasis on dideoynucleoside antitretroviral drugs (d-drugs), such as stavudine and didanosine. These drugs can be associated with HIV-SN. Nadir CD4 count since HIV infection was self-reported.

HIV-SN Definition

Trained personnel completed targeted, standardized neurological examinations to diagnose HIV-SN. Clinical signs associated with HIV-SN included diminished vibration sense in great toes, decreased or inability to discriminate between sharp and dull sensation in the feet, and absent or weakened bilateral ankle reflexes. HIV-SN diagnosis was based on presence of at least one sign bilaterally and symmetrically [4]. Self-reported symptoms of HIV-SN (including loss of sensation, hyperalgesia, tingling, pain, or bilateral burning in the feet) were recorded but not required for HIV-SN diagnosis.

MetS Definition

MetS was defined as ≥ 3 of the following criteria from the National Cholesterol Education Program Adult Treatment Panel III [11]: 1) elevated mean arterial pressure (MAP) ≥ 100 mm Hg; 2) elevated fasting triglycerides (TRG) (≥ 150 mg/dL) 3) reduced high-density lipoprotein cholesterol (HDL-C) (< 40 mg/dL (males) or < 50 mg/dL (females)); 4) obesity with elevated body mass index (BMI) (> 25 kg/m^2); 5) elevated fasting plasma glucose (GLU) (≥ 100 mg/dL) [12].

Clinical and Metabolic Laboratory Measures

All metabolic substudy subjects endorsed abstaining from food and drink for eight hours prior to their blood draw. Blood samples for glucose measurement were collected in serum separator tubes. After clotting, serum was separated and sent to each institution's laboratory for glucose, total cholesterol, triglycerides, high-density lipoprotein (HDL) and low-density lipoprotein (LDL) measures. MetS components were dichotomized as abnormal or normal. All BP measurements were obtained using automated calibrated mercury sphygmomanometers and appropriate cuff sizes. Systolic and diastolic BP measures were obtained from seated subjects [13,14]. Height and weight for calculating body mass index (BMI) were measured at entry. Diagnosis of DM II and use of diabetes medication were self-reported.

Statistical Analysis

Univariate analyses assessed for possible differences in prevalence of MetS within HIV+ subjects with and without SN using a Chi-square test. Risk factors including demographic characteristics (age and gender), markers of HIV disease progression (current CD4 counts, CD4 nadir, length of HIV infection, plasma and CSF viral load), antiretroviral characteristics (d-drug and protease inhibitor (PI) use, HAART duration), diabetes, and hepatitis C co-infection were evaluated for differences between those with and without HIV-SN using either t-tests or Chi-square tests. Logistic regression was used to determine the unadjusted odds ratios for each independent variable as a predictor for HIV-SN. Adjusted odds ratios were determined after correcting for age, current CD4 count, length of infection, d-drug and PI use. All MetS components were logarithmically transformed to reduce skewness and equalize variance. A univariate analysis for each MetS component and HIV-SN was assessed for the metabolic subgroup and larger cohort. All analyses were performed using JMP statistical package (V6.0, SAS Institute Inc.).

Results

Comparison of participants with and without HIV-SN

The cohort was primarily male (85%) ranging in age from 20–67 years old with HIV-SN participants tending to be older (Table 1). Most subjects were on a stable HAART regimen (72%) and were immune reconstituted as evidenced by a high current median CD4 count (513 cells/mm^3) and a median plasma viral load below limits of detection in both the CSF and plasma (50 or 1.7 log_{10} copies/mL), despite a relatively low median nadir CD4 count (137 cells/mm^3).

Risk factors of HIV-SN

Older age (OR=1.06, p<0.001, 95%CI: 1.05–1.15), increased duration of infection (OR=1.11, p<0.001, 95%CI: 1.07–1.25), and lower CD4 nadir (OR=1.01, p=0.04, 95%CI: 1.00–1.01) significantly increased the risk of HIV-SN. Likewise, past d-drug (OR=1.71, p<0.01, 95%CI: 1.30–6.68) and PI (OR=2.63, p<0.01, 95%CI: 1.23–5.52) use were associated with HIV-SN. Neither hepatitis C co-infection (n=17) nor self-reported type 2 diabetes mellitus (DM II, n=11) were associated with HIV-SN.

MetS and HIV-SN were unrelated

Both MetS and HIV-SN were prevalent in the metabolic substudy group. Overall, ~ 32% (42/130) of subjects met MetS criteria. HIV-SN was diagnosed in 55% of the cohort (71/130). However, MetS was not associated with HIV-SN (p=0.69).

After controlling for possible HIV-SN risk factors, MetS remained unrelated to HIV-SN

We assessed four potential HIV risk factors that could confound the relationship between MetS and HIV-SN (Table 1): demographic characteristics, HIV disease status, antiretrovirals, and aspects of past medical history. After controlling for age, CD4 current, length of HIV infection, d-drug use ever, and past PI use in a multivariate logistic regression model, MetS remained unrelated to HIV associated HIV-SN (p=0.72).

Of the MetS components, only elevated TRG increased the risk for HIV-SN

Since MetS is constellation of disorders, we assessed the role of each MetS component in HIV-SN. Univariate analysis using each factor as a continuous variable revealed that only elevated TRG was associated with HIV-SN (p=0.009, Table 2). From the larger CHARTER cohort who had either fasting or non-fasting TRG measures (n=1,518 subjects), elevated TRGs were associated with HIV-SN (OR=1.30, p=0.01, 95%CI: 1.06–1.59). However, no significant association occurred between age and TRG for the two groups.

Self-reported DM II was a risk factor in the larger CHARTER cohort

Since the total number of subjects with DM II was low (11/130) in the metabolic substudy group, we examined the association of self-reported DM II and HIV-SN in the CHARTER cohort. DM II was associated with increased risk of HIV-SN (OR=1.41, p<0.01, 95%CI: 1.08–1.81).

Discussion

MetS was not associated with HIV-SN in this cohort of HIV+ patients from multiple institutions (p=0.72). However, two MetS components, DM II and elevated TRG, increased the risk of HIV-SN. Specifically, elevated TRG (>150 mg/dl) conferred a 30% greater risk of HIV-SN and self reported DM II conferred a 40% greater risk of HIV-SN. Both factors have been associated with idiopathic SN in HIV-uninfected populations [15]. Without HIV− uninfected

controls, we cannot address whether the increased risks of HIV-SN from these MetS components produce an additive or interactive effect with HIV in causing SN.

Other clinical correlates for HIV-SN in this study were similar to those found in previous studies. These risk factors included increased age, low current CD4 count, length of HIV infection, and d-drug exposure [4,16,17].

The prevalence of HIV-SN has persisted despite widespread adoption of HAART regimens avoiding d-drugs [6]. This result is possibly due to persisting symptoms after discontinuing d-drugs [18]. This study, like earlier analyses, showed that the prevalence of HIV-SN depends on definition used. When electromyographic criteria was applied, the prevalence was low (1.8%) [19], but if subclinical or asymptomatic neuropathy measures were included, the prevalence was higher (62%) [20,21]. Our observed prevalence of HIV-SN (57%) is similar to the larger CHARTER cohort, in which it is nested [22].

The relationship between DM II and elevated fasting TRGs and SN is similar to a study of HIV-uninfected adults [23]. The association with DM II was clearly demonstrated in the larger CHARTER cohort, but may have been missed in the metabolic subgroup as a small number of DM II (8%) could lead to an underestimation of HIV-SN prevalence. The effect of TRG on HIV-SN may be complicated by statins used to treat elevated TRG as these medications may confer an additional risk. Future studies investigating the role of statins in HIV-SN are required.

The relationship between DM II and HIV-SN resembles a similar, but stronger, relationship in the same cohort between DM II and cognitive dysfunction. Diabetic patients had a 5-fold increased risk of neurocognitive impairment compared to nondiabetic HIV+ subjects. Median global deficit scores, a measure of each patient's overall cognitive dysfunction, were higher in diabetics compared to nondiabetics (median GDS=0.87 vs. 0.13, $p<0.01$). Whether the same mechanisms underlie damage in the CNS and peripheral nerves remains unclear.

Limitations of this study are its cross-sectional design and small study population which may have decreased the power to detect the impact of individual MetS components on HIV-SN. Its strengths include the recruitment of HIV+ subjects from multiple institutions across the US; the assessment of HIV-SN by trained personnel using standardized methods; and thorough accounting for potential confounding factors such as age, HIV infection status, and drug exposures.

These findings illustrate the pathogenic complexity HIV-SN to which both HIV infection and its treatment are known contributors. Additionally, this study shows that DM II and hypertriglyceridemia independently contribute to SN. While the widespread abandonment of d-drugs in developed countries should decrease the incidence of HIV-SN, the continued use of other HAART drugs causing SN, in particular PIs, may increase the risk of MetS and help sustain its high prevalence.

Acknowledgments

The CNS HIV Anti-Retroviral Therapy Effects Research (CHARTER) group is affiliated with the Johns Hopkins University, Mount Sinai School of Medicine, University of California, San Diego, University of Texas, Galveston, University of Washington, Seattle, Washington University, St. Louis, and is headquartered at the University of California, San Diego. Members of the CHARTER group include: Director: Igor Grant, MD; Co-Directors: J. Allen McCutchan, MD, Ronald J. Ellis, MD, PhD, Thomas D. Marcotte, PhD; Center Manager: Donald Franklin, Jr; Neuromedical Component: Ronald J. Ellis, MD, PhD (P.I.), J. Allen McCutchan, MD, Terry Alexander, RN; Laboratory, Pharmacology and Immunology Component: Scott Letendre, MD (P.I.), Edmund Capparelli, PharmD, Janis Durelle; Neurobehavioral Component: Robert K. Heaton, PhD (P.I.), J. Hampton Atkinson, MD, Steven Paul Woods, PsyD, Matthew Dawson; Virology Component: Joseph K. Wong, MD (P.I.); Imaging Component: Terry Jernigan, PhD (Co-P.I.), Michael J. Taylor, PhD (Co-P.I.), Rebecca Theilmann, PhD; Data Management Unit: Anthony C. Gamst, PhD (P.I.), Clint Cushman; Statistics Unit: Ian Abramson, PhD (P.I.), Christopher Ake, PhD, Florin Vaida, PhD; Protocol

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AIDS. Author manuscript; available in PMC 2010 November 13.

Table 1

Comparison of demographics, HIV disease status, antiretroviral exposure, and comorbidities in HIV infected patients with and without sensory neuropathy (HIV-SN)

Age, CD4 current, duration of HIV infection, past and currently dideoynucleoside antiretroviral (d-drug) exposure, and past protease inhibitor (PI) exposure were significantly associated with HIV-SN.

	HIV-SN (n=71)	No HIV-SN (n=59)	p value	OR	95% CI
Demographic Characteristics					
Age (years old)	49	43	**<0.001**	**1.06**	**1.05–1.15**
< 30 years old (% total)	**1 (1.4%)**	**3 (5.1%)**		Ref	
30–39 years old (% total)	**9 (12.7%)**	**15 (25.4%)**	0.63	1.8	0.16–20.02
40–49 years old (% total)	**30 (42.2%)**	**33 (55.9%)**	0.62	2.72	0.27–27.66
50–59 years old (% total)	**21 (29.6%)**	**7 (11.9%)**	**0.08**	**9.0**	**0.80–101.2**
≥ 60 years old (% total)	**10 (14.1%)**	**1 (1.7%)**	0.03	30.0	1.41–638.15
Men (%)	86	83	0.81		
HIV Disease Status					
Current CD4 (cells/mm3)	599	431	**0.04**	**1.01**	**1.00–1.01**
Nadir CD4 (cells/mm3)	112	181	0.14		
Length of infection (mos)	15	9	**< 0.001**	**1.11**	**1.07–1.25**
CSF VL ($\log_{10}$)	1.7	1.7	0.60		
Plasma VL ($\log_{10}$)	1.7	1.7	0.31		
Antiretroviral Characteristics					
D-drug exposure duration (mos)	4.6	4.0	0.53		
D-drug Use					
Current	19	11	**0.04**	**2.65**	**1.04–6.63**
Past Use	31	16	**< 0.01**	**1.71**	**1.30–6.68**
Never	21	32	-	-	
PIs Ever					
Current	6	5	0.41		
Past Use	45	25	**< 0.01**	**2.63**	**1.23–5.52**
Never	20	29	-	-	

	HIV-SN (n=71)	No HIV-SN (n=59)	p value	OR	95% CI
Medical History					
% HCV	11	15	0.6		
% Diabetes	11	4	0.18		

SN= sensory neuropathy

CSF= cerebral spinal fluid

PI- protease inhibitor

HCV= hepatitis C infection

AIDS. Author manuscript; available in PMC 2010 November 13.

Table 2

Of the components of the metabolic syndrome (MetS), only elevated triglycerides (TRG) emerged as a risk factor for HIV associated DSPN.

Means of MetS Components	HIV-SN	No HIV-SN	p value
Glucose (mg/dL)	86	89	0.44
Glucose (mg/dL) in <30 years old	85	81	0.65
Glucose (mg/dL) in 30–39 years old	84	90	0.22
Glucose (mg/dL) in 40–49 years old	89	92	0.57
Glucose (mg/dL) in 50–59 years old	86	93	0.54
Glucose (mg/dL) in ≥60 years old	84	78	0.75
BMI (kg/m^2)	26	25	0.36
MAP (mm Hg)	93	95	0.92
HDL (mg/dL)	42	46	0.41
TRG (mg/dL)	176	138	**0.009**
TRG (mg/dL) in <30 years old	64	149	0.18
TRG (mg/dL) in 30–39 years old	280	136	0.36
TRG (mg/dL) in 40–49 years old	248	174	**0.005**
TRG (mg/dL) in 50–59 years old	177	131	0.63
TRG (mg/dL) in ≥ 60 years old	203	57	0.11

BMI= body mass index

MAP= mean arterial pressure

HDL= high density lipoprotein

TRG= triglycerides