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The association between hepatitis C infection and prevalent cardiovascular disease among HIV-infected individuals

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Abstract

Objective— To examine the association between hepatitis C and prevalent cardiovascular disease (CVD) among HIV-infected individuals.

Design— A cross-sectional analysis of data from the HIV–Longitudinal Interrelationships of Viruses and Ethanol (HIV–LIVE) cohort, a prospective cohort of HIV-infected individuals with current or past alcohol problems.

Methods— We analysed health questionnaire and laboratory data from 395 HIV-infected individuals (50.1% co-infected with hepatitis C) using logistic regression to estimate the odds ratio (OR) for the prevalence of CVD among those co-infected with hepatitis C and HIV compared with those infected with HIV alone.

Results— The prevalence of CVD was higher among those co-infected with hepatitis C compared with those with HIV alone (11.1 versus 2.5%, respectively). After adjusting for age, the OR for the prevalence of CVD was significantly higher among those with hepatitis C co-infection (adjusted OR 4.65, 95% confidence interval 1.70–12.71). The relationship between hepatitis C and CVD persisted when adjusting for age and other sociodemographic characteristics, substance use, and cardiovascular risk factors in separate regression models.

Conclusion— Co-infection with hepatitis C among a cohort of HIV-infected individuals was associated with a higher age-adjusted odds for the prevalence of CVD. These data suggest that

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hepatitis C infection may be associated with an increased risk of CVD among those co-infected with HIV.

Keywords

Cardiovascular disease; cardiovascular risk factors; hepatitis C; HIV; myocardial infarction; substance abuse

Introduction

Whether certain chronic infections are a risk factor for cardiovascular disease (CVD) is uncertain. Studies suggest that there is a link between some infectious pathogens and atherosclerosis [1,2]. The mechanism for the development of atherosclerosis may be colonization of the vasculature by the pathogen [3] or stimulation of the inflammatory cascade [4]. Some studies have reported a higher prevalence of cardiovascular risk factors [5,6], carotid atherosclerosis [7–9] and coronary heart disease [10] among individuals with hepatitis C infection. In contrast, other studies found no such association [11–13], but were limited by the number of hepatitis C-infected participants [11] and the lack of hepatitis C viral RNA [11–13] and substance use data. The absence of information on injection drug, cocaine and alcohol use could confound the relationship between hepatitis C and CVD [11,12].

With the advent of antiretroviral therapy and prolonged survival, coronary heart disease is now common among individuals with HIV infection [14]. Of those infected with HIV, studies have reported co-infection with hepatitis C ranging from 16.1% [15] up to 30% [16]. Compared with individuals infected only with HIV or hepatitis C, those who are co-infected may be at a higher risk of atherosclerosis and CVD. Therefore, we investigated the association between hepatitis C and CVD in a cohort of individuals with HIV infection and substance abuse.

Methods

We analysed baseline data collected from 395 HIV-infected participants in the HIV–Longitudinal Interrelationships of Viruses and Ethanol (HIV–LIVE) study, a prospective cohort of individuals with HIV infection and current or past alcohol problems. During August 2001 to July 2003, 400 HIV–LIVE participants were enrolled from four different sources: (i) a previous cohort of HIV-infected participants with alcohol problems; (ii) Boston Medical Center’s Diagnostic Evaluation Unit, an intake clinic for HIV-infected patients; (iii) Beth Israel Deaconess Medical Center primary care and specialty clinics; and (iv) other local healthcare sites or shelters in the Boston area. Inclusion criteria included: (i) an HIV antibody test positive by enzyme-linked immunosorbent assay and confirmed by Western blot; (ii) two or more affirmative responses to the CAGE alcohol screening questionnaire [17] or by physician-investigator diagnosis of alcoholism; (iii) ability to speak English or Spanish; and (iv) at least one contact person who was likely to know the subject’s whereabouts. Participants were excluded if the 30-item Folstein Mini-Mental State Examination score [18] was less than 21 or a trained interviewer assessed that the patient was incapable of comprehending the informed consent or of answering interview questions. For the present cross-sectional analysis, five participants were excluded because of missing hepatitis C RNA data ($n=4$) or a history of CVD data ($n=1$). Additional study design details have been published elsewhere [19]. The Institutional Review Boards of the Boston Medical Center, Beth Israel Deaconess Medical Center, and the University of Pittsburgh approved the study.

The primary outcome variable was self-reported CVD. CVD was defined as a ‘yes’ response to one of the three following questions: ‘Has a doctor ever told you that you had. . .’ (1) peripheral vascular disease (hardening of the arteries in your neck or legs, atherosclerosis); (2)

a stroke, cerebrovascular accident, blood clot or bleeding in the brain, or transient ischemic attack; or (3) a heart attack (myocardial infarction). Our secondary outcome was myocardial infarction. Hepatitis C infection, defined as detectable hepatitis C viral RNA (either from serum collected at the time of enrollment or from participants' medical records), was the main independent variable. Additional co-variables included the following: age; race (white versus non-white); sex; current CD4 cell count (>200 cells/ μ l); obesity (body mass index ≥ 30 kg/ m^2); current adherent antiretroviral therapy use defined as 100% adherence to all antiretroviral medications over the preceding 3 days before enrollment in the study; and self-reported health conditions defined as a 'yes' response to 'Has a doctor ever told you that you had. . .' diabetes or high blood sugar or sugar, hypertension or high blood pressure, high cholesterol, renal disease (poor kidney function or blood tests showing high creatinine), anemia (low red blood cell count, hemoglobin), or cirrhosis of the liver. For substance use variables, we defined current smoking as a 'yes' response to 'Do you currently smoke cigarettes every day or on some days?'; current at-risk alcohol consumption as drinking more than 14 standard drinks for men (more than seven for women) per week or more than four drinks on one occasion for men (more than three drinks for women) in the past 30 days; cocaine use as self-reported use of 'cocaine, crack or free base'; and injection as a 'yes' response to 'In your lifetime, have you ever injected drugs?'. Homelessness was defined as any night on the street or in a shelter in the past 6 months. We defined lipodystrophy as a 'yes' response to 'Since you began taking HIV medications, have you noticed any of the following changes: more fat in the back of your neck or increase in your waist, 'butt' or chest out of proportion to your arms or legs?' [20].

Statistical analyses

We obtained descriptive statistics for all variables and assessed the relationship between hepatitis C and CVD and other co-variables using *t*-tests for continuous variables and chi-square analysis or Fisher's exact tests for categorical variables. We used logistic regression models to estimate the odds ratio (OR) for the prevalence of CVD and myocardial infarction (separate analyses for each outcome) using hepatitis C infection as the main independent variable while adjusting for age. Additional analyses adjusted for age and then each of the covariates separately. The limited number of self-reported CVD events precluded models that adjusted for all covariates simultaneously. When hepatitis C-positive and hepatitis C-negative participants were missing data on body mass index ($n=10$ and $n=9$, respectively), CD4 cell count ($n=9$ and $n=13$, respectively), HIV viral RNA ($n=16$ and $n=20$, respectively), and a history of lipodystrophy ($n=6$ and $n=1$, respectively) or when hepatitis C-negative participants were missing data on a history of injection drug use ($n=1$) and current at-risk alcohol consumption ($n=1$), we excluded those participants from the analyses adjusted for those factors. As previous studies reported an association between hepatitis C and diabetes, we performed secondary analyses restricting the sample to those who reported no history of diabetes.

Results

Compared with the 197 HIV-infected participants who were not infected with hepatitis C, those with hepatitis C co-infection ($n=198$) were significantly older (43.9 versus 40.9 years), had a higher prevalence of ever having had diabetes (10.1 versus 4.1%), cirrhosis (10.6 versus 2.5%), myocardial infarctions (6.5 versus 0.5%), and CVD (11.1 versus 2.5%), and a lower prevalence of hypercholesterolemia (19.7 versus 33.0%, $P < 0.05$ for all; Table 1). The unadjusted OR for the prevalence of CVD and myocardial infarction were significantly higher among those with hepatitis C co-infection compared with those who were infected only with HIV [OR 4.80; 95% confidence interval (CI) 1.78–12.95 and OR 13.77; 95% CI 1.78–106.33, respectively]. After adjusting for age, the association between hepatitis C and both CVD and myocardial infarction persisted (OR 4.65; 95% CI 1.70–12.71 and OR 12.86; 95% CI 1.65–100.33, respectively;

Table 2). Furthermore, after each covariate was added separately to the age-adjusted model, the relationship between hepatitis C co-infection and CVD remained unchanged (Table 2). In participants without diabetes, the association between hepatitis C and CVD was similar (OR 4.11; 95% CI 1.47–11.49) to analyses that included participants with diabetes.

Discussion

In the HIV–LIVE cohort, those co-infected with hepatitis C and HIV had a higher unadjusted and age-adjusted prevalence of CVD than those who were infected with HIV alone. When individual confounders were added separately to the age-adjusted models, the relationship between hepatitis C and CVD remained unchanged. Analyses excluding participants with diabetes yielded similar results.

Previous studies describing the relationship between hepatitis C and CVD among HIV-infected individuals are sparse and their results have been inconsistent. In the HIV Epidemiologic Research Study, hepatitis C antibody-positive women with HIV infection had higher rates of hospitalizations for CVD conditions compared with those only infected with HIV [21]. In contrast, among HIV/hepatitis C co-infected participants of the HIV Out-patient Study, the age-adjusted prevalence of CVD was not significantly higher compared with those infected with HIV alone [22]. The association between HIV and hepatitis C co-infection and CVD, however, may have varied between the studies because the definition of CVD used was not the same.

Among individuals infected with hepatitis C, but not HIV, our findings are consistent with previous studies reporting an increased prevalence of carotid atherosclerosis [7–9] and prevalent CVD [10]. Whereas some studies reported no significant association between hepatitis C and atherosclerosis, those studies were limited by sparse data on substance abuse habits, particularly injection drug use [13], and no data describing the risks of co-infection with HIV and hepatitis C, or analyses of hepatitis C viral RNA data [11–13]. Data on hepatitis C viral RNA is valuable because it can reduce the misclassification that might occur as a result of the approximately 20% of hepatitis C antibody-positive individuals who spontaneously clear the virus [23]. Given the association between hepatitis C infection and diabetes, it is possible that the effect of hepatitis C on cardiovascular risk might be mediated solely through diabetes [5]. However, our analyses, which adjusted for a history of diabetes and also restricted the sample to individuals without diabetes, suggest that diabetes is not the mechanism responsible for this association among HIV-infected individuals.

Our study has several limitations that merit comment. First, the outcome variable and several covariates were obtained by self-report, which could have resulted in some non-differential misclassification. However, our self-reported variables that were traditional CVD risk factors (e.g. hypertension and cholesterol) were associated with an increased prevalence of self-reported CVD. Second, as our definition of lipodystrophy included fat accumulation in the buttock region, there is the possibility that some non-differential misclassification occurred because lipodystrophy among HIV-infected individuals typically results in fat loss, rather than accumulation in the buttock region. Third, it is possible that differential misclassification might have occurred among those infected with hepatitis C because they might have had more contact with the healthcare system and consequently had more opportunities to be told of other health conditions (e.g. CVD). However, this potential misclassification was probably minimized by the fact that all the participants had several important health problems (e.g. HIV and alcohol) requiring frequent use of the healthcare system. Furthermore, when hepatitis C might have resulted in additional clinic visits (e.g. hepatology clinic), this patient–physician encounter would probably not have focused on CVD issues. Fourth, as there were a small number of self-reported cardiovascular events, our ability to adjust for confounders in the multivariable models

was limited. Finally, as all the participants in this cohort had alcohol problems, the findings may not be generalizable to the HIV-infected population without alcohol problems.

Nevertheless, this study suggests that among HIV-infected individuals, co-infection with hepatitis C may be independently associated with an increased risk of CVD. This finding, if confirmed in other studies, has important implications for the prevention and treatment of CVD for the substantial number of individuals around the world who are co-infected with HIV and hepatitis C.

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Table 1

Characteristics of 395 individuals with HIV and alcohol problems with and without hepatitis C co-infection.

Subject characteristics	Undetectable hepatitis C viral RNA (N=197)	Detectable hepatitis C viral RNA (N=198)
Age (median in years)	40.9	43.9*
Female, n (%)	46 (23.3)	54 (27.3)
Race, n (%)		
White		
Non-white	63 (32.0)	68 (34.0)
Prevalent CVD, n (%)	134 (68.0)	130 (66.0)
Ever had a heart attack (myocardial infarction), n (%)	5 (2.5)	22 (11.1)*
Ever had peripheral vascular disease (hardening of the arteries in your neck or legs; atherosclerosis), n (%)	1 (0.5)	13 (6.5)*
Ever had a stroke, cerebrovascular accident, blood clot or bleeding in the brain, or transient ischemic attack, n (%)	0 (0.0)	4 (2.0)
Ever had hypertension, n (%)	47 (23.9)	4 (2.0)
Ever had high cholesterol, n (%)	65 (33.0)	55 (27.9)
Ever had diabetes, n (%)	8 (4.1)	39 (19.7)*
Current smoker, n (%)	145 (73.6)	20 (10.1)*
Current at-risk alcohol consumption, n (%) ^a	66 (33.7)	157 (79.3)
Ever cocaine use, n (%)	84 (42.6)	58 (29.3)
Ever injection drug use, n (%) ^b	40 (20.4)	91 (46.0)
BMI ≥ 30 kg/m ² , n (%) ^c	39 (20.9)	176 (88.9)*
Current CD4 cell count >200 cells/ μ l, n (%) ^d	155 (82.5)	37 (19.6)
HIV viral RNA undetectable, n (%) ^e	59 (32.6)	144 (77.8)
Current adherent antiretroviral medication, n (%) ^f	86 (43.6)	52 (29.2)
Ever had lipodystrophy, n (%) ^g	49 (25.7)	99 (50.0)
Ever had cirrhosis, n (%)	5 (2.5)	56 (28.4)
Ever anemic, n (%)	47 (23.9)	21 (10.6)*
Ever had kidney disease, n (%)	7 (3.6)	51 (25.8)
Ever homeless, n (%)	40 (20.3)	17 (8.6)
		56 (28.3)

BMI, Body mass index; CVD, cardiovascular disease.

^a N = 196 hepatitis C-negative participants and n = 198 hepatitis C-positive participants who had current alcohol consumption assessed.

^b N = 196 hepatitis C-negative participants and n = 198 hepatitis C-positive participants who had history of injection drug use assessed.

^c N = 187 hepatitis C-negative participants and n = 189 hepatitis C-positive participants who had BMI measurement.

^d N = 188 hepatitis C-negative participants and n = 185 hepatitis C-positive participants who had CD4 cell count measurement.

^e N = 181 hepatitis C-negative participants and n = 178 hepatitis C-positive participants who had HIV-RNA measurement.

^f N = 185 participants (46.8%) were on antiretroviral therapy and were 100% adherent in the past 3 days; 60 participants (15.2%) were on antiretroviral therapy and were not 100% adherent; 150 (38%) were not on antiretroviral therapy.

^g N = 191 hepatitis C-negative participants and n = 197 hepatitis C-positive participants who answered the lipodystrophy question.

* P < 0.05.

Table 2

Age-adjusted associations between hepatitis C infection and cardiovascular disease among 395 individuals with HIV infection and alcohol problems.

Adjustment variable (all are adjusted for age)	Adjusted OR (95% CI) for association between hepatitis C and CVD ^a
Age only	4.65 (1.70,12.71)
Female	4.66 (1.70,12.74)
Race (white versus non-white)	4.68 (1.70, 12.83)
Ever had hypertension	4.69 (1.70, 12.89)
Ever had high cholesterol	5.44 (1.96, 15.11)
Ever had diabetes	4.52 (1.65, 12.40)
Current smoker	4.86 (1.77, 13.36)
Current at-risk alcohol consumption ^a	4.65 (1.70,12.72)
Ever cocaine use	4.66 (1.70, 12.74)
Ever injection drug use ^b	5.53 (1.99,15.33)
BMI ≥ 30 kg/m ^{2c}	4.00 (1.44, 11.14)
Current CD4 cell count >200 cells/ μ ^d	4.51 (1.64, 12.39)
HIV viral RNA (detectable versus undetectable) ^e	4.61 (1.67,12.71)
Current adherent antiretroviral medication ^f	4.65 (1.70, 12.71)
Ever had lipodystrophy ^g	4.56 (1.66, 12.51)
Ever had cirrhosis	4.91 (1.80, 13.43)
Ever anemic	4.66 (1.70, 12.79)
Ever had kidney disease	4.80 (1.76, 13.13)
Ever homeless	4.67 (1.70, 12.81)

BMI, Body mass index; CVD, cardiovascular disease; CI, confidence interval; OR, odds ratio.

^a $N=196$ hepatitis C-negative participants and $n=198$ hepatitis C-positive participants who had current alcohol consumption assessed.

^b $N=196$ hepatitis C-negative participants and $n=198$ hepatitis C-positive participants who had history of injection drug use assessed.

^c $N=187$ hepatitis C-negative participants and $n=189$ hepatitis C-positive participants who had BMI measurement.

^d $N=188$ hepatitis C-negative participants and $n=185$ hepatitis C-positive participants who had CD4 cell count measurement.

^e $N=181$ hepatitis C-negative participants and $n=178$ hepatitis C-positive participants who had HIV-RNA measurement.

^f $N=185$ participants (46.8%) were on antiretroviral therapy and were 100% adherent in the past 3 days; 60 participants (15.2%) were on antiretroviral therapy and were not 100% adherent; 150 (38%) were not on antiretroviral therapy.

^g $N=191$ hepatitis C-negative participants and $n=197$ hepatitis C-positive participants who answered the lipodystrophy question. Referent group is HIV-infected hepatitis C RNA-negative participants.