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Methamphetamine and cardiac disease among people with HIV infection

TCS Martin[1], **S Gianella**[1], **D Franklin**[1], **P Hsue**[2,*], **DM Smith**[1,3,*]

[1]Division of Infectious Diseases and Global Public Health, University of California San Diego, San Diego, CA, USA,

[2]Division of Cardiology, University of California San Francisco, San Francisco, CA, USA

[3]Veterans Affairs San Diego Healthcare System, San Diego, CA, USA

Abstract

Objectives—People living with HIV (PWH) are at elevated risk of cardiac disease compared to the general population. Methamphetamine use has been associated with structural heart disease and increased mortality from cardiovascular disease but has not been explored as a cause of cardiac disease among PWH. We sought to evaluate the association of methamphetamine use and cardiac disease among PWH.

Methods—We performed a case–control study of participant data in the HIV Neurobehavioral Research Program. Cases were defined as PWH with a history of myocardial infarction or a history of heart failure (systolic or diastolic). Covariates, including methamphetamine abuse/dependence, were assessed using multiple logistic regression.

Results—Among 3747 PWH, there was a history of myocardial infarction in 115 subjects (3.1%), and a history of heart failure in 41 (1.1%). Current or prior methamphetamine abuse/dependence was reported in 1036 (27.9%) and was not associated with myocardial infarction (P = 0.27) or heart failure (P = 0.84). In addition to traditional risk factors, variables associated with myocardial infarction included the presence of HIV infection (P = 0.01) and duration of HIV infection (P = 0.05). Variables associated with heart failure among PWH included older age, hypertension and myocardial infarction.

Conclusions—No association between methamphetamine abuse/dependence and a diagnosis of myocardial infarction or heart failure was found among PWH. Significant covariates for myocardial infarction and heart failure included traditional risk factors, the presence of HIV infection and the duration of HIV infection, emphasizing the need for optimal traditional cardiovascular risk factor management among PWH.

Correspondence: Dr Thomas C. S. Martin, Department of Infectious Diseases and Global Public Health, 9500 Gilman Drive, La Jolla, CA 92093, USA. Tel: +1 858 822 1779; fax: +1 858 246 0958; thm001@health.ucsd.edu.

*Senior authors.

Author contributions

TM, DS, SG, DF designed the study. TM, DS, SG, PH and DF were involved in data compilation, analysis and interpretation. TM wrote the manuscript which was edited and added to by DS, SG, PH and DF. All authors received and verified a final version prior to submission.

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Keywords

heart failure; HIV; methamphetamine; substance use

Introduction

Despite the beneficial effects of antiretroviral therapy (ART), people living with HIV (PWH) continue to experience higher rates of cardiovascular disease, including atherosclerotic heart disease, systolic heart failure and diastolic heart failure, when compared to the general population [1–9]. A number of mechanisms leading to increased atherogenesis and heart failure have been proposed, including increased systemic inflammation, disrupted autophagy, oxidative stress, direct viral protein effects, inflammasome formation and endoplasmic reticulum stress [10,11]. Observational studies have highlighted the presence of HIV viraemia, certain antiretroviral medication classes, immunosuppression and HIV-associated dyslipidaemia as HIV-associated factors that may predis-pose PWH to heart disease [1,3,4,12–14].

Methamphetamine use is highly prevalent among PWH in the USA and has been associated with structural heart disease and increased mortality from cardiovascular causes in the general population [15–24]. However, no studies to date have examined the association of methamphetamine use with cardiac disease among PWH. Methamphetamine is thought to exert its cardiotoxic effects through a multifactorial process, with accelerated atherosclerosis, coronary vasospasm and excessive adrenergic stimulation leading to contraction band-necrosis, inflammation and fibrosis in the myocardium [25–27]. The use of methamphetamine has also been associated with altered immune function, including increased lipopolysaccharide (LPS)-induced tumour necrosis factor (TNF) production and increased T-cell activation, proliferation and exhaustion [28,29]. We hypothesized that some of the excess cardiovascular risk observed among PWH may be accounted for by methamphetamine use.

Methods

This was a case–control study of participant data from the HIV Neurobehavioral Research Program, an organisation that coordinates several prospective research programmes [the HIV Neurobehavioral Research Center (HNRC) [30], CNS HIV Anti-Retroviral Therapy Effects Research (CHARTER) [31] and the National NeuroAIDS Tissue Consortium (NNTC) [32]] evaluating the effects of HIV, methamphetamine and other factors on the nervous system. Participant inclusion criteria differed between studies, although none excluded cardiac disease as a comorbidity. The majority of diagnoses recorded as part of this study were from self-report during study interviews. Where possible, study sites performed medical record review for confirmation.

Cases were defined as PWH with a current or prior diagnosis of heart failure (systolic or diastolic) or myocardial infarction; controls were unmatched PWH without these diagnoses. Participants without HIV infection were included to calculate the association of HIV with heart failure or myocardial infarction. Study visits for cases and controls were dispersed in

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time and occurred between 1999 and 2019; for individuals with multiple visits, only the last visit was included. Methamphetamine use was defined as present if participants fulfilled the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) criteria for methamphetamine abuse ever, methamphetamine abuse in the past 30 days, methamphetamine dependence ever or methamphetamine dependence in the past 30 days. Methamphetamine abuse or dependence would include prescription methamphetamine if DSM-IV criteria were fulfilled. Recorded co-variables included age, ethnicity, race, smoking status (current/former/never), prior diagnoses of diabetes, hyperlipidaemia or hypertension, body mass index (BMI) >/≤25, hepatitis C status, history of cocaine abuse, alcohol abuse, estimated duration of HIV infection (years), HIV viral load (>/≤ 500 copies/mL), nadir CD4 cell count </≥ 200 cells/uL), current CD4 cell count (stratified <200/200–349/350–499/ ≥500) and total ART exposure (years). Death occurring after the last study visit was also recorded.

Statistical analysis was performed using Stata 12.0 (StataCorp LLC, College Station, TX, USA). Univariate logistic regression was performed with either heart failure or myocardial infarction as the outcome. Variables found to be associated with the outcome with a *P*-value < 0.15 were included in the multiple logistic regression model. Variables that were associated with heart failure or myocardial infarction were reported with adjusted odds ratio (aOR) with 95% confidence interval (CI). Variables with *P*-value < 0.05 were considered significant.

Results

Across the included studies there were 5148 participants: the mean age was 46 years, 51.6% were white, 27.3% were black, 17.2% were Hispanic and 3747 were PWH. The presence of HIV infection was associated with a history of myocardial infarction (aOR 2.7; 95% CI 1.26–5.7; $P = 0.01$) after correcting for age, gender, smoking status, hypertension, diabetes and hyperlipidaemia.

Demographics of the 3747 PWH included in the study are shown in Table 1. Among the included PWH, 15.3% had CD4 count < 200 cells/μL, 80.0% were on ART, 29.5% had viral load > 500 copies/mL, 20.6% were seropositive for hepatitis C virus (HCV) infection and alcohol dependence or abuse was recorded for 55%. A history of myocardial infarction was reported for 115 PWH (3.1%), a history of heart failure in 41 (1.1%), current or prior methamphetamine use in 1036 (27.9%), cocaine use in 1271 (34.2%) and methamphetamine/cocaine overlap in 503 (13.5%). A total of 487 PWH died following their last recorded visit (13.0%).

The results of the univariate and multiple logistic regressions for myocardial infarction are shown in Table 2. A history of methamphetamine use was not associated with myocardial infarction among PWH (OR 1.3; $P = 0.27$). Notably, older age (aOR 1.8 per decade; $P < 0.001$), hypertension (aOR 2.1; $P = < 0.01$), hyperlipidaemia (aOR 2.0; $P = 0.01$), prior or current smoking (aOR 2.0; $P = 0.03$), HIV RNA > 500 copies/mL (aOR 2.0; $P = 0.05$) and longer duration of HIV infection (aOR 1.04 per year; $P = 0.05$) were associated with a diagnosis of myocardial infarction. CD4 count, nadir CD4 count, duration of ART and

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diabetes demonstrated associations with myocardial infarction; however, these did not hold on multiple logistic regression.

Results of the regression for presence of heart failure are shown in Table 3. Use of methamphetamine was not associated with a diagnosis of heart failure (OR 1.07; 95% CI 0.54–2.11; $P = 0.84$). On multiple logistic regression, age (aOR 1.49 per decade; $P = 0.03$), hypertension (aOR 10.1; $P < 0.001$) and myocardial infarction (aOR 3.51; $P = 0.002$) were associated with heart failure.

Death occurring after last follow-up visit was not associated with methamphetamine use (aOR 0.97; 95% CI 0.71–1.3) or cocaine use (OR 0.97; $P = 75$). Lower CD4 count, HCV serology positivity, smoking history and presence of comorbidities (myocardial infarction, heart failure and diabetes) were associated with recorded death after the last study visit.

A post-hoc analysis was performed to investigate the effects of methamphetamine use among individuals under the age of 50 years. In univariate analysis, no association was found between heart failure and methamphetamine use; however, a borderline significant association was noted between methamphetamine use and myocardial infarction (OR 2.11; $P = 0.06$). In PWH, reporting of both methamphetamine and cocaine abuse was not found to be associated with myocardial infarction or heart failure ($P = 0.12$ and 0.27, respectively).

Discussion

This study provides the first evidence regarding methamphetamine use and cardiac disease among PWH. Interestingly, in this large cohort we did not find an association between methamphetamine use and cardiac outcomes but rather associations with traditional risk factors (age, smoking, hyperlipidaemia, hypertension and myocardial infarction). In addition to traditional risk factors, the presence and duration of HIV infection were associated with myocardial infarction. Among younger methamphetamine users, where the odds ratio for cardiac disease would be expected to be greatest, we found a borderline significant trend for an association between methamphetamine use and myocardial infarction ($P = 0.06$).

Our study highlights the role that traditional risk factors for heart disease play and emphasizes the importance of managing these risk factors with nonpharmacological and pharmacological therapies. In particular, hypertension demonstrated strong associations for both myocardial infarction (aOR 2.15) and heart failure (aOR 10.1). The presence of HIV infection and viraemia is associated with the presence of cardiac disease [4]; however, while suppressing viral replication is recommended, it remains uncertain whether this will directly improve cardiovascular risk. The Strategies to Manage Antiretroviral Therapy (SMART) study found that continuous ART reduced the risk of cardiovascular disease compared to interrupted treatment, yet the well designed early ART START trial failed to demonstrate a definite cardiovascular benefit of early ART at CD4 counts > 500 cells/μL [33,34]. It is interesting to note that, even after adjustment for age, duration of HIV infection was associated with myocardial infarction, suggesting that there is cumulative damage to the heart, perhaps from the known persistent inflammatory state associated with HIV infection [35–37].

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While methamphetamine use has gained acceptance as a significant cause of cardiac morbidity in the general population, we did not find an association with heart disease among PWH in our study. This may have been attributable to the relatively small number of events, which may be a reflection of the fact that this study population was not recruited directly for evaluating heart disease. Nevertheless, strengths of our study were the large number of participants and systematic recording of reported methamphetamine use in our study population. If a strong association was present, we would have expected to see some association.

Prior studies investigating cardiac disease among PWH have focused on the associations of myocardial infarction and heart failure with HIV-related parameters such as CD4 cell count and HIV viraemia but have been limited by the absence of data, or only limited data, on substance use [3,4]. Our data support prior findings regarding the elevated risk of myocardial infarction among PWH compared to people without HIV infection (aOR 2.7).

Our study had some limitations: it was retrospective in nature and thus causation could not be attributed; methamphetamine use and medical diagnoses were dependent largely on self-report, which may lead to overestimation or underestimation of effects; and a smaller than expected number of events, possibly as a consequence of incomplete ascertainment, reduced the power of this study. In addition, survival bias may have limited our results as high-risk methamphetamine users or individuals with advanced heart failure may not have been included. While the total duration of ART was available, use of particular antiretroviral drugs was not available. Strengths of our study include its large size, the systematic recording of substance use, a diverse racial/ethnicity profile and multi-centre recruitment.

In conclusion, our study found no strong association between methamphetamine use and myocardial infarction or heart failure among PWH. Traditional risk factors for atherosclerosis should be aggressively managed, including hypertension, hyperlipidaemia, smoking and diabetes, as well as ensuring viral suppression with ART.

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Table 1

Demographics of persons living with HIV (PWH) included in the study

Variable	PWH with methamphetamine use*	PWH without methamphetamine use*	Persons without HIV infection*
Number of participants	1036	2680	1401
Sex assigned at birth *[n* (%)]			
Male	945 (91.2)	2138 (79.8)	890 (64)
Female	91 (8.8)	542 (20.2)	511 (36.5)
Race/ethnicity *[n* (%)]			
Asian	10 (1.0)	20 (0.7)	26 (1.9)
Black	159 (15.3)	1015 (37.9)	219 (15.6)
Hispanic	167 (16.1)	468 (17.5)	246 (17.6)
Other	28 (2.7)	61 (2.2)	50 (3.6)
White	672 (64.9)	1115 (41.6)	860 (61.4)
Age (years) [mean (SD)]	45 (10)	47 (11)	44 (14)
Diabetes *[n* (%)]	85 (8.5)	335 (13.0)	101 (7.5)
Smoking [n/total (%)]			
Never	135/810 (16.7)	492/1875 (26.2)	194/703 (27.6)
Prior	378/810 (46.7)	883/1875 (47.1)	313/703 (44.5)
Current	297/810 (36.7)	500/1875 (26.7)	196/703 (27.9)
Hypertension *[n* (%)]	297 (28.7)	806 (30.1)	275 (19.6)
Hyperlipidaemia *[n* (%)]	212 (21.3)	607 (23.5)	194 (14.4)
CKD *[n* (%)]	10 (1.0)	46 (1.8)	0 (0)
BMI > 25 *[n* (%)]	544 (56.0)	1432 (56.2)	894 (67.5)
Alcohol use *[n* (%)]	711 (70.1)	1063 (48.1)	527/1035 (50.9)
History of cocaine use *[n* (%)]	503 (48.6)	768 (28.7)	204 (14.7)
Methamphetamine use *[n* (%)]	1036 (100)	0 (0.0)	352 (25.3)
CD4 count (cells/μL) (mean)	540	490	n/a
HIV VL (copies/mL) [median (IQR)]	30 (0–939)	30 (0–2280)	n/a
CD4:CD8 ratio [median (IQR)]	0.55 (0.34–0.88)	0.52 (0.28–0.83)	n/a
Nadir CD4 count < 200 cells/μL *[n* (%)]	517 (49.9)	1688 (63.0)	n/a
Duration of HIV infection (years) [median (IQR)]	12.4 (4.7–20.0)	13.1 (6.–19.7)	n/a

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Variable	PWH with methamphetamine use*	PWH without methamphetamine use*	Persons without HIV infection*
Duration of ART (years) [median (IQR)]	6.0 (1.6–12.2)	5.4 (1.4–11.3)	n/a
History of myocardial infarction *[n* (%)]	37 (3.6)	77 (2.9)	11 (0.8)
History of heart failure *[n* (%)]	12 (1.2)	29 (1.1)	12 (0.9)
Deceased *[n* (%)]	107 (10.3)	370 (13.8)	16 (1.1)

CKD, chronic kidney disease; BMI, body mass index; SD, standard deviation; IQR, interquartile range; ART, antiretroviral therapy; n/a, not applicable; VL, viral load.

*Denominators shown if ascertainment < 80%.

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Table 2

Univariate and multiple logistic regressions of factors associated with myocardial infarction

	Univariate logistic regression		Multiple logistic regression	
Variable	**OR (95% CI)**	***P*-value**	**aOR (95% CI)**	***P*-value**
Recorded methamphetamine abuse/dependence (long-term or current)	1.25 (0.84–1.87)	0.27		
History of cocaine abuse	1.22 (0.83–1.79)	0.32		
Age (decades)	2.32 (1.95–2.76)	< 0.001	**1.82 (1.39–2.39)**	**< 0.001**
Sex assigned at birth				
Male	1.00 ref			
Female	1.10 (0.68–1.77)	0.71		
Ethnicity/race				
Asian	nr	nr		
Black	0.78 (0.51–1.18)	0.24		
Hispanic	0.65 (0.37–1.15)	0.14	0.79 (0.37–1.67)	0.53
Other	0.93 (0.29–3.03)	0.91		
White	1.00 (ref)			
Diabetes	3.46 (2.28–5.24)	< 0.001	1.19 (0.69–2.06)	0.53
Hypertension	6.23 (4.14–9.40)	< 0.001	**2.15 (1.24–3.71)**	**< 0.01**
Hyperlipidaemia	5.29 (3.60–7.76)	< 0.001	**2.01 (1.16–3.49)**	**0.01**
CKD	1.09 (0.26–4.50)	0.91		
Smoking				
Never	1.00 (ref)			
Prior	1.99 (1.05–3.77)	0.04	**Combined**	**0.03**
Current	1.80 (0.90–3.58)	0.09	**1.99 (1.05–3.77)**	
BMI > 25	1.12 (0.76–1.66)	0.55		
Duration of HIV infection (years)	1.1 (1.07–1.12)	< 0.001	**1.04 (1.00–1.07)**	**0.05**
CD4 count	1.18 (0.99–1.40)	0.07	1.14 (0.89–1.46)	0.31
CD4:CD8 ratio	0.91 (0.62–1.34)	0.63		
Nadir CD4 count < 200 cells/μL	1.98 (1.30–3.01)	0.002	1.38 (0.80–2.39)	0.25
HIV VL > 500 copies/mL	0.60 (0.37–0.96)	0.03	**1.97 (1.00–3.89)**	**0.05**
HCV seropositive	0.95 (0.62–1.45)	0.81		

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	Univariate logistic regression		Multiple logistic regression	
Variable	**OR (95% CI)**	***P*-value**	**aOR (95% CI)**	***P*-value**
Duration of ART (years)	1.00 (1.00–1.01)	0.05	0.98 (0.94–1.02)	0.27

Bold indicates statistical significance.

aOR, adjusted odds ratio; CI, confidence interval; OR, odds ratio; nr, no result as a consequence of collinearity; CKD, chronic kidney disease; BMI, body mass index; VL, viral load; HCV, hepatitis C virus; ART, antiretroviral therapy.

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Table 3

Univariate and multiple logistic regression of factors associated with a history of systolic or diastolic heart failure

	Univariate logistic regression		Multiple logistic regression	
Variable	**OR (95% CI)**	***P*-value**	**aOR (95% CI)**	***P*-value**
Recorded methamphetamine abuse/dependence (long-term or current)	1.07 (0.54—2.11)	0.84		
History of cocaine abuse	1.11 (0.59–2.10)	0.75		
Age (decades)	2.25 (1.70–2.98)	< 0.001	**1.48 (1.04–2.13)**	**0.03**
Race/ethnicity				
Asian	nr	nr		
Black	1.81 (0.93–3.54)	0.08		
Hispanic	0.70 (0.23–2.10)	0.52		
Other	2.53 (0.57–11.1)	0.22		
White	1.00 (ref)			
Gender				
Male	1 (ref)			
Female	1.01 (0.44–2.28)	0.99		
Diabetes	4.03 (2.10–7.75)	< 0.001	1.53 (0.75–3.08)	0.24
Smoking history				
Never	1 (ref)			
Prior	1.91 (0.71–5.13)	0.20		
Current	0.63 (0.17–2.35)	0.49		
Hypertension	17.48 (6.84–44.67)	< 0.001	**10.1 (3.68–27.6)**	**< 0.001**
Hyperlipidaemia	3.63 (1.96–6.72)	< 0.001	0.80 (0.38–1.67)	0.55
History of myocardial infarction	9.55 (4.45–20.51)	< 0.001	**3.51 (1.56–7.90)**	**0.002**
History of alcohol use	1.13 (0.58–2.12)	0.71		
Nadir CD4 count < 200 cells/μL	0.97 (0.52–1.81)	0.92		
CD4 count	0.91 (0.70–1.18)	0.48		
CD4:CD8 ratio	0.73 (0.33–1.59)	0.29		
HIV VL > 500 copies/mL	0.76 (0.36–1.60)	0.47		
Duration of HIV infection (years)	1.07 (1.04–1.11)	< 0.001	1.00 (0.96–1.04)	0.97
ART exposure (years)	1.00 (1.00–1.00)	0.21		

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Variable	Univariate logistic regression		Multiple logistic regression	
	OR (95% CI)	***P*-value**	**aOR (95% CI)**	***P*-value**
HCV seropositive	1.37 (0.72–2.62)	0.34		

Bold indicates statistical significance.

OR, odds ratio; aOR, adjusted odds ratio; CI, confidence interval; nr, no result as a consequence of collinearity; VL, viral load; ART, antiretroviral therapy; HCV, hepatitis C virus.