Published in final edited form as:
J Acquir Immune Defic Syndr. 2011 December 15; 58(5): 458–462. doi:10.1097/QAI.0b013e31823801c4.

Control of Medical Comorbidities in Individuals with HIV

Anne K. Monroe, MD, MSPH[1], **Geetanjali Chander, MD, MPH**[1], and **Richard D. Moore, MD, MHS**[1]

[1]Division of General Internal Medicine, The Johns Hopkins University School of Medicine, Baltimore, Maryland

Abstract

Background—With improved combination antiretroviral therapy (ART)-related survival, diabetes and hypertension increasingly contribute to morbidity and mortality among individuals with HIV. However, there is limited data on diabetes and blood pressure control in this population. We examined whether virologic control is associated with control of diabetes and hypertension.

Methods—We examined HIV viral load, hemoglobin A1c (HbA1c), and blood pressure measurements from 70 diabetics and 291 hypertensives in the Johns Hopkins HIV Clinical Cohort, an urban, university-based cohort. All patients were treated for HIV and diabetes or hypertension. HbA1c and HIV-1 RNA were captured electronically from laboratory data, and blood pressure was collected electronically from vital signs taken at clinic visits. We used HIV-1 RNA values within 30 days of the HbA1c measurement or blood pressure measurement. The relationships between HIV-1 RNA and HbA1c and HIV-1 RNA and blood pressure were examined using separate random effects generalized least squares linear regression models.

Results—The study sample was predominantly male and black, with a high prevalence of comorbid hepatitis C virus infection and psychiatric illness. In multivariable analysis, each $\log_{10}$ increase in HIV-1 RNA was associated with higher HbA1c (β=0.47 units, $p<0.001$) among diabetics and higher mean arterial pressure (MAP) among hypertensive patients (β=1.95 mmHg, $p<0.001$).

Conclusions—Suboptimal control of HIV, indicated by detectable viral load, correlates with suboptimal control of diabetes and hypertension, indicated by higher HbA1c and MAP. Achieving control of multiple medical comorbidities and HIV simultaneously may require expansion of current adherence interventions focused primarily on antiretroviral therapy.

Keywords

HIV; Diabetes; Hypertension

Introduction

With the decrease in AIDS-related morbidity and mortality since the introduction of combination antiretroviral therapy (ART), HIV-infected individuals are experiencing complications from other comorbid conditions including diabetes and hypertension.[1–3] Both

Corresponding Author/Address for Reprints: Anne K. Monroe, MD, MSPH, The Johns Hopkins University School of Medicine, 2024 East Monument Street, Suite 2-600, Baltimore, MD 21205, 305-962-4679 (tel); 410-955-7733 (fax), amonroe4@jhmi.edu.

Conflicts of Interest: Drs. Monroe, Chander, and Moore have no conflicts of interest to disclose.

Parts of this data were presented in abstract from at the Society of General Internal Medicine 33rd Annual Meeting, April 28-May 1, 2010, Minneapolis, Minnesota

traditional diabetes risk factors and ART use contribute to diabetes prevalence among individuals with HIV.[4, 5] Indeed, prevalent and incident diabetes are more common among men with HIV on ART compared to men without HIV.[6] Hypertension rates are similar among individuals with HIV compared with the general population,[7] and duration of ART is associated with hypertension independent of age.[8] HIV, diabetes, and hypertension all require high levels of treatment adherence to achieve treatment goals and optimize clinical outcomes.[9–11]

Although the prevalence of diabetes and hypertension in HIV disease has been studied extensively, little data exists on diabetes and blood pressure control among individuals with HIV. Given the high level of adherence to HIV medications required to obtain viral suppression and the high level of adherence to diabetes and blood pressure medications required to control those conditions, it might be expected that individuals with better control of their HIV would also have better control of their diabetes or hypertension. However, it is unknown if control of viral load among individuals taking ART is related to control of other chronic conditions.

The current study examines the relationship between HIV control, indicated by undetectable viral load, and both diabetes control and blood pressure control. We hypothesized that poor virologic control would be associated with poor control of diabetes and hypertension.

Methods

We performed an analysis of data from the Johns Hopkins HIV Clinical Cohort (JHHCC), a clinic-based observational longitudinal cohort of patients who receive primary HIV care at Johns Hopkins.

Study Population

The JHHCC was initiated in 1989 to understand and quantify the processes and outcomes of care for HIV-infected patients in an urban, university-based clinical practice. Enrollment into the dynamic cohort occurs at a patient's first visit to the Hopkins ambulatory HIV clinic. The data set includes comprehensive demographic, clinical, laboratory, pharmaceutical, and psychosocial data. Data is abstracted from medical records, electronic laboratory sources, and pharmacy claims. The details of the study design and follow-up have been described previously.[12]

The study was approved by the Johns Hopkins Hospital Institutional Review Board and all participants signed informed consent.

Selection Criteria

Our sample included patients on antiretroviral therapy with a provider assigned diagnosis of diabetes or hypertension, who were concurrently receiving pharmacotherapy for either their diabetes or hypertension. Patients were identified through standardized manual chart abstraction. A total of 70 individuals with diabetes and 291 individuals with hypertension were identified. Twenty-three patients had both diabetes and hypertension and were included in both analyses.

Outcomes

HbA1c was captured electronically from laboratory data, and included measurements from August 1998 to November 2009. Blood pressure was abstracted from the medical record at clinic visits from March 2007 to December 2009. We converted the systolic and diastolic

blood pressure measurements to mean arterial pressure (MAP) using the formula (1/3 * SBP + 2/3 * DBP).

Measures of Interest

The primary independent variable was HIV-1 RNA, which was log-transformed because of non-normal distribution. This laboratory value was collected within 30 days of either the HbA1c measurement or blood pressure measurement. Demographic factors of interest included age, sex, race, and HIV transmission risk factor. Clinical variables extracted from the database included CD4 cell count (dichotomized to greater than or less than 200 cells/mL), type of antiretroviral therapy (protease inhibitor-containing combination ART vs. non-protease inhibitor containing ART), baseline substance abuse, hepatitis C status (presence of antibody to hepatitis C virus electronically captured through laboratory data) and presence of a psychiatric disorder. Baseline substance abuse was defined as self-reported alcohol use (daily or heavy), heroin use, cocaine use, or intravenous drug use (IVDU) as an HIV transmission risk factor. Presence of a psychiatric disorder was determined by manual abstraction of psychiatric and medical records by trained abstractors, and included diagnoses of major depression, depression with psychotic features, generalized anxiety disorder, mania, personality disorder, and schizophrenia. Finally, for the diabetic patients, we determined whether they were on insulin therapy (alone or with other agents) at the time of their first HbA1c measurement as a marker of disease severity.

Statistical Analysis

We generated summary statistics for demographic and clinical characteristics, considering the diabetic and hypertensive patients separately. We examined the relationship between HIV-1 RNA and both HbA1c and MAP using random effects generalized least squares linear regression to account for repeated measures from individual participants. HIV-1 RNA, HbA1c, and MAP were all examined as continuous variables. We performed both bivariate and multivariate analyses using separate models for the two outcomes.

The following variables were considered as potential confounders in the two multivariate analyses: age, sex, race, CD4 cell count, baseline substance abuse, and presence of a psychiatric disorder. We controlled for these variables and for additional variables (PI-containing ART and Hepatitis C status in the diabetes analysis) based on previous research. In the diabetes analysis, we adjusted for insulin use.

All analyses were performed using the STATA statistical package version 11.0 (College Station, Texas).

Results

Demographics

Table 1 presents the distribution of demographic and clinical characteristics among HIV-infected individuals with diabetes and hypertension in the cohort. Our study sample was predominantly male and black, and there was a high prevalence of IVDU as an HIV transmission risk factor, as well as comorbid HCV and psychiatric illness. Mean age (SD) was 44.7 years (9.9 years) for those diagnosed with diabetes and 45.3 years (9.3 years) for those diagnosed with hypertension. Mean (SD) baseline BMI was 25.8 kg/m^2 (6.9 kg/m^2) among those with diabetes with height and weight data available (N=42) and 26.2 kg/m^2 (6.0 kg/m^2) among those with hypertension with height and weight data available (N=211).

Baseline Data

Using the baseline data for diabetic patients, the median HbA1c was 7.3% (IQR = 6 to 9.8%). The range was 3.4 to 18.6%. The median $\log_{10}$ HIV-1 RNA was 2.1 (IQR = 1.7 to 3.3), corresponding to a median HIV-RNA of 126 copies/mL (IQR = 50 to 1995 copies/mL). Using the baseline data for hypertensive patients, the median MAP was 99.3 mmHg (IQR = 90.3 to 107.3 mmHg). The range was 66.0 to 134.7 mmHg. The median $\log_{10}$ HIV-1 RNA was 1.7 (IQR = 1.7 to 2.6), corresponding to a median HIV-RNA of 50 copies/mL (IQR = 50 to 398 copies/mL).

Relationship between Log Viral Load and Hemoglobin A1c

Our analysis of the relationship between log viral load and HbA1c included 420 HbA1c observations from 70 patients (mean number of HbA1c observations per patient = 6.0, range 1 to 28). An increase of one $\log_{10}$ in the HIV-1 RNA level was associated with higher HbA1c (β=0.43 units, $p<0.001$) in unadjusted analysis. Table 2 shows the association between increasing $\log_{10}$ HIV-1 RNA and higher HbA1c (β=0.47 units, $p<0.001$), which was preserved in multivariate analysis adjusted for sex, race, age, PI-containing ART, CD4 level, substance abuse, presence of a psychiatric disorder, hepatitis C status, and insulin use.

Relationship between Log Viral Load and Mean Arterial Pressure

Our analysis in those with hypertension included 1058 observations from 291 patients (mean number of blood pressure observations per patient = 3.6, range 1 to 13). An increase of one $\log_{10}$ in the HIV-1 RNA level was associated with increasing mean arterial pressure (MAP) (β=1.90 mmHg, $p<0.001$) in unadjusted analysis. Table 3 shows the association between increasing $\log_{10}$ HIV-1 RNA and higher MAP (β=1.95, $p<0.001$), which was preserved in multivariate analysis adjusted for sex, race age, CD4 level, substance abuse, and presence of a psychiatric disorder.

Discussion

In this study of patients with HIV and diabetes or hypertension, increasing HIV-1 RNA was associated with higher HbA1c and higher MAP. This is the first study to demonstrate that poor control of HIV-1 RNA is directly correlated with poor control of diabetes and hypertension, two comorbidities of increasing importance in the management of the patients with HIV infection. We suspect that adherence accounts for our findings, and that poor adherence to antiretroviral therapy correlates with poor adherence to therapy for other medical comorbidities, explaining the relationship between poor control of both conditions.

There are possible alternate explanations for the observed association. Uncontrolled HIV causes systemic inflammation,[13] and may contribute to both the development of hypertension and diabetes and to difficulty controlling the conditions. Therapeutic inertia, or failure to increase therapy despite unmet treatment goals, may also hinder control, [14] and may be more pronounced in patients with multiple chronic conditions, especially if they are unrelated chronic conditions.[15] Among individuals with HIV, especially those with poor control of their HIV or infectious complications related to immune compromise, less priority may be given to managing their diabetes and hypertension. We found only a weak association between CD4 count and HbA1c, and no association between CD4 count and MAP, suggesting that the level of immunosuppression does not play a significant role. Irrespective of the reason for poor control of both conditions, our study emphasizes the new challenges of multi-morbidity, and suggests that interventions targeting both patients and providers may be needed.

Three prior studies of diabetes control in HIV disease have been conducted. The most recent study by Satlin and colleagues revealed that about one-third of patients in the study sample did not meet American Diabetes Association (ADA) HbA1c goals and over half did not meet blood pressure goals. Similar proportions of patients with adequate and inadequate glycemic control had an undetectable viral load (p=0.21).[16] Another study involving 40 HIV-infected diabetics demonstrated that fewer than half of patients attained ADA hemoglobin A1c and blood pressure targets. Undetectable viral load was associated with not meeting LDL and total cholesterol goals.[17] A third study examined glycemic, lipid, and blood pressure control among 216 HIV-infected diabetic patients from an urban clinic,[18] finding that 54% of patients met the ADA HbA1c target and 65% reached the blood pressure target. The authors noted that an undetectable HIV viral load was associated with being less likely to meet total cholesterol and LDL cholesterol targets, which they attributed to antiretroviral-induced hyperlipidemia. There was no mention of an association between HbA1c at goal or blood pressure at goal and undetectable viral load. Our study expands on prior research by examining the relationship between HIV control and diabetes and hypertension control, with the ultimate goal of understanding how to optimize control of multiple conditions simultaneously.

The prevalence of diabetes in individuals with HIV has been reported as ranging from 2–12%, [6, 19] and diabetes has been associated with antiretroviral therapy, particularly with nucleoside reverse transcriptase inhibitors.[20, 21] The overall prevalence of hypertension among HIV-infected individuals was 31.7% (56.4% in men older than 50) in a large cohort study with HIV-uninfected controls,[8] with no difference in hypertension prevalence by HIV status. Both conditions contribute to cardiovascular and renal disease and may increase non-HIV related morbidity and mortality in this population.[22] Controlling multiple comorbidities is increasingly important as the population of individuals with HIV in the United States ages. In 2008, almost twenty percent of new HIV cases in the U.S. occurred in individuals aged 50 and older.[23] An aging population with HIV faces complications from both long-term metabolic toxicities of ART and common medical comorbidities whose prevalence increases with increasing age, such as diabetes and cardiovascular disease.[24] Mortality in older individuals with HIV is higher than in younger individuals with HIV,[25] the result of both infectious complications and medical comorbidities, and achieving good control of other medical comorbidities in addition to HIV is necessary to decrease mortality in older individuals.

The challenges of multi-morbidity are pronounced for individuals who receive HIV care, as competing priorities make adhering to complex medication regimens difficult. Up to 50% of patients (irrespective of comorbidity) do not take their medications as prescribed, resulting in significant morbidity and cost.[26, 27] Despite the importance of adherence in HIV clinical care,[13, 28] antiretroviral adherence is suboptimal: a meta-analysis of HIV treatment adherence interventions revealed baseline adherence rates of 55–95% among all participants.[29]

Similar to HIV, uncontrolled diabetes and hypertension are often caused by poor adherence[29] Only about twelve percent of diabetics meet ADA goals for lipid, blood pressure, and glycemic control simultaneously[30] and around 35% of hypertensives have controlled blood pressure.[31] Self-reported medication adherence is associated with lower HbA1c in diabetes,[32, 33] and high adherence to antihypertensive therapy has been associated with blood pressure control.[34] Sub-optimal adherence stems from a variety of factors which can be categorized as patient-level barriers, patient-provider barriers, and patient-system barriers.[27, 35] Patient-level barriers include low self-efficacy, low literacy, substance abuse and depression.[36] These are compounded by patient-provider and patient-system barriers

such as poor communication and misunderstanding of treatment instructions, poverty, poor education and housing, lack of insurance and medication costs.[37–39]

Our study has several potential limitations. Although HbA1c is used clinically in the management of HIV-infected diabetics, it underestimates glycemia.[40] This effect should be evenly distributed throughout all study participants, however, and should not affect the associations detected. Blood pressure measurements were part of the routine vital signs measurement done at clinic visits and were not standardized. In addition, we do not have a direct measure of adherence, such as patient self-report, electronic monitoring caps, pill counts, or pharmacy refill data.[41] Suppressed viral load is an accepted surrogate for treatment adherence, despite the fact that less than perfect adherence may also result in undetectable viral load.[36]

Our results demonstrate that poor HIV control is related to poor control of diabetes and hypertension, and we suspect that poor adherence to therapy for HIV is correlated with poor adherence to therapy for other conditions. Research on how patients prioritize medications for their comorbidities in relation to their HIV medications could shape future treatment adherence programs as our patient population ages and more individuals develop comorbid conditions. The most successful adherence programs combine several interventions such as incorporating behavioral interventions with education and increased convenience of care.[42] They are typically long-term, individually-tailored interventions focused on practical medication management skills.[29, 43] Our results argue that the scope of these programs should be expanded to include both antiretroviral agents and agents for other comorbidities.

Acknowledgments

Sources of Support: This work was supported by NIAAA K23 AA015313, NIH R01 DA11602, K24 DA00432, and R01 AA16893.

References

1. Sterne JAC, Hernán MA, Ledergerber B, et al. Long-term effectiveness of potent antiretroviral therapy in preventing AIDS and death: a prospective cohort study. Lancet. 2005; 366:378–384. [PubMed: 16054937]
2. Barbaro G. Highly Active Antiretroviral Therapy-Associated Metabolic Syndrome: Pathogenesis and Cardiovascular Risk. Am J Ther. 2006; 13:248–260. [PubMed: 16772767]
3. Friis-Møller N, Weber R, Reiss P, et al. Cardiovascular disease risk factors in HIV patients-association with antiretroviral therapy. Results from the DAD study. AIDS. 2003; 17:1179–1193. [PubMed: 12819520]
4. Butt A, Fultz S, Kwoh C, et al. Risk of diabetes in HIV infected veterans pre-and post-HAART and the role of HCV coinfection. Hepatology. 2004; 40:115–119. [PubMed: 15239093]
5. Tien PC, Schneider MF, Cole SR, et al. Antiretroviral therapy exposure and incidence of diabetes mellitus in the Women's Interagency HIV Study. AIDS. 2007; 21:1739–1745. [PubMed: 17690572]
6. Brown TT, Cole SR, Li X, et al. Antiretroviral therapy and the prevalence and incidence of diabetes mellitus in the multicenter AIDS cohort study. Arch Intern Med. 2005; 165:1179–1184. [PubMed: 15911733]
7. Bergersen BM, Sandvik L, Dunlop O, et al. Prevalence of hypertension in HIV-positive patients on highly active retroviral therapy (HAART) compared with HAART-naive and HIV-negative controls: results from a Norwegian study of 721 patients. Eur J Clin Microbiol Infect Dis. 2003; 22:731–736. [PubMed: 14610658]
8. Baekken M, Os I, Sandvik L, et al. Hypertension in an urban HIV-positive population compared with the general population: influence of combination antiretroviral therapy. J Hypertens. 2008; 26:2126–2133. [PubMed: 18854751]

9. Gæde P, Vedel P, Parving H, et al. Intensified multifactorial intervention in patients with type 2 diabetes mellitus and microalbuminuria: the Steno type 2 randomised study. Lancet. 1999; 353:617–622. [PubMed: 10030326]
10. Chobanian AV, Bakris GL, Black HR, et al. Seventh report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure. Hypertension. 2003; 42:1206–1252. [PubMed: 14656957]
11. Bangsberg D, Perry S, Charlebois E, et al. Non-adherence to highly active antiretroviral therapy predicts progression to AIDS. AIDS. 2001; 15:1181–1183. [PubMed: 11416722]
12. Moore RD. Understanding the clinical and economic outcomes of HIV therapy: the Johns Hopkins HIV clinical practice cohort. J Acquir Immune Defic Syndr Hum Retrovirol. 1998; 17 (Suppl 1):S38–S41. [PubMed: 9586651]
13. Volberding P, Deeks S. Antiretroviral therapy and management of HIV infection. Lancet. 2010; 376:49–62. [PubMed: 20609987]
14. Okonofua E, Simpson K, Jesri A, et al. Therapeutic inertia is an impediment to achieving the Healthy People 2010 blood pressure control goals. Hypertension. 2006; 47:345–351. [PubMed: 16432045]
15. Turner B, Hollenbeak C, Weiner M, et al. Effect of unrelated comorbid conditions on hypertension management. Ann Intern Med. 2008; 148:578–587. [PubMed: 18413619]
16. Satlin MJ, Hoover DR, Glesby MJ. Glycemic Control in HIV-Infected Patients with Diabetes Mellitus and Rates of Meeting American Diabetes Association Management Guidelines. Aids Patient Care ST. 2011; 25:5–12.
17. Bury JE, Stroup JS, Stephens JR, et al. Achieving American Diabetes Association goals in HIV-seropositive patients with diabetes mellitus. Proc (Bayl Univ Med Cent). 2007; 20:118–123. [PubMed: 17431444]
18. Adeyemi O, Vibhakar S, Max B. Are We Meeting the American Diabetes Association Goals for HIV-Infected Patients with Diabetes Mellitus? Clin Infect Dis. 2009; 49:799–802. [PubMed: 19635024]
19. Adeyemi O. Cardiovascular risk and risk management in HIV-infected patients. Top HIV Med. 2007; 15:159–162. [PubMed: 18073451]
20. Brown TT, Li X, Cole SR, et al. Cumulative exposure to nucleoside analogue reverse transcriptase inhibitors is associated with insulin resistance markers in the Multicenter AIDS Cohort Study. AIDS. 2005; 19:1375–1383. [PubMed: 16103768]
21. De Wit S, Sabin CA, Weber R, et al. Incidence and Risk Factors for New-Onset Diabetes in HIV-Infected Patients. Diabetes Care. 2008; 31:1224–1229. [PubMed: 18268071]
22. Neuhaus J, Angus B, Kowalska J, et al. Risk of all-cause mortality associated with nonfatal AIDS and serious non-AIDS events among adults infected with HIV. AIDS. 2010; 24:697–706. [PubMed: 20177360]
23. Centers for Disease Control and Prevention. Diagnoses of HIV Infection and AIDS in the United States and Dependent Areas, 2008. Atlanta, GA: Centers for Disease Control and Prevention, US Department of Health and Human Services; 2009.
24. Gebo K, Justice A. HIV infection in the elderly. Curr Infect Dis Rep. 2009; 11:246–254. [PubMed: 19366568]
25. Greenbaum A, Wilson L, Keruly J, et al. Effect of age and HAART regimen on clinical response in an urban cohort of HIV-infected individuals. AIDS. 2008; 22:2331–2339. [PubMed: 18981772]
26. DiMatteo MR. Variations in patients' adherence to medical recommendations: a quantitative review of 50 years of research. Med Care. 2004; 42:200–209. [PubMed: 15076819]
27. Osterberg L, Blaschke T. Adherence to medication. N Engl J Med. 2005; 353:487–497. [PubMed: 16079372]
28. Paterson D, Swindells S, Mohr J, et al. Adherence to protease inhibitor therapy and outcomes in patients with HIV infection. Ann Intern Med. 2000; 133:21–30. [PubMed: 10877736]
29. Rueda S, Park-Wyllie L, Bayoumi A, et al. Patient support and education for promoting adherence to highly active antiretroviral therapy for HIV/AIDS. Cochrane Database Syst Rev. 2006; 3:CD001442. [PubMed: 16855968]

30. Cheung B, Ong K, Cherny S, et al. Diabetes prevalence and therapeutic target achievement in the United States, 1999 to 2006. Am J Med. 2009; 122:443–453. [PubMed: 19375554]
31. Caro JJ, Salas M, Speckman JL, et al. Persistence with treatment for hypertension in actual practice. Can Med Assoc J. 1999; 160:31–37. [PubMed: 9934341]
32. Rozenfeld Y, Hunt J, Plauschinat C, et al. Oral antidiabetic medication adherence and glycemic control in managed care. Am J Manag Care. 2008; 14:71–75. [PubMed: 18269302]
33. Krapek K, King K, Warren S, et al. Medication adherence and associated hemoglobin A1c in type 2 diabetes. Ann Pharmacother. 2004; 38:1357–1362. [PubMed: 15238621]
34. Bramley T, Gerbino P, Nightengale B, et al. Relationship of blood pressure control to adherence with antihypertensive monotherapy in 13 managed care organizations. J Manag Care Pharm. 2006; 12:239–245. [PubMed: 16623608]
35. Haynes R, McDonald H, Garg A. Helping patients follow prescribed treatment: clinical applications. JAMA. 2002; 288:2880–2883. [PubMed: 12472330]
36. Wagner JH, Justice AC, Chesney M, et al. Patient-and provider-reported adherence toward a clinically useful approach to measuring antiretroviral adherence. J Clin Epidemiol. 2001; 54:91–98.
37. Hill-Briggs F, Gary T, Hill M, et al. Health-related quality of life in urban African Americans with type 2 diabetes. J Gen Intern Med. 2002; 17:412–419. [PubMed: 12133154]
38. Hill-Briggs F, Gary T, Bone L, et al. Medication adherence and diabetes control in urban African Americans with type 2 diabetes. Health Psychol. 2005; 24:349–357. [PubMed: 16045370]
39. Rubin R. Adherence to pharmacologic therapy in patients with type 2 diabetes mellitus. Am J Med. 2005; 118(Suppl 5A):27S–34S. [PubMed: 15850551]
40. Kim PS, Woods C, Georgoff P, et al. Hemoglobin A1c Underestimates Glycemia in HIV Infection. Diabetes Care. 2009
41. Liu H, Golin C, Miller L, et al. A comparison study of multiple measures of adherence to HIV protease inhibitors. Ann Intern Med. 2001; 134:968–977. [PubMed: 11352698]
42. Haynes R, Yao X, Degani A, et al. Interventions for enhancing medication adherence. Cochrane Database Syst Rev. 2005; 2:CD000011.
43. Taiwo B. Adherence to antiretroviral therapy: the more you look, the more you see. Curr Opin HIV AIDS. 2009; 4:488–492. [PubMed: 20048715]

Table 1

Demographic characteristics of individuals with treated diabetes and hypertension in the JHHCC

Characteristic	Diabetes (N=70) N (%)	Hypertension (N=291) N (%)
Age (years)		
Mean (SD)	44.7 (9.9)	45.3 (9.3)
Sex		
Male	44 (62.9)	190 (65.3)
Race		
Black	62 (88.6)	258 (88.7)
White	5 (7.1)	29 (10.0)
Hispanic	2 (2.9)	3 (1.0)
Other	1 (1.4)	1 (0.3)
Hepatitis C Virus infection		
Yes	43 (61.4)	154 (52.9)
HIV risk factor: Homosexual/Bisexual intercourse		
Yes	10 (14.3)	50 (17.2)
HIV risk factor: Heterosexual intercourse		
Yes	39 (55.7)	165 (56.7)
HIV risk factor: Injection drug use (IDU)		
Yes	28 (43.1)	127 (43.6)
Enrollment CD4 (cells/μL)		
Median (IQR)	256 (105–432)	256 (95–486)
Presence of a psychiatric diagnosis		
Yes	25 (35.7)	119 (40.9)

Table 2

Multivariate Analysis of Log Viral Load and Hemoglobin A1c (HbA1c) among individuals with diabetes in the JHHCC (N=70)

Independent Variable	β (SE)	P value
Log_{10} HIV-1 RNA	0.47* (0.10)	<0.001
Sex (male v. female)	−0.51 (0.46)	0.27
Race (white v. non-white)	−0.97 (0.88)	0.27
Age	0.00 (0.02)	0.92
ART with protease inhibitor	0.39 (0.46)	0.40
CD4 ≥ 200	0.66 (0.27)	0.02
Hepatitis C virus infection	0.00 (0.53)	1.00
Psychiatric disorder	0.17 (0.49)	0.72
Substance abuse	−0.16 (0.54)	0.77
Insulin use	0.44 (0.48)	0.37

* For each unit increase in log viral load, average increase in HbA1c was 0.47 units

Table 3

Multivariate Analysis of Log Viral Load and Mean Arterial Pressure (MAP) among individuals with hypertension in the JHHCC (N=291)

Independent Variable	β (SE)	P value
Log_{10} HIV-1 RNA	1.95* (0.44)	<0.001
Sex (male v. female)	0.36 (1.30)	0.78
Race (white v. non-white)	0.24 (2.04)	0.91
Age	−0.05 (0.06)	0.43
CD4 ≥ 200	0.21 (1.22)	0.86
Psychiatric disorder	−1.21 (1.23)	0.33
Substance abuse	−0.73 (1.32)	0.58

*For each unit increase in log viral load, average increase in mean arterial pressure was 1.95 mmHg