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Vitamin D Deficiency is Associated with Type 2 Diabetes Mellitus in HIV Infection

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Abstract

Background—Metabolic complications, including type 2 diabetes mellitus (DM) and metabolic syndrome, are increasingly recognized among HIV-infected individuals. Low vitamin D levels increase the risk of type 2 DM, and vitamin D supplementation has been shown to decrease the risk of type 2 DM in patients without HIV infection.

Objectives—The primary objective was to determine whether vitamin D deficiency (serum 25-hydroxyvitamin D <20 ng/mL) was associated with type 2 DM among HIV-infected patients. Our secondary objective was to determine whether vitamin D deficiency was associated with metabolic syndrome in HIV.

Methods—We conducted a cross-sectional study among subjects enrolled in the prospective Modena (Italy) HIV Metabolic Clinic Cohort. Clinical and laboratory data, including history of type 2 DM, fasting blood glucose, components of metabolic syndrome, and 25-hydroxyvitamin D levels, were obtained for all subjects.

Results—After adjusting for vitamin D supplementation, sex, age, body mass index, and hepatitis C virus co-infection, vitamin D deficiency was associated with type 2 DM (adjusted OR, 1.85; 95% CI, 1.03–3.32; $p=.038$). The association between vitamin D deficiency and metabolic syndrome was not significant after adjusting for vitamin D supplementation, sex age and body mass index (adjusted OR 1.32; 95% CI, 1.00–1.75; $p=.053$).

Conclusions—Our study demonstrates an association between vitamin D deficiency and type 2 DM. Clinical trials are needed to better characterize the association between vitamin D deficiency

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and type 2 DM in HIV infection and to evaluate whether vitamin D is able to prevent or delay the onset of type 2 DM.

Keywords

vitamin D deficiency; type 2 DM; insulin resistance; metabolic syndrome

INTRODUCTION

Patients infected with HIV are surviving longer because of antiretroviral therapy (ART). Consequently, there has been an increased awareness of adverse events related to ART, particularly metabolic complications such as insulin resistance [1,2]. The etiology of insulin resistance includes patient-level factors, HIV infection, certain protease inhibitors [3], and thymidine analogues [4]. Insulin resistance *contributes to* the development of type 2 diabetes mellitus (DM) and cardiovascular disease among HIV-infected patients.

Deficiency of vitamin D, the major steroid hormone involved in calcium homeostasis and bone metabolism, has been associated with impaired β -cell function and insulin resistance in humans [5, 6]. In HIV-uninfected patients, vitamin D deficiency (25-hydroxyvitamin D <20 ng/mL) is a risk factor for both type 2 DM and metabolic syndrome [7–9]. However, in the setting of HIV infection, the clinical consequences of vitamin D deficiency, which has been reported to have a prevalence ranging from 12 to 86% [10–16], remain unclear. In particular, the association between vitamin D deficiency and type 2 DM and metabolic syndrome among HIV-infected patients is not known.

Our primary objective was to determine whether a low serum concentration of 25-hydroxyvitamin D was associated with type 2 DM among HIV-infected patients. We hypothesized that a low 25-hydroxyvitamin D level was an independent risk factor for type 2 DM in HIV. Our secondary objective was to determine if vitamin D deficiency among HIV-infected patients was associated with the metabolic syndrome, for which the underlying mechanism is considered to be insulin resistance. We hypothesized that individuals with HIV and low serum concentrations of 25-hydroxyvitamin D would have metabolic syndrome *which has been linked to* higher cardiovascular risk, in comparison to individuals with higher serum concentrations of 25 hydroxyvitamin D levels. Confirmation of these associations would identify hypovitaminosis D as a potentially modifiable cardiovascular risk factor among HIV-infected patients.

METHODS

Study Design and Participants

We performed a cross-sectional study among subjects enrolled in the Modena HIV Metabolic Clinic Cohort, a prospective cohort study initiated in September 2004 to evaluate metabolic alterations among HIV-infected patients followed at the Metabolic Clinic of the University of Modena and Reggio Emilia School of Medicine (Modena, Italy) [17]. *HIV infected patients with metabolic complications are referred to this clinic for further evaluation.* Subjects have laboratory-confirmed HIV, provide informed consent, and complete a standardized questionnaire that collects demographic, medical, and HIV data at enrollment. Laboratory evaluation at enrollment includes blood for fasting lipids, fasting glucose, 25-hydroxyvitamin D, HIV RNA, immune function, and hepatitis status.

All subjects enrolled in the cohort between January 1, 2005 and January 31, 2008 who had documented 25-hydroxyvitamin D level were eligible for inclusion. For our primary objective, we included subjects with a 25-hydroxyvitamin D level and either fasting serum

glucose level or documented type 2 DM diagnosis *and use of insulin or oral antidiabetic medication*. For our secondary objective, we included subjects with a 25-hydroxyvitamin D level and presence or absence of the metabolic syndrome diagnosis. This study was reviewed and approved by the Institutional Review Boards of the University of Modena and University of Pennsylvania.

Main Study Outcomes

The primary outcome was type 2 DM, defined as a fasting glucose ≥ 126 mg/dL [18], a physician's diagnosis of type 2 DM, and/or receipt of an oral anti-diabetic agent or insulin.

As a secondary outcome, we evaluated the metabolic syndrome, defined by the presence of any 3 of the following 5 characteristics according to the National Cholesterol Education Program's Adult Treatment Panel III report: abdominal obesity based on waist circumference (>102 cm in men and >88 cm in women), triglycerides ≥ 150 mg/dL, high density lipoprotein (HDL) <40 mg/dL for men and <50 mg/dL for women, blood pressure $\geq 130/85$ mm Hg, and fasting glucose ≥ 110 mg/dL [19].

Data Collection

Demographic (age; sex), clinical (height; body weight; blood pressure; history of type 2 DM [as defined above]), and HIV data (duration of HIV diagnosis; antiretroviral use and duration) were recorded at subject enrollment.

Blood was drawn from all subjects after at least 8 hours of fasting for determination of HIV RNA (Abbott RealTime HIV-1 assay; Abbott Laboratories; lower limit of detection: 50 copies/mL); CD4 T lymphocyte count; hepatitis C virus (HCV) antibody (anti-HCV; Abbott HCV EIA 3.0 enzyme immunoassay; Abbott Laboratories); glucose (Rapidpoint 400 analyzer; Bayer); total cholesterol (Cholesterol/HP; Roche); triglyceride (Triglycerides/GPO; Roche); HDL (HDL-C plus 3rd generation; Roche); and 25-hydroxyvitamin D (DiaSorin 25-hydroxyvitamin D chemiluminescence immunoassay; Stillwater, MN). Vitamin D deficiency was defined as a serum 25-hydroxyvitamin D level <20 ng/mL (50 nmol per liter). Vitamin D insufficiency was defined as 25-hydroxyvitamin D ≥ 30 ng/mL and <20 ng/mL. [20]

Statistical Analysis

Differences between groups were assessed using Student's t-tests for continuous variables and chi-square tests for categorical variables. Multivariable logistic regression determined unadjusted and adjusted odds ratios (ORs) and 95% confidence intervals (CIs) of type 2 DM and metabolic syndrome between subjects with and without low vitamin D. *Potential confounding variables evaluated* included age, sex, body mass index, vitamin D supplementation, *season of vitamin D measurement*, thymidine analogues, specific protease inhibitors such as lopinavir, and HCV co-infection.

Backward elimination procedures removed non-significant potential confounders from adjusted models. All analyses were performed using STATA 10.0 (College Station, TX).

RESULTS

Vitamin D Status

The mean serum concentration of 25-hydroxyvitamin D was 19 ng/mL (95% Confidence Interval [CI]=18–20 ng/mL). A total of 64% were vitamin D deficient (25-hydroxyvitamin D <20 ng/mL), and an additional 20% of the subjects had vitamin D insufficiency. Among

1405 subjects with available information regarding supplementation, 93 (7%) were taking a vitamin D supplement (7500 IUs vitamin D3 weekly).

Association Between 25(OH) Vit D Levels and Type 2 DM

Mean 25-hydroxyvitamin D levels among type 2 DM subjects were lower than in subjects without type 2 DM (Table 1). After adjusting for vitamin D supplementation, sex, age, body mass index and HCV co-infection, vitamin D deficiency was associated with type 2 DM (adjusted OR, 1.85; 95% CI, 1.03–3.32; $p=.038$) (Table 2).

Association Between 25-hydroxyvitamin D Levels and Metabolic Syndrome

Overall among 1687 subjects, 412 (24%) met the definition of metabolic syndrome. Mean 25-hydroxyvitamin D levels among subjects with metabolic syndrome were lower than in subjects without metabolic syndrome (17 ± 12 versus 20 ± 10 ng/mL; $p<.001$). The association between vitamin D deficiency was not significant after adjusting for age, sex, body mass index, and vitamin D supplementation (Table 2).

DISCUSSION

Our study identified a high prevalence of vitamin D deficiency among HIV-infected patients. Patients with type 2 DM in this study had lower 25-hydroxyvitamin D levels compared with subjects without type 2 DM, although both groups met criteria for vitamin D deficiency. We also demonstrated that vitamin D deficiency was independently associated with type 2 DM in this population. However, we did not find an association between vitamin D deficiency and metabolic syndrome.

The prevalence of vitamin D deficiency among the HIV-infected patients in this sample was similar to that reported by Garcia-Aperico et al [11] among 30 HIV-infected males in Spain (86%). The prevalence of vitamin D deficiency in our population was higher than in several previous studies, which reported prevalences of 12% to 47% [12–16]. In contrast, a large Italian study of more than 6000 outpatients and inpatients reported a 52% prevalence of vitamin D deficiency [21]. The higher prevalence of vitamin D deficiency in this study may be related to the northern geographic location of Modena (44°N) which may lead to more indoor activities and less sun exposure.

Several cross-sectional studies have shown that low serum 25-hydroxyvitamin D concentration is associated with impaired glucose tolerance in HIV-uninfected patients [7,8]. In the Nurses' Health Study, Pittas et al [8] demonstrated an inverse association between vitamin D supplementation and the development of type 2 DM. A combined daily intake of at least 1,200 mg of calcium and 800 IUs of vitamin D was associated with a 33% lower risk of type 2 DM. The findings of our study are consistent with those of HIV-uninfected individuals and suggest that the association between vitamin D deficiency and type 2 DM is also present among HIV-infected individuals.

In our study, subjects with metabolic syndrome had lower serum 25-hydroxyvitamin D levels. However, we did not find an association between vitamin D deficiency and metabolic syndrome.

The role of HIV and ART in vitamin D deficiency is complex. HIV might affect α -hydroxylation of 25-hydroxyvitamin D in the kidneys to its active form 1, 25-dihydroxyvitamin D [22]. Protease inhibitors and non-nucleoside reverse transcriptase inhibitors utilize the cytochrome p450 enzyme, which is also the site of hydroxylation of vitamin D. Thus, certain antiretrovirals may also predispose to vitamin D deficiency [23]. If individuals with HIV are at a higher risk of developing vitamin D deficiency due to HIV and

ART, then the consequences of vitamin D deficiency, such as type 2 DM, may be more prevalent among HIV-infected individuals, as shown by our results.

Additionally, inflammation has been shown to play a key role in the development of type 2 DM and metabolic syndrome. Vitamin D has anti-inflammatory effects among HIV-uninfected individuals, which may explain its protective role in type 2 DM *and cardiovascular disease* [24,25]. Vitamin D may lower the risk of type 2 DM and metabolic syndrome in HIV by reducing the heightened inflammation associated with HIV infection . [26]

Our study had several limitations. First, since we used a cross-sectional design, we cannot establish whether the relationship between vitamin D deficiency and type 2 DM is causal. *A small prospective study by van Den Bout et al evaluated the impact of vitamin D supplementation on insulin sensitivity in 20 HIV infected subjects. At 24 weeks insulin sensitivity was decreased but by 48 weeks insulin sensitivity returned to baseline* [27]. *Further larger prospective studies are needed to evaluate whether* vitamin D deficiency may be a modifiable risk factor for the development of type 2 DM.

Second, our definition of type 2 DM may have resulted in outcome misclassification. Patients with type 2 DM with well controlled fasting blood glucose levels who failed to report anti-diabetic medication use may have been misclassified as not having type 2 DM. However, such outcome misclassification would have caused us to underestimate the association between vitamin D deficiency and type 2 DM.

Additionally, it is also possible that vitamin D deficiency is associated with insulin resistance, even among those not meeting criteria for type 2 DM. Patients with more modest degrees of insulin resistance were classified as not having type 2 DM in this study. Such misclassification would bias our results towards the null. This limitation would cause us to underestimate the association between vitamin D deficiency and insulin resistance.

Lastly, subjects were from Italy, a homogeneous population, potentially limiting the generalizability of our results.

In summary, we demonstrated that vitamin D deficiency was independently associated with type 2 DM in HIV. Future studies should examine whether vitamin D supplementation can prevent or treat type 2 DM in HIV and possibly reduce complications associated with HIV infection and its treatment.

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Table 1

Demographic and clinical characteristics of subjects by type 2 DM and by Metabolic Syndrome (MS)

Characteristic*	Overall n=1811	Type 2 DM n=116	No Type 2 DM n=1653	MS n=412	No MS n=1275	p value
Age	44±7	50±8	44±7	47±7	44±7	<.001
Gender (%male)	1131(62%)	88(76%)	1021(60%)	286(70%)	778(61%)	.002
25(OH)VitD, ng/ml ^a	19±12	15±9	19±12	17±10	20±12	<.001
Vitamin D supplement(%)(n=1405) ^a (n=1313 for MS)	93(7%)	8(8%)	82(6%)	18(5%)	66(7%)	.392
BMI, kg/m ^{2,ab}	23±4	25±5	23±4	25±5	23±4	<.001
HDL, mg/dL ^{ab}	45±15	39±12	45±16	35±9	48±16	<.001
Triglycerides ^a , mg/dL	200±168	264±191	195±165	295±198	170±147	<.001
Systolic BP ^{ab} , mm Hg	120±15	128±16	120±15	130±14	117±15	<.001
HCV co-infection (n=1357) ^{ab}	598(44%)	0(46%)	552(44%)	123(40%)	441(46%)	.049
AST/ALT ratio ^a (n=1588) (n=1485 for MS)	.924±.359	.877±.336	.926±.358	.891±.356	.933±.354	.048
On HAART containing: Nevirapine	666(37%)	38(33%)	616(37%)	148(36%)	472(37%)	.688
Efavirenz	760(42%)	48(45%)	702(42%)	191(46%)	522(41%)	.053
Median nadir CD4 count (cells/uL) (n=1744) ^a (n=1638 for MS)	111(0–986)	84(1–400)	112(0–986)	103(1–870)	114(0–986)	.159
Current median CD4, cells/uL (n=1633) ^{ab}	470(3–1865)	388(11–1400)	478(3–1865)	468(11–1618)	466(3–1865)	.920
HIV VL (%undetectable)	1021(56%)	70(60%)	941(57%)	221(54%)	751(59%)	.06

* Mean±sd,n(%) or median(range)

^a Less than 5% missing among subjects by type 2 DM^b Less than 5% missing among subjects by Metabolic Syndrome

Abbreviations: 25(OH) Vit D, 25-hydroxyvitamin D; BMI, Body Mass Index; HDL, High Density Lipoprotein; BP, Blood Pressure; HCV, Hepatitis C Virus; HAART, Highly Active Antiretroviral Therapy; VL, Viral Load

Table 2

Factors associated with type 2 DM and Metabolic Syndrome

Characteristics	Adjusted Odds Ratio* (95% Confidence Interval)	P Value	Adjusted Odds Ratio* (95% Confidence Interval)		P Value
			Type 2 DM	Metabolic Syndrome	
25-hydroxyvitamin D <20 ng/mL	1.85 (1.03–3.32)	.038		1.32(1.00–1.75)	.053
Vitamin D Supplementation	.87(.35–2.18)	.767		.76(.42–1.36)	.352
Gender (male)	1.57(.89–2.82)	.128		1.05(.79–1.40)	.722
Age, per year	1.08(1.05–1.11)	<.01		1.05(1.03–1.06)	<.01
BMI kg/m², per 1 U	1.09(1.03–1.14)	<.01		1.15(1.11–1.19)	<.01
Hepatitis C Virus coinfection[†]	1.42(.86–2.36)	.171			

Abbreviations: BMI, Body Mass Index

* Model adjusted for vitamin D supplementation, gender, age, body mass index,

[†] Type 2 DM model adjusted for hepatitis C virus co-infection