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Association Between Diabetes Mellitus and Left Ventricular Hypertrophy in a Multi-Ethnic Population

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Abstract

It is still controversial whether type 2 diabetes mellitus (T2DM) is associated with increased left ventricular (LV) mass independent of body size. We tested the hypothesis that T2DM is independently associated with LV mass in a multi-ethnic cohort. In the Northern Manhattan Study (NOMAS) cohort sample, a total of 1932 subjects (67.9±9.6 years; 769 men and 1163 women; 443 DM and 1489 non-DM) were studied by transthoracic echocardiography, and LV mass was calculated. LV hypertrophy was defined as the upper quartile of LV mass. Multivariable models were used to assess the association of T2DM with LV mass after adjusting for age, sex, race, body mass index (BMI), systolic BP, education, history of coronary artery disease (CAD), physical activity and alcohol consumption. LV mass (189±60 vs. 174±59g; $p<0.0001$), BMI, and systolic BP were higher in the DM group than in the non-DM group, while age and sex distribution were similar between the groups. In multivariable analysis, T2DM was independently associated with increased LV mass ($P=0.03$). The presence of T2DM was associated with increased risk of LV hypertrophy (adjusted odds ratio 1.46; 95%CI, 1.13–1.88, $P=0.004$). Although no interactions were observed between T2DM and BMI on LV hypertrophy ($p=0.6$), there was a significant interaction between T2DM and waist circumference on LV hypertrophy ($P=0.01$). In conclusion, T2DM was independently associated with increased LV hypertrophy independent of various covariates in this multi-ethnic sample. The presence of T2DM increased the risk of LV hypertrophy by about 1.5-fold, and it possibly interacted with central obesity.

Keywords

Type 2 diabetes; left ventricular mass; multi-ethnic; central obesity

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Introduction

Insulin resistance, which frequently accompanies type 2 diabetes mellitus (DM), is reported to be associated with left ventricular (LV) hypertrophy.^{1,2} Because type 2 DM and insulin resistance are closely associated with obesity, the association of type 2 DM and insulin resistance with LV hypertrophy may disappear after adjusting for body mass³ or fat mass.⁴ There have been reports suggesting that the relationship between type 2 DM and LV hypertrophy is due to the interactions of DM with age and obesity.⁵ In pre-diabetic and diabetic elderly subjects, fat mass was a major determinant of LV mass in women.⁶ Therefore, the reported relationship between type 2 DM and LV hypertrophy could have been an epiphenomenon of the relationship between body mass (fat) and LV hypertrophy rather than the effect of DM per se. However, few studies have shown that DM is associated with increased LV mass independent of other risk factors in the general population, especially in multiethnic cohorts. Furthermore, the strength of association between DM and LV hypertrophy has not been evaluated, nor has its possible interaction with patient characteristics, especially body habitus. Thus, we performed this study to test the hypothesis that the presence of type 2 DM per se is independently associated with LV hypertrophy independent of other factors especially body fat, and that its effect has some interaction with other factors such as age, race/ethnicity, and obesity.

Methods

Subjects were participants in the Northern Manhattan Study (NOMAS), a population-based prospective cohort study designed to investigate cardiovascular and stroke incidence, risk factors, and prognosis in a multiethnic sample from northern Manhattan. The methods of subject recruitment and enrollment into NOMAS have been described elsewhere.⁷ Briefly, random digit dialing of approximately 25 000 households was performed and community participants were enrolled in NOMAS if they: (1) had never had a stroke diagnosed; (2) were older than age 40; and (3) resided in Northern Manhattan for 3 months in a household with a telephone. Ninety-one percent of those called participated in a telephone interview, and 75% of those who were eligible and invited to participate came to Columbia University Medical Center for an in-person evaluation (overall participation rate 68%). The study was approved by the Institutional Review Board at Columbia University Medical Center. All participants gave consent directly or through a surrogate when appropriate. As part of NOMAS, 3298 participants underwent extensive in-person evaluation, and transthoracic echocardiograms were performed on 2003 eligible subjects between 1993 and 1998. Echocardiograms that were technically adequate for analysis were obtained in 1932 subjects and are included in this study.

Blood pressure (BP) was measured with mercury sphygmomanometers and cuffs of appropriate size. Hypertension was defined as a BP recording 140/90 mm Hg (based on the average of 2 BP measurements during one sitting by a trained research assistant), the patients' self-report of a history of hypertension, or antihypertensive medication use. Type 2 DM was defined by the patients' self-report of such a history, use of insulin or hypoglycemic agent, or fasting glucose ≥ 126 mg/dl, and type 1 DM was not included in this study. Coronary artery disease (CAD) was defined as having either a history of bypass surgery, angioplasty, or myocardial infarction. Physical activity was assessed with a standardized questionnaire that recorded the frequency and duration of 14 different recreational activities during the 2-week period before the interview. These analyses used the total duration of physical activity in hours per week. Height and weight were determined by the use of calibrated scales. Moderate alcohol use was defined as current drinking of more than one drink per month and less than or equal to 2 drinks per day. Assessments were conducted in English or Spanish, depending on the primary language of the participant. Race—ethnicity was based on self-identification through a series

of interview questions modeled after the 2000 US census and conformed to the standard definitions outlined by Directive 15.

Transthoracic echocardiography was performed and measurements were taken by standard two-dimensional protocols according to the guidelines of the American Society of Echocardiography. LV diastolic dimension, LV systolic dimension, ventricular septal thickness (VS), and posterior wall thickness (PWT) were measured in all patients. LV mass was calculated with the use of the corrected ASE method: $0.8 \times (1.04 \times [(VS + LV \text{ diastolic dimension} + PWT)^3 - (LV \text{ diastolic dimension})^3] + 0.6)$.⁸

We used LV mass itself to avoid a bias of partition values in particular race/ethnic group,⁹ but we adjusted body mass index (BMI) in multivariable analyses. Interpretation of echocardiographic studies was performed off-line by researchers blinded to the subject's clinical and demographic characteristics. Four readers over the period of 1993 to 2000 were involved in the analysis of all the echocardiographic studies. For quality-control measures, all readers were trained by senior echocardiographers (S.H. or M.D.T.) and interobserver variability for the variables ranged between 8% and 10%. LV hypertrophy was defined as the 75th percentile of LV mass (207.1 g), which was calculated from overall population. The gender-specific 75th percentile cutoffs (female, 187.5g; male, 230.2g) were also used as in a sub-analysis.

Educational level was used as an indicator of socioeconomic status (SES) and classified into 2 categories: "less than high school" included those who never went to high school or had completed only part of high school, "completed high school" included those who had completed high school or other vocational training beyond primary.

The data are expressed as the mean ($\pm$ SD) or percentage. The chi-square test was used to compare proportions. Unpaired t-test was performed to test mean differences between groups. The prevalence of diabetes and the distribution of LV mass were evaluated in the total cohort and among the 3 race-ethnic groups. Simple and multiple linear regression analyses were used to analyze the association between diabetes and LV mass before (Model 1) and after adjusting for additional variables such as BMI (Model 2), demographics and education (Model 3), systolic BP (Model 4), and history of CAD, physical activity and moderate alcohol consumption (Model 5). Logistic regression analyses were performed using LV hypertrophy as the dependent variable, and the same covariates described above as independent variables. Same analyses were repeated using waist circumference instead of BMI. Additionally, the use of antihypertensive medication was adjusted for in the final models of multivariable analyses. The interaction terms set in the hypothesis were tested in the final models of multiple linear regression analyses: age-DM, race-DM, BMI-DM, and waist-DM. All statistical analyses were performed with the SAS software (version 9.0, SAS Institute, Cary, NC).

Results

Baseline cohort characteristics are shown in Table 1. As expected, BMI, waist circumference, triglycerides, hypertension, systolic BP, and a history of CAD were significantly higher in the Diabetic group than in the Non-diabetic group. Percentages of blacks and Hispanics were also higher in the Diabetic group. Antihypertensive medication use was significantly more frequent in the Diabetic than in the Non-diabetic group. Since the time of enrollment was the mid 1990's, angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers were not commonly used at the time of this study.

Table 2 shows the comparison of echocardiographic data. The Diabetic group had higher LV mass than in the Non-diabetic group in the overall cohort and in each race-ethnic subgroup.

Wall thicknesses, and LV diastolic dimension were also higher in the Diabetic group than in the Non-diabetic group.

We performed multiple regression analyses to evaluate the relationship between DM and LV mass. As shown in Table 3, the presence of DM was associated with increased LV mass in univariate (Model 1) and multivariable analyses when various covariates were added in the models (Models 2–5). These relationships were consistent when waist circumference was substituted for BMI as a parameter of body size, and systolic BP was substituted for the presence of hypertension. When the use of antihypertensive medication was added to Model 5, the result was similar (estimate of diabetes, 6.05; SE, 3.14), although the p-value decreased to 0.05. We performed a separate multiple logistic regression analyses to determine the effect of DM on the likelihood of having LV hypertrophy. As shown in Table 4, DM was associated with 1.7-fold increase in risk of having LV hypertrophy in univariate analysis (Model 1), and with about 1.5-fold higher likelihood of LV hypertrophy in multivariable analyses (Models 2–5). These relationships were maintained when waist circumference instead was substituted for BMI, and systolic BP was substituted for the presence of hypertension in the models. When the use of antihypertensive medication was added to Model 5, the significance of the result was not affected (OR, 1.53; 95%CI, 1.17–2.01; P=0.002). The results based on gender-specific 75th percentile cutoffs (female, 187.5g; male, 230.2g) were similar to those based on the overall 75th percentile in the entire study population.

Finally, we analyzed the possible interactions of DM with age, body size, and race/ethnicity on the risk of LV hypertrophy. When we used BMI as an index of body size, no interactions were observed between diabetes and BMI (p =0.6). On the other hand, when we used waist circumference, there was a significant interaction between DM and waist circumference on LV mass (P=0.01).

Discussion

In this study, we confirmed the previous findings of a positive relation between type 2 DM and increased LV mass. The presence of type 2 DM was associated with approximately 1.5-fold increase in risk of having LV mass above the 75th percentile of the general population. The relationship between type 2 DM and LV mass was independent of obesity in general (as expressed by elevated BMI), but central obesity (waist circumference) and DM had a positive interaction on the risk of LV hypertrophy.

In this study, the presence of type 2 DM was associated with increased LV mass independent of other confounders, including race/ethnicity, education and physical activity. Similar findings, although with lower strength of association, were observed after adjustment for the use of antihypertensive medication. This is the second report to show the positive association between type 2 DM and increased LV mass in multiethnic populations.¹⁰ There is a continuing controversy on whether DM itself is associated with increased LV mass. There have been some reports showing that type 2 DM is associated with increased LV mass in hypertensive patients,¹¹ the general population,^{1,12} and a multiethnic population.¹⁰ On the other hand, the association in those studies was not strong, and was only seen under the coexistence of hypertension.^{13,14} The relationship between DM and increased LV mass was also reported to be secondary to an interaction with age and obesity in a mostly obese sample.⁵ Type 2 DM is usually accompanied by other risk factors such as aging, obesity, hypertension, and socio-cultural factors.¹⁵ Therefore, we performed multivariable analyses to adjust for various confounding factors including socio-economical status and lifestyle indicators, which were rarely performed in previous literatures.^{2,10–12}

In our study, the likelihood of having LV mass above the 75th percentile of the distribution was 1.46-fold (95% CI, 1.13 to 1.88) higher in diabetics than in non-diabetics, independent of various covariates including hypertension. Very similar results were obtained by Palmieri et al., who reported that the likelihood of LV hypertrophy (partition values 46.7g/m² in women and 49.2g/m² in men) was 1.32-fold higher in diabetic than non-diabetic subjects. Although the cutoff values of LV mass were different from those used in our study, the consistency of the results supports the clinical impact of DM on increased LV mass.

There have been several studies showing that body size is strongly associated with increased LV mass.^{3,4,16,17} Galvan et al showed that, when body mass was adequately taken into account, insulin resistance was not associated with LV mass.³ Body fat has been shown to be associated with LV mass.^{4,16} Type 2 DM may have some interaction with obesity on increased LV mass.⁵ Because obesity and insulin resistance are closely associated with each other, obesity itself may have an impact on increased LV mass in diabetics. In our study, when BMI was used as an index of obesity, no interaction with DM on LV mass was observed. On the other hand, when waist circumference was used, there was a significant interaction between DM and waist on the risk of LV hypertrophy. This result suggests that the impact of type 2 DM on LV mass may be partly explained by its interaction with central obesity. It is possible that the different types of adipocytokines contained in visceral fat may have a direct influence on LV mass.¹⁸

Important strengths of our study are the large sample size based on a population-based cohort, the tri-ethnic nature of the sample, and the comprehensive in-person risk assessment. On the other hand, there are some limitations to this study. First, this is a cross-sectional study, therefore a cause-effect relationship between type 2 DM and increased LV mass cannot be established. Second, we did not measure the amount of body fat or visceral fat, a circumstance that precludes the analysis of the impact of visceral obesity on increased LV mass.

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Table 1

Baseline characteristics

Variable	Overall (N=1932)	Diabetes Mellitus		P*
		Yes (N=443)	No (N=1489)	
Male sex	39.8 %	41.1 %	39.4 %	0.8
Age (years)	67.9 ± 9.6	67.8 ± 8.4	67.9 ± 10.0	0.9
Height (m)	1.63 ± 0.10	1.63 ± 0.10	1.62 ± 0.10	0.4
Body mass index (kg/m ²)	27.7 ± 5.3	29.2 ± 5.7	27.2 ± 5.1	<0.0001
Waist (cm)	93.8 ± 12.9	97.8 ± 12.2	92.7 ± 13.0	<0.0001
Race				<0.0001
Black	21.9 %	25.5 %	20.8 %	
Hispanic	57.4 %	61.2 %	56.2 %	
White	20.8 %	13.3 %	23 %	
History of coronary artery disease	8.4 %	11.1 %	7.7 %	0.02
Low-density lipoprotein cholesterol (mg/dl)	131 ± 36	128 ± 39	131 ± 35	0.08
High-density lipoprotein cholesterol (mg/dl)	46 ± 15	44 ± 14	47 ± 15	<0.0001
Triglycerides (mg/dl)	138 ± 85	160 ± 113	131 ± 74	<0.0001
Fasting blood glucose (mg/dl)	107 ± 50	169 ± 74	89 ± 14	<0.0001
Hypertension	67.9 %	79.7 %	64.4 %	<0.001
Antihypertensive medication	862 (44.6%)	263 (59.4%)	599 (40.2%)	<0.001
Diuretics	849 (43.9%)	586 (52.0%)	263 (65.3%)	
Beta-blockers	319 (16.5%)	212 (28.0%)	107 (43.3%)	
Calcium channel blockers	77 (4.0%)	53 (8.9%)	24 (14.6%)	
Angiotensin-converting enzyme inhibitors	18 (0.9%)	9(1.6%)	9(6.0%)	
Systolic blood pressure (mmHg)	144 ± 21	147 ± 20	143 ± 21	0.002
Diastolic blood pressure (mmHg)	83 ± 11	84 ± 11	83 ± 11	0.2
Current smoker	17.0 %	16.5 %	17.1 %	0.9
Moderate alcohol consumption	35.7 %	28.2 %	38.0 %	<0.001

* P values indicate comparisons between diabetic and non-diabetic subjects.

History of coronary artery disease: having either a history of bypass surgery, angioplasty, or myocardial infarction.

Moderate alcohol consumption: current drinking of more than one drink per month and less than or equal to 2 drinks

Table 2

Comparison of echocardiographic data

Variable	Overall (N=1932)	Diabetes Mellitus		P*
		Yes (N=443)	No (N=1489)	
Left ventricular mass (g)	Overall	177 ± 59	174 ± 59	<0.0001
	Black	187 ± 64	181 ± 65	0.003
	Hispanic	175 ± 59	173 ± 58	0.009
Septal wall thickness (mm)	White	173 ± 55	170 ± 54	0.03
		11.3 ± 2.2	11.1 ± 2.2	<0.0001
Posterior wall thickness (mm)		11.3 ± 1.8	10.8 ± 1.8	<0.0001
Left ventricular end-diastolic diameter (mm)		44.3 ± 5.4	44.1 ± 5.3	0.03

* P values indicate comparisons between diabetes and non-diabetics.

Table 3

Variables associated with increased left ventricular mass

Variables	Model 1			Model 2			Model 3			Model 4			Model 5		
	Estimate	SE	p	Estimate	SE	p	Estimate	SE	p	Estimate	SE	p	Estimate	SE	p
Age (years)	14.8	3.2	<0.0001	10.2	3.2	0.001	7.5	3.0	0.01	6.7	2.9	0.02	6.3	2.9	0.03
Body mass index (kg/m ²)				2.4	0.25	<0.0001	3.1	0.2	<0.0001	2.7	0.24	<0.0001	2.6	0.2	<0.0001
Sex (male=1, female=0)							0.8	0.14	<0.0001	0.5	0.14	<0.0001	0.5	0.14	<0.0001
Race (Black vs. White)							41.5	2.6	<0.0001	41.2	2.5	<0.0001	39.3	2.5	<0.0001
Ethnicity (Hispanic vs. White)							13.0	3.9	<0.0001	8.9	3.8	0.02	10.5	3.8	0.006
Education (less than high school vs. high school or above)							1.9	3.9	0.63	-0.13	3.8	0.97	0.4	3.8	0.9
Diabetes (yes vs. no)							0.3	3.0	0.92	1.5	2.9	0.6	0.9	2.9	0.7
Arterial hypertension (yes vs. no)										0.58	0.06	<0.0001	0.6	0.06	<0.0001
Arterial disease (yes vs. no)													21.9	4.4	<0.0001
Physical activity (yes vs. no)													-2.7	2.5	0.3
Alcohol consumption (yes vs. no)													3.6	2.6	0.2

Estimates and standard errors (SE).

Variables shown in each model were entered altogether for adjustment in multiple regression analysis.

Table 4
Variables associated with above the 75th percentile of left ventricular mass

	Model 1		Model 2		Model 3		Model 4		Model 5	
	Odds ratio	p	Odds ratio	p	Odds ratio	p	Odds ratio	p	Odds ratio	p
Diabetes (yes or no)	1.70 (1.35–2.14)	<0.0001	1.54 (1.22–1.95)	0.0003	1.48 (1.15–1.90)	0.002	1.45 (1.12–1.87)	0.004	1.46 (1.13–1.88)	0.004
Body mass index (kg/m ²)			1.05 (1.03–1.07)	<0.0001	1.09 (1.07–1.11)	<0.0001	1.08 (1.05–1.10)	<0.0001	1.08 (1.05–1.10)	<0.0001
Age (years)					1.03 (1.01–1.04)	<0.0001	1.02 (1.01–1.03)	0.005	1.02 (1.01–1.03)	0.008
Sex (male=1, female=0)					4.32 (3.42–5.46)	<0.0001	4.53 (3.57–5.76)	<0.0001	4.35 (3.41–5.54)	<0.0001
Race Black vs. White					1.51 (1.08–2.12)	0.016	1.34 (0.95–1.90)	0.095	1.40 (0.99–1.99)	0.07
Hispanics vs. White					0.93 (0.66–1.31)	0.7	0.87 (0.62–1.24)	0.5	0.86 (0.60–1.23)	0.4
Education above high school					1.02 (0.78–1.32)	0.9	1.07 (0.82–1.40)	0.6	1.04 (0.79–1.37)	0.8
Systolic blood pressure (mmHg)							1.02 (1.02–1.03)	<0.0001	1.02 (1.01–1.03)	<0.0001
Coronary artery disease									1.66 (1.15–2.39)	0.007
Any physical activity									0.90 (0.71–1.14)	0.4
Moderate alcohol consumption									1.12 (0.88–1.42)	0.4

Values are odds ratio and 95% confidence intervals.

All covariates shown in each model were entered altogether for adjustment in multiple logistic regression analysis.