Association of Albuminuria With Systolic and Diastolic Left Ventricular Dysfunction in Type 2 Diabetes

The Strong Heart Study

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OBJECTIVES

We sought to compare systolic and diastolic function in American Indians with diabetes mellitus (DM) based on albuminuria status.

BACKGROUND

Albuminuria has been shown to predict cardiovascular disease (CVD) in populations with DM. However, the mechanism of the association of albuminuria and CVD is unclear.

METHODS

We compared echo-derived indices of left ventricular (LV) systolic and diastolic function in three groups of American Indians with DM based on albuminuria status: I = no albuminuria (<30 mg albumin/g creatinine); II = microalbuminuria (30 to 300 mg/g); and III = macroalbuminuria (>300 mg/g).

RESULTS

Group II and III were slightly older than Group I with no significant gender difference between groups. Systolic blood pressure increased and body mass index decreased from Group I to Group III. Left ventricular systolic function was lower in the groups with albuminuria with step-wise decreases in ejection fraction and stress-corrected midwall shortening (MWS) from Group I to Group III. Similar findings were noted in diastolic LV filling with lower mitral E/A ratios and longer deceleration times in groups with albuminuria. The proportion of participants with abnormal MWS and abnormal LV diastolic relaxation showed step-wise increases from no albuminuria to macroalbuminuria. In multivariate analysis, albuminuria status remained independently associated with both systolic and diastolic dysfunction after adjusting for age, gender, body mass index, systolic blood pressure, duration of diabetes, coronary artery disease, and LV mass.

CONCLUSIONS

Albuminuria is independently associated with LV systolic and diastolic dysfunction in type 2 DM; this may explain in part the relationship of albuminuria to increased cardiovascular (CV) events in the DM population. Screening for albuminuria identifies individuals with high CV risk and possible cardiac dysfunction. (J Am Coll Cardiol 2003;41:2022–8) © 2003 by the American College of Cardiology Foundation

Albuminuria has been shown to predict cardiovascular (CV) morbidity and mortality in individuals with both type 1 and type 2 diabetes mellitus (DM) independent of conventional CV risk factors including age, arterial hypertension, and hypercholesterolemia (1–5). Although the mechanism of the association of albuminuria with cardiac events is not clear, it is possible that the vascular changes that lead to renal dysfunction may also be present in the vasculature of the heart and, thus contribute to cardiac dysfunction (6,7).

This is supported by the finding of higher incidence of coronary heart disease (CHD) mortality in diabetic patients with microalbuminuria (3,5,8). In addition, the existence of a distinct diabetic cardiomyopathy, characterized by systolic and diastolic dysfunction, may also contribute to the increased CV mortality seen in diabetic patients. However, whether albuminuria is also associated with abnormal intrinsic left ventricular (LV) myocardial function independent of other confounding factors remains unclear. The aim of this cross-sectional study was to examine the relationship of albuminuria to LV systolic and diastolic function in diabetic adults from a population-based sample of adult American Indians.

METHODS

The Strong Heart Study (SHS) is a population-based cohort survey of CV risk factors and prevalent and incident cardiovascular disease (CVD) in American Indian communities in Arizona, Oklahoma, and South and North Dakota.
Abbreviations and Acronyms

- CHD = coronary heart disease
- CV = cardiovascular
- CVD = cardiovascular disease
- DM = diabetes mellitus
- ECG = electrocardiogram/electrocardiographic
- LV = left ventricular
- MWS = midwall shortening
- SHS = Strong Heart Study

As previously described (9,10), tribal members ages 45 to 74 years of three communities in Arizona, seven tribes in Southwestern Oklahoma, and three communities in South and North Dakota, were recruited from tribal members living on the reservations or (in Oklahoma) in a defined geographic area (overall participation rate = 62%) for an initial examination in 1989 to 1992.

The second SHS examination was conducted from 1993 to 1995 to assess change over time in body habitus, blood pressure, and most other baseline measures, and to add echocardiography among surviving participants. A total of 3,630 surviving phase I enrollees participated in the second SHS examinations, for an 89% return rate. Standardized measurements of seated brachial blood pressure; aspects of body habitus including body mass index, waist-hip ratio, and percent body fat by bioelectric impedance; fasting glucose, insulin, lipid, and lipoprotein concentrations; and 2-h glucose tolerance test and glycosylated hemoglobin levels were obtained. Diabetes was diagnosed by World Health Organization criteria (11), if the fasting blood sugar level was >140 mg/dl, 2-h post-glucose challenge level was >200 mg/dl, or participants were receiving hypoglycemic medication.

As previously described (12), albuminuria was measured by collection of fasting random urine specimen on arrival to the clinic, usually in the morning. Urine creatinine content was measured by a sensitive, nephelometric technique (14). Microalbuminuria was defined as albumin/creatinine ratios ≥30 and <300 mg albumin/g creatinine. Macroleluminuria was recognized as urine albumin/creatinine ratios ≥300 mg/g. The SHS participants were classified as hypertensive if resting blood pressure was ≥140 mm Hg systolic and/or 90 mm Hg diastolic, or if participants were on antihypertensive medications. Echocardiograms were performed in 3,501 participants (97%) in the second SHS examination. For the present study, SHS participants with diabetes (n = 1,748) were selected.

Eligible diabetic participants in the second SHS examination were divided into three nonoverlapping groups based on their albuminuria status: I = 756 participants with no albuminuria (<30 mg albumin/creatinine); II = 581 participants with microalbuminuria (30 to 299 mg/g creatinine); and III = 411 participants with macroleluminuria (≥300 mg/g creatinine). Participants were categorized as having definite fatal or nonfatal CHD on the basis of clinical and electrocardiographic (ECG) evidence of coronary disease or myocardial infarction, as determined by a physician review panel and Minnesota code and ECGs (15).

Echocardiographic methods. Imaging and Doppler echocardiograms were performed using methods adapted from those employed in previous studies from the Cornell Medical Center laboratory that served as the echocardiography reading center. Studies were performed using phased-array echocardiographs with M-mode, two-dimensional and pulsed, continuous wave, and color-flow Doppler capabilities following a standardized protocol (16,17).

Echocardiographic measurements. Correct orientation of planes for imaging and Doppler recordings was verified as previously described (18). Measurements were made using a computerized review station equipped with a digitizing tablet and monitor screen overlay for calibration and performance of each needed measurement. The LV internal dimension and septal and posterior wall thicknesses were measured according to the American Society of Echocardiography recommendations (19,20) during up to three cardiac cycles.

To facilitate relating measurements of LV diastolic transmitral blood flow velocity to volume flow, the pulsed Doppler sample volume was placed at the middle of the mitral annulus, the diameter of which varies relatively modestly during the cardiac cycle, as opposed to the level of the leaflet tips where the mitral orifice shows substantial variation through the cardiac cycle. Participants were asked to hold their breath during pulse-wave Doppler interrogation. The leading edge of the transmitral Doppler flow pattern was traced to derive the peak of early diastolic and atrial phase LV filling (‘E’ and “A,” respectively), their ratio, the deceleration time of early diastolic LV filling, and the atrial filling fraction. Doppler measurements were performed off-line from an average of several cardiac cycles. Heart rate was measured simultaneously.

Calculation of derived variables. End-diastolic LV dimensions were used to calculate LV mass by a formula that yields values closely correlated with necropsy LV weight (r = 0.90, p < 0.001) (21) and which showed good reproducibility (RHO = 0.93, p < 0.001) in a series of 183 hypertensive patients studied twice by echocardiography (22). Left ventricular mass was normalized for body height2.7, where 2.7 is the power of the allometric or growth relation between LV mass and body height (23).

Measures of myocardial performance. The primary approach used to assess myocardial contractile efficiency was examination of LV systolic shortening in relation to end-systolic stress. Because it has been documented that the traditional practice of relating endocardial shortening to the mean level of end-systolic stress across the ventricular wall may yield misleading results in individuals with either concentric geometry (24) or with LV dilation (25), primary reliance was placed on the relation of midwall fractional shortening to midwall circumferential end-systolic stress.
Table 1. General Characteristics

<table>
<thead>
<tr>
<th></th>
<th>No Albuminuria (n = 685)</th>
<th>Microalbuminuria (n = 519)</th>
<th>Macroalbuminuria (n = 372)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>59 ± 8†</td>
<td>61 ± 8‡</td>
<td>61 ± 8§</td>
</tr>
<tr>
<td>Gender (% female)</td>
<td>70</td>
<td>66</td>
<td>65</td>
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<tr>
<td>Center</td>
<td></td>
<td></td>
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<tr>
<td>Arizona (%)</td>
<td>225 (33)</td>
<td>274 (53)</td>
<td>225 (60)</td>
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<tr>
<td>Oklahoma (%)</td>
<td>233 (34)</td>
<td>124 (24)</td>
<td>73 (20)</td>
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<td>North/South Dakota (%)</td>
<td>228 (33)</td>
<td>120 (23)</td>
<td>74 (20)</td>
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<tr>
<td>Body mass index (kg/m²)</td>
<td>33 ± 6†</td>
<td>32 ± 7</td>
<td>32 ± 6‡</td>
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<tr>
<td>Systolic BP (mm Hg)</td>
<td>127 ± 17†</td>
<td>132 ± 19‡</td>
<td>144 ± 24‡</td>
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<tr>
<td>Diastolic BP (mm Hg)</td>
<td>75 ± 10†</td>
<td>75 ± 10‡</td>
<td>77 ± 11‡</td>
</tr>
<tr>
<td>Hypertension (%)</td>
<td>45†</td>
<td>56†</td>
<td>74‡</td>
</tr>
<tr>
<td>CHD (%)</td>
<td>5†</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td>Hemoglobin A1C</td>
<td>7.7 ± 2.4‡</td>
<td>8.8 ± 2.3‡</td>
<td>9.2 ± 2.3‡</td>
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<tr>
<td>Duration of DM (yrs)</td>
<td>7 ± 8†</td>
<td>11.3 ± 9‡</td>
<td>17 ± 10‡</td>
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<tr>
<td>Total cholesterol (mg/dl)</td>
<td>187 ± 38†</td>
<td>183 ± 39†</td>
<td>200 ± 45‡</td>
</tr>
<tr>
<td>Total triglyceride</td>
<td>166 ± 126†</td>
<td>180 ± 128‡</td>
<td>205 ± 138‡</td>
</tr>
<tr>
<td>HDL cholesterol (mg/dl)</td>
<td>39 ± 11</td>
<td>39 ± 12</td>
<td>39 ± 12</td>
</tr>
<tr>
<td>LDL cholesterol (mg/dl)</td>
<td>117 ± 32*</td>
<td>111 ± 32‡</td>
<td>118 ± 37‡</td>
</tr>
<tr>
<td>Fibrinogen (mg/dl)</td>
<td>356 ± 70†</td>
<td>373 ± 71‡</td>
<td>453 ± 108‡</td>
</tr>
<tr>
<td>Serum creatinine (mg/dl)</td>
<td>0.85 ± 0.33‡</td>
<td>0.86 ± 0.28‡</td>
<td>1.63 ± 1.59‡</td>
</tr>
</tbody>
</table>

*p < 0.05 compared to microalbuminuria by ANOVA followed by the Scheffé post-hoc test; †p < 0.05 compared to macroalbuminuria by ANOVA followed by the Scheffé post-hoc test; ‡p < 0.05 compared to no albuminuria by ANOVA followed by the Scheffé post-hoc test.

BP = blood pressure; CHD = coronary heart disease; DM = diabetes mellitus; HDL = high-density lipoprotein; LDL = low-density lipoprotein.

measured at the level of the LV minor axis (24,26,27). Midwall fractional shortening was calculated taking into account the epicardial migration of the midwall during systole. Circumferential end-systolic stress was also estimated at the midwall from M-mode tracings, using a cylindrical model (27). To evaluate LV performance taking end-systolic stress into account, observed midwall shortening (MWS) was expressed as a percentage of the value predicted from circumferential end-systolic stresses using equations derived from previously studied normal subjects (24). For convenience, this variable is termed stress-corrected MWS (28).

Subnormal stress-corrected MWS was defined as stress-corrected MWS <88.7%. Abnormal diastolic function was defined as E/A ratios of <0.6 (compatible with impaired early diastolic relaxation) and >1.5 (compatible with restricted LV filling). These represented approximately the 5th and 95th percentiles in a reference group of 124 SHS participants (mean age = 58 years; 78 women) who had normal blood pressure (mean = 113/69 mm Hg) on no CV medications, normal body weight (body mass index <25 kg/m²), and no prevalent heart disease. These partition values have recently been shown to have prognostic significance independent of conventional CV risk factors (29).

Statistical analyses. Data were analyzed using SPSS software (SPSS Inc., Chicago, Illinois). Data are expressed as mean ± SD. Differences between groups of participants were assessed by ANOVA followed by the Scheffé post-hoc test. The independence of differences from effects of covariates was assessed using single factorial ANOVA in the general linear model that is a form of analysis of covariance. The procedure was used to specify a single categorical factor that identified groups, with other confounding and categorical variables used as covariates, rather than as additional factors. The differences between the adjusted groups were assessed using Sidak’s post-hoc test (30). Comparison of abnormal diastolic function between the no albuminuria and the micro- and macroalbuminuria groups was assessed using logistic regression with use of indicator variables representing micro- and macroalbuminuria.

RESULTS

Clinical characteristics (Table 1). Diabetic participants with microalbuminuria and macroalbuminuria were slightly older than those without albuminuria with no significant gender difference among groups. Microalbuminuria and macroalbuminuria were more common in participants in Arizona than in the other two centers. Step-wise increases were seen in systolic blood pressure and prevalences of hypertension and coronary artery disease from the group without albuminuria to that with macroalbuminuria with a reverse trend for body mass index. Duration of diabetes and hemoglobin A1C, and fibrinogen levels also increased step-wise from no albuminuria to macroalbuminuria. Total cholesterol and triglyceride levels were highest in the macroalbuminuria group. There was no difference in the high-density lipoprotein levels among the three groups. Serum creatinine was higher in the group with macroalbuminuria. Consequently, a step-wise increase in preva-
lence of LV hypertrophy was seen from no albuminuria to macroalbuminuria. Circumferential wall stress was higher in the group with macroalbuminuria than in the other two groups. Ejection fraction and stress-corrected MWS decreased step-wise from no albuminuria to macroalbuminuria. Consequently, the prevalences of abnormal stress-corrected MWS and abnormal ejection fraction (<55%) showed step-wise increases from no albuminuria to macroalbuminuria (Fig. 1).

**LV diastolic filling (Table 3).** Mitral E velocity was lower and mitral A velocity was higher in the two groups with albuminuria. Consequently, the mitral E/A ratio was lower in the groups with micro- or macroalbuminuria than in the group without albuminuria. Mitral deceleration time was longer and atrial filling fraction was higher with albuminuria than without albuminuria. Consequently, a step-wise increase in the prevalence of abnormal relaxation was seen from no albuminuria to macroalbuminuria (Fig. 2).

**Systemic hemodynamics (Table 4).** Heart rate and pulse pressure were higher in the groups with albuminuria than in the group without albuminuria. Stroke volume and stroke index were similar in the three groups. Cardiac index was highest in the group with macroalbuminuria. There was no difference in total peripheral resistance or its index among the three groups. Pulse pressure/stroke index, a non-invasive measure of arterial stiffness, increased from the group without albuminuria to the group with macroalbuminuria. This difference remained statistically significant after adjusting for age.

**Regression analyses.** In multivariate analysis, stress-corrected MWS was lower in participants with macroalbuminuria after adjusting for age, gender, center, present CHD, duration of diabetes, systolic blood pressure, and LV mass ($p = 0.005$). Micro- and, especially, macroalbuminuria were associated with abnormal diastolic function (E/A $<0.6$ or E/A $>1.5$), after adjusting for the above covariates with the addition of ejection fraction ($p < 0.01$).

**DISCUSSION**

The present study provides the first data on LV systolic and diastolic function and systemic hemodynamics in a population-based sample of diabetic adults with micro- or macroalbuminuria as compared to normoalbuminuria. Although previous studies have shown albuminuria to be a strong predictor of CV morbidity and mortality, the mechanism underlying this relationship has not been well elucidated. Associations of albuminuria with coronary artery disease and peripheral vascular disease have been reported in diabetic (6–8) and nondiabetic patients (31). The present study provides new and potentially important findings, identifying associations of albuminuria with abnormal systolic and diastolic function independent of age, gender, blood pressure, duration of diabetes, and other covariates. Because albuminuria is associated with severity of diabetes and diabetes itself is associated with “a distinct diabetic cardiomyopathy,” LV dysfunction in the albuminuria groups may be partially associated with a greater severity of diabetes. However, the relationship of albuminuria to LV dysfunction remained unchanged after adjusting for hemo-

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### Table 2. LV Systolic Function

<table>
<thead>
<tr>
<th></th>
<th>No Albuminuria (n = 685)</th>
<th>Microalbuminuria (n = 519)</th>
<th>Macroalbuminuria (n = 372)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV internal dimension (cm)</td>
<td>4.9 ± 0.5*</td>
<td>4.9 ± 0.5*</td>
<td>5.1 ± 0.6†‡</td>
</tr>
<tr>
<td>Relative wall thickness</td>
<td>0.35 ± 0.05*</td>
<td>0.36 ± 0.05*</td>
<td>0.37 ± 0.06†‡</td>
</tr>
<tr>
<td>LV mass (g)</td>
<td>155 ± 36*</td>
<td>160 ± 39*</td>
<td>181 ± 50†‡</td>
</tr>
<tr>
<td>LV mass/height*2</td>
<td>41 ± 9†‡</td>
<td>43 ± 10†‡</td>
<td>49 ± 13†‡</td>
</tr>
<tr>
<td>LV hypertrophy (%)</td>
<td>23†‡</td>
<td>31†‡</td>
<td>49†‡</td>
</tr>
<tr>
<td>Circumferential wall stress (kdynes/cm²)</td>
<td>149 ± 38*</td>
<td>152 ± 43*</td>
<td>172 ± 58†‡</td>
</tr>
<tr>
<td>Stress-corrected MWS (%)</td>
<td>105 ± 13†‡</td>
<td>102 ± 13†‡</td>
<td>98 ± 16†‡</td>
</tr>
<tr>
<td>Fractional shortening (%)</td>
<td>35 ± 6*</td>
<td>35 ± 6*</td>
<td>33 ± 8†‡</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>64 ± 8*</td>
<td>63 ± 9*</td>
<td>60 ± 12†‡</td>
</tr>
<tr>
<td>Midwall dysfunction (%)</td>
<td>9*</td>
<td>12*</td>
<td>24†‡</td>
</tr>
<tr>
<td>Abnormal ejection fraction (%)</td>
<td>9</td>
<td>13</td>
<td>22</td>
</tr>
</tbody>
</table>

* $p < 0.05$ compared to macroalbuminuria by ANOVA followed by the Scheffe post-hoc test; † $p < 0.05$ compared to microalbuminuria by ANOVA followed by the Scheffe post-hoc test; ‡ $p < 0.05$ compared to no albuminuria by ANOVA followed by the Scheffe post-hoc test.

LV = left ventricular; MWS = midwall shortening.

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**Figure 1.** Prevalence of subnormal midwall shortening (MWS) based on albuminuria status, where I = no albuminuria; II = microalbuminuria; and III = macroalbuminuria. **Solid bars** = percentage of subnormal MWS. Comparison between each group was made using chi-squared statistics with Bonferroni correction.
albuminuria. Solid bars with Bonferroni correction. Comparison between each group was made using chi-squared statistics due to the low vessel wall content of heparan sulfate (32) clear. It has been proposed that albuminuria re
etic mechanisms behind this association are not entirely
demonstrated that albuminuria is associated with renal alter-
ations, proliferative retinopathy, and CVD in diabetic and
vascular dysfunction (32). Studies have dem-
results remained unchanged.
Excluding the participants with CHD (total 142) and the
confounding in the influence of CHD, we repeated the analysis
independently impact on LV function. To remove the
confounding influence of CHD, we repeated the analysis
excluding the participants with CHD (total 142) and the
findings remained unchanged.
Albuminuria has been proposed to represent a marker of
a generalized vascular dysfunction (32). Studies have dem-
strated that albuminuria is associated with renal alter-
ations, proliferative retinopathy, and CVD in diabetic and nondiabetic populations (6,31,33). The specific pathogene-
etic mechanisms behind this association are not entirely clear. It has been proposed that albuminuria reflects a renal and systemic transvascular albumin leakage that is perhaps due to the low vessel wall content of heparan sulfate (32) that has been shown not only in the glomerular basement
membrane (34) but also in the atherosclerotic aorta (35) and
coronary arteries (36). This generalized increase of vascular
permeability can also cause leakiness of collagen, choles-
terol, and advanced glycated end products that have been
reported in the myocardium of human hearts (37,38). These

tissue alterations can increase end-diastolic myocardial stiff-
ness as well as LV mass, and alter normal systolic function.
Furthermore, a strong negative correlation between the
accumulation of lipids and concentration of the heparan sulfate in the arterial walls has been reported (39). The change in permeability causing insudation of lipoproteins into the intima of large vessels can lead to atherosclerosis of the epicardial coronary arteries as well as small arterioles of the heart. In addition, heparan sulfate proteoglycan in plasma membranes of endothelial cells has important anti-
thrombogenic properties (40). Loss of normal sulfated
heparan sulfate might, therefore, contribute to the forma-
tion of microthrombi and occlusion of the small vessels of the
heart. Small vessel disease can lead to subendocardial
ischemia causing systolic and diastolic myocardial dysfunc-
tion.
Our study also revealed an association of microalbumin-
uria with increased arterial stiffness, independent of age.
Impaired arterial dilatory capacity in clinically healthy sub-
jects with increased urinary albumin excretion has also been
reported (41). This can be partially explained by the
accumulation of lipoproteins in the vascular walls due to
endothelial permeability.

**STUDY LIMITATIONS**

Although our analysis used only the albumin measurement
taken at the clinic examination when other data were
collected including the echocardiogram, we believe that these
data are representative of the patient’s long-term
values. The albumin/creatinine ratios measured about 3.5
years earlier at the first SHS examination showed step-wise
increases from the nonalbuminuric group to those with
micro- or macroalbuminuria (41 ± 357 mg/g to 85 ± 162
mg/g to 1,312 ± 3,654 mg/g, respectively; p < 0.001).
Our study was performed in a Native-American Indian popu-
lation of middle-age individuals with a high prevalence of
obesity and hypertension that are typical of patients with

<table>
<thead>
<tr>
<th>Table 3. Left Ventricular Diastolic Filling</th>
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<tr>
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<tr>
<td>E velocity (cm/s)</td>
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<tr>
<td>A velocity (cm/s)</td>
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<tr>
<td>E/A</td>
</tr>
<tr>
<td>Atrial filling fraction</td>
</tr>
<tr>
<td>Deceleration time (ms)</td>
</tr>
<tr>
<td>Diastolic dysfunction (%)</td>
</tr>
</tbody>
</table>

* p < 0.05 compared to microalbuminuria by ANOVA followed by the Scheffe post-hoc test; † p < 0.05 compared to macroalbuminuria by ANOVA followed by the Scheffe post-hoc test; ‡ p < 0.05 compared to no albuminuria by ANOVA followed by Scheffe post-hoc test.

Figure 2. Prevalence of abnormal diastolic function based on albuminuria status where I = no albuminuria; II = microalbuminuria; and III = macroalbuminuria. Solid bars = percentage of abnormal diastolic function. Comparison between each group was made using chi-squared statistics with Bonferroni correction.
Table 4. Systemic Hemodynamics

<table>
<thead>
<tr>
<th></th>
<th>No Albuminuria</th>
<th>Microalbuminuria</th>
<th>Macroalbuminuria</th>
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<tbody>
<tr>
<td></td>
<td>(n = 685)</td>
<td>(n = 519)</td>
<td>(n = 372)</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>68 ± 10†</td>
<td>70 ± 10‡</td>
<td>71 ± 10‡</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>55 ± 15†</td>
<td>57 ± 16‡</td>
<td>68 ± 20‡</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>73 ± 14</td>
<td>72 ± 14</td>
<td>72 ± 15</td>
</tr>
<tr>
<td>Stroke volume/BSA (m²/kg)</td>
<td>38 ± 9</td>
<td>38 ± 7</td>
<td>38 ± 8</td>
</tr>
<tr>
<td>Cardiac output (ml/min)</td>
<td>4,938 ± 1,171</td>
<td>4,973 ± 1,193</td>
<td>5,087 ± 1,266</td>
</tr>
<tr>
<td>Cardiac index (m²/kg min)</td>
<td>2,579 ± 564†</td>
<td>2,625 ± 566</td>
<td>2,705 ± 639†</td>
</tr>
<tr>
<td>Total peripheral resistance (dynes/cm⁵·m⁻²)</td>
<td>1,668 ± 422</td>
<td>1,673 ± 442</td>
<td>1,718 ± 482</td>
</tr>
<tr>
<td>Total peripheral resistance/BSA (dynes/cm⁵·m⁻²)</td>
<td>3,185 ± 768</td>
<td>3,166 ± 804</td>
<td>3,218 ± 856</td>
</tr>
<tr>
<td>Pulse pressure/stroke index (mm Hg·m²/kg)</td>
<td>1.5 ± 0.5‡</td>
<td>1.6 ± 0.5†‡</td>
<td>1.9 ± 0.9†‡</td>
</tr>
</tbody>
</table>

* p < 0.05 compared to microalbuminuria by ANOVA followed by the Scheffé post-hoc test; † p < 0.05 compared to macroalbuminuria by ANOVA followed by the Scheffé post-hoc test. ‡ p < 0.05 compared to no albuminuria by ANOVA followed by Scheffé post-hoc test.

BSA = body surface area.

CONCLUSIONS

Among diabetic members of a population-based sample, albuminuria is independently associated with worse systolic and diastolic LV function, which may contribute to the relation of albuminuria among diabetics with an increased rate of CV events. Thus, albuminuria in individuals with DM identifies high CV risk due to cardiac dysfunction.

REFERENCES


