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Cardiovascular Disease Risk, Vascular Health, and Erectile Dysfunction among Middle-Aged, Clinically Depressed Men

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Abstract

Background—Erectile dysfunction (ED) is especially common in men with major depressive disorder (MDD). This study examined the extent to which risk factors for cardiovascular disease (CVD) and vascular dysfunction were associated with ED severity in a series of MDD patients.

Methods—The sample included 46 middle-aged [*M* (*SD*) age = 53 (7)], sedentary men diagnosed with MDD. ED severity was assessed by the Arizona Sexual Experiences Scale (ASEX), item 3. Depression severity was measured by the Beck Depression Inventory (BDI). CVD risk factors were quantitated by the Framingham Cardiovascular Disease Risk Profile score. Vascular function was measured by flow-mediated dilation (FMD) of the brachial artery.

Results—The average ASEX score for this sample was 3.2 (*SD* = 1.2). Regression analysis revealed that ASEX scores were predicted by greater CVD risk factors ($p = .008$, $\beta = .41$) and lower FMD ($p = .03$, $\beta = -.33$). When FMD was included in the regression model, the relationship between CVD risk factors and ASEX scores was partially attenuated ($p = .08$, $\beta = .28$).

Conclusions—ED was associated with CVD risk and impaired vascular function, although it appears that CVD risk factors may affect ED through impairment of vascular functioning.

Keywords

Erectile dysfunction; cardiovascular disease; endothelium; depression

Introduction

It is estimated that 34 million American men have some degree of erectile dysfunction (ED) (1). The most common pathophysiological factor related to ED appears to be subclinical cardiovascular disease (CVD) associated with aging, as well as CVD risk factors such as hypertension, hyperlipidemia, and diabetes mellitus (2–7), and ED is increasingly being viewed as a marker for possible underlying cardiovascular disease (CVD) (8;9). The prevalence of ED appears to be higher among patients with multiple CVD risk factors (10) or significant CVD (11), as compared to healthy controls (e.g., 65% vs 22%) (11). Furthermore, ED in otherwise health men has been observed to be a potent predictor of incident CVD in large, observational studies (12). For example, among 25 000 without CVD who were customers of a managed care organization, ED at baseline was associated with a two-fold increase in the risk for a first myocardial infarction, after adjusting for confounds

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(13). Among 4200 men without CVD or ED who were followed for up to 7 years, incident ED was associated with any cardiovascular event (Hazard Ratio = 1.27), with an effect size comparable to family history of MI (HR = 1.36), smoking (HR = 1.56), or use of antihypertensives at baseline (HR = 1.29) (14). Given these data, is it not surprising that medical providers have been encouraged to evaluate patients with ED for cardiovascular risk factors (14).

ED and CVD appear to share pathogenic mechanisms by which traditional CVD risk factors, such as cigarette smoking, diabetes, and obesity, result in a reduction in nitric oxide (NO) activity (15;16). NO availability is critical both for erectile function (17;18) and for endothelial function (15;19), and any impairment in NO activity can result in endothelial dysfunction and CVD (19). In small scale studies, endothelial dysfunction, as measured by flow-mediated dilation, has been observed to correlate with ED severity (20), and also to differentiate patients with ED from controls (21;22).

Erectile dysfunction also can be affected by psychological co-morbidities, especially the presence of clinical depression (5;10;23;24). Decreased interest in sex and other pleasurable activities has long been recognized as a classic depressive symptom (25), and epidemiologic studies have observed positive correlations between ED and depression (23). For example, among 1709 men from the Massachusetts Male Aging Study (MMAS), depression (CES-D ≥ 16) was associated with 1.82 times greater risk for moderate or severe ED, after controlling for potential confounds (24). To date, the relationship between CVD risk factors and ED has not been studied among clinically depressed patients.

We examined the relationship between CVD risk factors and vascular function in adult men with MDD. Data were obtained from baseline assessments of men enrolled in a clinical trial of exercise and depression that has been previously reported (26). We hypothesized that, among this sample of clinically depressed, sedentary, middle-aged and older men, depression severity would be related to ED severity, that traditional CVD risk factors would be related to ED severity, independent of the effects of depression, and that the relationship between CVD risk factors and ED would be explained by vascular dysfunction assessed by flow mediated dilation (FMD).

Methods

The sample consisted of 46 male participants of the SMILE Study, a randomized clinical trial of exercise and depression (26). Briefly, participants were recruited between October 2000 and November 2005. The Declaration of Helsinki protocols were followed and all study participants gave their written, informed consent. Participants included adults with scores of 12 or greater on the Beck Depression Inventory-II (BDI) (27) who met diagnostic criteria for Major Depressive Disorder (MDD) (28) using the Structured Clinical Interview for Depression (SCID) (29). Exclusion criteria included poorly-controlled diabetes mellitus, inability to participate in regular exercise, medical exclusion to taking antidepressant medication, current participation in exercise, current use of antidepressant medication, or diagnosis of bipolar disorder or psychotic depression. Psychological assessments were performed by clinicians blind to group assignment. Variables were measured as follows:

Erectile Dysfunction

Erectile Dysfunction (ED) was measured by response to a single item on the 5-item Arizona Sexual Experiences Scale (ASEX) (30), in which each item corresponds to a core element of sexual functioning (i.e., sex drive, arousal, erection, orgasm, and satisfaction). For the purposes of this study, participants responded to the question “can you easily get and keep an erection?” Response options rage from 1 = “Extremely” to 6 = “Never.” In the original

validation study, the ASEX was examined both as a series of single-time instruments, and also as an overall measure of global sexual functioning (31). Similar single item measures of ED have been shown to be reliable, valid, and sensitive to change (12).

Depression

All patients were diagnosed with MDD based upon a clinical interview administered by a trained clinical psychologist (29). Depression severity was measured with the Beck Depression Inventory-II (BDI), a self-report scale consisting of 21 items, each corresponding to a specific category of symptoms and attitudes (27). The severity of each symptom is rated on a 4-point scale, and the total score is determined by adding the sum of the individual items. The BDI has been widely used as a measure of depression severity in studies of healthy adults as well as adults with CVD (27).

CVD Risk

CVD Risk was indexed with the Framingham CVD Risk Profile (CVD-RP) (32) and body mass index. The CVD-RP uses Cox proportional-hazards regression coefficients to calculate the estimated 10-year risk for any CVD event (i.e., coronary heart disease, cerebrovascular events, peripheral artery disease, or heart failure) from sex (m/f), smoking (yes/no), diabetes (yes/no), and the natural logarithm of age, total cholesterol, high density lipoprotein (HDL), and systolic blood pressure (SBP; different coefficients are used depending on whether a patient is taking antihypertensive medication). The CVD-RP algorithm was developed from a data set consisting of nearly 8500 Framingham study participants who were followed for 12 years. Body mass index (BMI) also was included as a measure of CVD risk because, although it is not part of the CVD-RP algorithm, it is associated with CVD events, and it is also associated with ED. Data for CVD-RP and BMI calculations (i.e., height, weight, SBP, diabetes diagnosis, current use of any medications, and current smoking) were gathered from each participant by a study Physician Assistant. Lipid values were derived from fasting blood work, drawn and analyzed in an on sight, dedicated research laboratory.

Endothelial Function

Endothelial function was measured by flow-mediated dilation (FMD) of the brachial artery, using standard procedures (33). Longitudinal B-mode ultrasound images of the brachial artery, 4–6 cm proximal to the antecubital crease, were obtained using an Acuson Aspen (Mountain View, California) ultrasound platform with an 11 MHz linear array transducer. All images were obtained by the same sonographer (blinded to treatment), who had extensive experience with the FMD technique, and held the transducer manually. Images were obtained under the following conditions: (i) after 10 min of supine relaxation; (ii) during reactive hyperemia, induced following inflation for 5-minutes to supra-systolic pressure (~200 mmHg) of a pneumatic occlusion cuff placed around the forearm, and (iii) after administration of 400 μg sublingual glyceryl trinitrate (GTN) spray. End-diastolic images were stored to a magnetic-optical disk and arterial diameters were measured as the distance between the proximal and distal arterial wall intima-media interfaces using PC-based software (Brachial Analyzer Version 4.0, Medical Imaging Applications LLC, Iowa City, Iowa).

Peak hyperemic flow was assessed by Doppler velocity measurement during the first 10 seconds post-deflation of the occlusion cuff, and hyperemic flow response was defined as percent change in flow relative to resting baseline. Peak FMD response was assessed from 10 –120 seconds post-deflation of the cuff, with peak arterial diameter quantified using polynomial curve fitting, and FMD was defined as the maximum percent change in arterial diameter relative to resting baseline.

FMD has been shown to correlate with CVD risk factors (34) and is predictive of future CVD events (35).

Data Analysis

A bivariate correlation matrix was generated to examine relationships between depression severity, CVD risk, endothelial function, and erectile function. The relationship between ED and CVD risk was modeled with a regression analysis in which ASEX Item 3 score served as the independent variable and CVD-RP served as the predictor. The primary analysis consisted of a step-wise multiple-regression analysis in which the dependent variable was ASEX and the predictors were BDI, BMI, CVD-RP, and FMD, entered in that order. Because age is a component of CVD-RP, age was not included as a separate predictor in our primary analyses. All analyses were performed with SAS v 9.1 (36).

Results

Demographics

The sample consisted of 46 middle-aged (53.4 years old), moderately depressed (BDI = 27.8) men, who generally were Caucasian (73.9%), overweight (BMI = 29.8), and married (60%) (see Table 1). The average ASEX score (on the 1–6 scale) was 3.2, which falls in between “somewhat easily” and “somewhat difficult.” A total of 35% of the sample had been diagnosed with hypertension, 11% were diabetic, and 13% were smokers; 30% were taking a medication that is known to possibly interfere with erectile function (typically a beta-blocker). Half of the men in this study were non-smokers, non-diabetics, non-hypertensives, and were not taking medications with ED as a known side effect. None were taking antidepressants or Viagra. The average CVD-RP score was 14.0, which corresponds to an estimated 14% chance of having a CVD event in the next 10 years.

Correlation Analyses

A bivariate correlation matrix was constructed to examine the two-way relationships between all key variables. ED was not associated with depression severity ($r = .00$, $p = .99$). Also, the association between ED and BMI was small, and nonsignificant ($r = .19$, $p = .20$). However, greater ED was associated with higher CVD risk ($r = .42$, $p = .004$) and worse FMD ($r = -.49$, $p = .0006$). Age was strongly associated with CVD-RP ($r = .60$, $p < .0001$); however, greater ED also was associated with older age ($r = .43$, $p = .003$).

ED and CVD Risk

The relationship between erectile function and CVD risk was modeled with a regression analysis in which ED served as the independent variable and CVD-RP served as the predictor. Self-reported ED predicted CVD risk ($t = 3.1$; $p = .004$, $\beta = .42$); a 1-point increase in self-reported ED was associated with a 3.2% increase in calculated CVD risk over the next decade.

Depression, CVD Risk, Endothelial Function, and ED

The contributions of depression severity, BMI, CVD-RP, and FMD were examined with a step-wise multiple-regression analysis in which the dependent variable was ED and the predictors were BDI, BMI, CVD-RP, and FMD, entered in that order. Depression severity did not predict self-reported ED ($t = 0.0$, $p = .997$). When BMI was added to the model, it also did not predict ED ($t = 1.28$; $p = 21$, $\beta = .19$). However, when calculated CVD risk was added to the model, it was a significant and independent predictor of ED ($t = 2.76$, $p = .008$, $\beta = .41$). Furthermore, when FMD was added to the model, it was a significant predictor of ED ($t = -2.23$, $p = .03$, $\beta = -.33$), and the effect of CVD risk on ED was partially attenuated

($t = 1.81$, $p = .08$, $\beta = .28$). Taken together, these results indicate that CVD risk was a significant contributor to self-reported ED, and that this relationship was partially mediated by endothelial function.

Secondary analyses

The final model of the multiple regression analysis was repeated several times in order to examine the impact of baseline brachial artery diameter, as well as the relative impact of endothelial-dependent and endothelial-independent vasodilatation. When baseline brachial artery diameter was included in the model, it was not a significant predictor of ED ($t = .51$, $p = .60$, $\beta = .08$), and it had little impact on the contribution of CVD-RP ($t = 1.82$, $p = .08$, $\beta = .28$) or FMD ($t = -2.33$, $p = .03$, $\beta = -.35$) to ED. The contribution of FMD to ED remained essentially unchanged when the analyses were repeated excluding diabetes ($n = 41$, $p = .02$, $\beta = -.39$), and when excluding men who were taking anti-hypertensive medication ($n = 32$, $p = .04$, $\beta = -.41$).

In order to further examine the unique effects of endothelial-mediated vasodilatation, the primary regression analysis was repeated, but with nitroclycerine-mediated vasodilation (NGT, an index of endothelial-*in*dependent vasodilation) serving as the final variable, instead of FMD. When NGT was included in the final model instead of FMD, NGT contributed relatively little to ED ($t = -1.95$, $p = .06$, $\beta = -.30$), and the inclusion of NGT did not attenuate the relationship between CVD-RP and ED ($t = 2.75$, $p = .009$, $\beta = .39$). When both NGT and FMD were included in the model, FMD trended towards contributing to ED ($t = -2.0$, $p = .05$, $\beta = -.31$), whereas NGT did not ($t = -1.19$, $p = .24$, $\beta = -.19$).

Discussion

The presence of CVD risk factors were associated with greater self-reported ED severity. In addition, impaired endothelial function also was associated with greater self-reported ED, and accounted for a significant proportion of the effects of CVD risk on ED. These results are consistent with the hypothesis that ED is a marker of CVD risk, and they add further evidence to the body of data suggesting that endothelial dysfunction may underlie the relationship between ED and CVD risk. These results also extend these findings to a population of clinically depressed men.

In secondary analyses, the effect of FMD on ED was similar, regardless of whether patients with hypertension or diabetes were included in the analyses. Also, whereas the inclusion of FMD in the model attenuated the relationship between cardiovascular risk and ED, the inclusion of NGT did not. Taken together, these secondary analyses reinforce the robustness of the primary findings, and lend additional support to the hypothesis that cardiovascular risk and ED are linked at the level of the endothelium.

Neither depression severity nor BMI contributed to self-reported ED in this study. This stands in contrast to our hypotheses, as well as previous studies of patients with ED (3;5). The lack of association between depression severity and ED in this sample of clinically depressed men may indicate that it is the categorical presence of depression, and not the severity of depression, that contributes to ED. If this is true, then the correlation between depression and ED should only be observable among samples that include both depressed and non-depressed men [e.g., (3;10)], and it should not be observable in studies such as ours, which did not include non-depressed controls. Although the association between BMI and ED did not reach statistical significance, the effect sizes associated with the relationship between BMI and ED ($r = .19$ in the correlation analyses, and $\beta = .19$ in the regression analyses) suggest that our sample size may simply have been too small to detect the impact of BMI on ED.

In addition to the small sample size and the absence of a non-depressed control group, these findings are limited by the use of a single-item measure of self-reported erectile function. Also, the current findings may not generalize to a men who are younger than 40, non-obese, and/ or physically active.

Overall, these findings suggest that, even among clinically depressed adults, ED may be a sentinel for the heart and for cardiovascular disease in general. These findings should encourage medical and mental health providers to avoid attributing ED to clinical depression simply because a patient is clinically depressed. Rather, providers should consider CVD risk in all of their patients with ED.

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Conflicts of Interest

Dr. Doraiswamy has received grants and honoraria from several pharmaceutical companies. Dr. Blumenthal previously received an investigator initiated research grant from Pfizer/Eisai for an unrelated study.

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Table 1

Sample descriptive statistics

Variable	M (SD) or n (%)		Range		
*Age in years, M (SD)	53.4	(7.0)	40	to	70
Caucasian, n (%)	34	(73%)			
Married, n (%)	27	(60%)			
Beck Depression Inventory, M (SD)	27.7	(6.0)	12	to	39
Body Mass Index, M (SD)	29.8	(6.2)	15.9	to	42.4
*Diabetic, n (%)	5	(11%)			
*Smoking, n (%)	6	(13%)			
*Anti-Hypertensive Medication, n (%)	14	(30%)			
*Systolic Blood Pressure, M (SD)	123.3	(16.9)	92	to	175
*Total Cholesterol, M (SD)	202.9	(33.6)	142	to	280
*High-Density Lipoprotien, M (SD)	48.3	(12.7)	27	to	76
Framingham CVD-RP, M (SD)	14.0	(9.0)	2.9	to	42.5
Baseline Arterial Diameter, M (SD)	4.9	(0.7)	3.3	to	6.7
Flow-Mediated Vasodilation, M (SD)	4.0	(3.1)	−0.2	to	10.0
Nitroglycerine-Mediated Vasodilation, M (SD)	15.6	(5.2)	4.4		29.6
ASEX Item 3, M (SD)	3.2	(1.15)	1	to	5

*Variables included in the CVD Risk Profile calculation.

Table 2

Bivariate correlation coefficients

	ASEX	BDI	BMI	CVD-RP	Diameter	FMD	NGT
BDI	.00	.	.	.	.	.	.
BMI	.19	.09	.	.	.	.	.
CVD-RP	**.42****	−.16	.28	.	.	.	.
Diameter	.22	−.01	**.45****	.12	.	.	.
FMD	**−.49*****	−.07	−.27	**−.40****	**−.33***	.	.
NGT	**−.34***	−.15	**−.46****	−.15	**−.52*****	**.42****	.
Age	**.43****	−.01	−.03	**.60*****	.12	−.27	−.17

Notes: ASEX = Arizona Sexual Experiences Questionnaire, item 3; BDI = Beck Depression Inventory; CVD-RP = Framingham Cardiovascular Disease Risk Profile; Diameter = Baseline corotid arterial diameter; FMD = flow-mediated dilation; NGT = nitroglicerine mediated dilation;

* p<.05;

** p<.01;

*** p<.001.

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Table 3

Stepwise multiple regression analysis predicting ED

	Variable	b	SE	t	p	*β*
Step 1	BDI	0.00	0.03	0.00	1.00	0.00
Step 2	BMI	0.04	0.03	1.28	.21	0.19
Step 3	**CVD-RP**	**5.23**	**1.89**	**2.76**	**0.01***	**0.41**
Final	BDI	−0.03	0.03	−0.91	.37	−0.16
	BMI	0.00	0.03	−0.01	.99	0.00
	CVD-RP	1.14	3.17	0.36	.72	0.07
	FMD	**−0.14**	**0.07**	**−2.16**	**0.04***	**−0.41**

Notes: Dependent variable = Arizona Sexual Experiences Questionnaire, item 3; BDI = Beck Depression Inventory; CVD-RP = Framingham Cardiovascular Disease Risk Profile; Diameter = baseline corotid arterial diameter; FMD = flow-mediated dilation;

* p<.05;

** p<.01;

*** p<.001.