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Erectile Dysfunction as a Marker of Subclinical Biventricular Diastolic Dysfunction: A Prospective Echocardiographic Study

Subklinik Biventriküler Diyastolik Disfonksiyonun Bir Belirteci Olarak Erektil Disfonksiyon: Prospektif Ekokardiyografik Çalışma

ABSTRACT

Objective: Erectile dysfunction (ED) and cardiovascular disease share common vascular pathologies, particularly endothelial dysfunction and atherosclerosis. Growing evidence indicates that ED may serve as an early indicator of underlying cardiac abnormalities, particularly diastolic dysfunction (DD), even in the absence of clinically apparent cardiovascular disease.

Method: This prospective, single-center study included 87 patients with ED, matched with 53 healthy controls based on age and body mass index. The severity of ED was assessed using the International Index of Erectile Function (IIEF) and categorized as mild, moderate, or severe. Diastolic dysfunction was evaluated according to established guidelines.

Results: Patients with ED exhibited significant impairments in left ventricular diastolic function, including reduced E/A and e′ velocities, prolonged isovolumetric relaxation time (IVRT), and left atrial (LA) enlargement. A correlation was observed between the severity of ED and worsening right ventricular (RV) diastolic indices, specifically reduced RV e′ and elevated RV E/e′ ratios. Notably, LA enlargement and prolonged IVRT emerged as independent predictors of ED.

Conclusion: Erectile dysfunction is independently associated with subclinical biventricular DD, even in the absence of overt cardiovascular disease. Echocardiography may help detect subclinical cardiac dysfunction in men with ED and improve cardiovascular risk assessment.

Keywords: Diastolic dysfunction, erectile dysfunction, transthoracic echocardiography

ÖZET

Amaç: Erektil disfonksiyon (ED) ile kardiyovasküler hastalıklar, özellikle endotel disfonksiyonu ve ateroskleroz gibi benzer vasküler patolojileri paylaşmaktadır. Artan kanıtlar, klinik kardiyovasküler hastalık bulunmasa dahi, ED'nin altta yatan kardiyak anormalliklerin, özellikle diyastolik disfonksiyonun (DD) erken bir belirtisi olabileceğini göstermektedir.

Yöntem: Bu prospektif, tek merkezli çalışmada, yaş ve beden kitle indeksi açısından eşleştirilmiş 87 ED hastası ile 53 sağlıklı birey karşılaştırılmıştır. ED şiddeti, Uluslararası Erektil Fonksiyon İndeksi (IIEF) kullanılarak değerlendirilmiş ve hafif, orta ve ağır ED olarak sınıflandırılmıştır. Diyastolik fonksiyon, kılavuzlara uygun şekilde ekokardiyografi ile değerlendirilmiştir.

Bulgular: ED'li hastalarda sol ventrikül DD'sinde anlamlı bozulmalar gözlenmiştir; bunlar azalmış E/A ve e′ hızları, uzamış interventriküler gevşeme süresi (IVRT) ve sol atriyum (LA) genişlemesi ile karakterizedir. ED şiddetindeki artışla birlikte, sağ ventrikül (RV) diyastolik parametrelerinde bozulmalar (düşük RV e′, artmış RV E/e′ oranı) arasında korelasyon saptanmıştır. Özellikle LA genişlemesi ve IVRT, ED'nin bağımsız belirleyicileri olarak tanımlanmıştır.

Sonuç: ED, belirgin kardiyovasküler hastalık olmasa bile subklinik biventriküler DD ile bağımsız olarak ilişkilidir. ED'li erkeklerde transtorasik ekokardiyografi ile yapılacak değerlendirme, gizli kardiyak disfonksiyonun erken saptanmasına katkı sağlayabilir ve kardiyovasküler risk değerlendirmesini iyileştirebilir.

Anahtar Kelimeler: Diyastolik disfonksiyon, erektil disfonksiyon, transtorasik ekokardiyografi

Erectile dysfunction (ED) is characterized by the persistent inability to achieve or maintain an erection sufficient for satisfactory sexual activity, affecting nearly one-third of men over the age of 40.[1] There is a notable overlap in the risk factors for

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ED and cardiovascular disease (CVD), including advancing age, hypertension, hyperlipidemia, smoking, obesity, and diabetes. These shared risk factors suggest a common underlying vascular pathophysiology, particularly involving endothelial dysfunction and atherosclerosis.[1-3]

While atherosclerosis is a systemic condition, its clinical signs often do not appear simultaneously across different vascular territories, likely due to differences in vessel size.[4] The smaller penile arteries, for instance, which measure 1–2 mm in diameter, may become obstructed earlier and exhibit symptoms sooner than larger arteries, such as the coronary arteries.[5] Additionally, microvascular dysfunction, a hallmark of endothelial impairment, has been strongly associated with left ventricular diastolic dysfunction, an early marker of CVD.[6] However, the specific connection between ED and left ventricular diastolic dysfunction (LV DD) has not been thoroughly investigated.

Accurate evaluation of diastolic function is critical, and echocardiography is the primary non-invasive method for assessing abnormalities in cardiac relaxation.[7] Current guidelines recommend assessing cardiovascular risk in men presenting with ED, even in the absence of overt heart disease. Yet, echocardiographic evaluations are not routinely performed in these patients, despite their potential to reveal subclinical cardiac dysfunction related to endothelial health.[8] Consequently, this study aims to explore the relationship between ED and DD parameters as assessed by echocardiography.

Materials and Methods

Study Design

This was a prospective, single-center study conducted at a regional hospital. All procedures were conducted in accordance with institutional regulations and established clinical guidelines to ensure the integrity of the research. Ethical approval was obtained from University of Health Sciences Istanbul Training and Research Hospital Clinical Research Ethics Committee (Approval Number: 2504, Date: 04.09.2020). Written informed consent was obtained from each participant or their authorized representative prior to enrollment. The research protocol adhered to the ethical principles outlined in the Declaration of Helsinki (1975).

Study Population

Between October 2020 and September 2024, male participants presenting to the urology outpatient clinic with complaints of ED were evaluated for eligibility. Eligible participants were men aged 18 to 65 who reported erectile difficulties in at least 25% of sexual encounters over the previous six months and had maintained a stable sexual partnership for at least one year. Exclusion criteria included a history of significant pelvic surgery, neurological or spinal trauma, poorly controlled diabetes, end-stage renal disease, hormonal disorders such as hypothyroidism or hypogonadism, and a diagnosis of premature ejaculation. Participants with urinary tract stones, infections, malignancies, known cardiac conditions, or cardiac-related symptoms were also excluded. Men using exogenous testosterone or receiving treatment with thiazide diuretics, beta-blockers, or spironolactone were also excluded. While patients with a clinical diagnosis of hypogonadism or other hormonal disorders were excluded, routine measurements of serum total testosterone or gonadal hormones were not performed for all participants. This decision was based on the primary focus of the study being echocardiographic correlates of ED in cardiovascular asymptomatic individuals. A total of 87 eligible participants were assigned to the ED group. The control group consisted of 53 male patients with normal erectile function, presenting with unrelated complaints. Both groups were matched for age and body mass index (BMI). All participants underwent a comprehensive physical examination, including measurements of height, weight, BMI, and blood pressure. To rule out cardiovascular diseases, a standardized cardiovascular assessment was conducted by a cardiologist. This evaluation included a detailed medical history focused on symptoms such as chest pain, dyspnea, palpitations, and syncope; a 12-lead resting electrocardiogram (ECG); and transthoracic echocardiography (TTE) to assess cardiac structure and function, including left ventricular ejection fraction and valvular status. Any participant with abnormal findings suggestive of underlying cardiovascular disease was excluded from the study.

Erectile Dysfunction Evaluation

Erectile function was assessed using the International Index of Erectile Function (IIEF) score, a widely recognized, multidimensional, self-administered questionnaire that thoroughly evaluates male sexual health. The IIEF comprises 15 insightful questions covering five essential domains: erectile function, orgasmic function, sexual desire, intercourse satisfaction, and overall satisfaction. Based on the scores, individuals were categorized into distinct levels of erectile dysfunction: no erectile dysfunction (ED) (26–30), mild ED Grade 1 (17–25), moderate ED Grade 2 (11–16), or severe ED Grade 3 (0–10).[9]

Transthoracic Echocardiography

Transthoracic echocardiographic assessments, incorporating advanced two-dimensional and Doppler imaging techniques, were performed by two experienced cardiologists using the Vivid E7 imaging platform (General Electric, USA). Comprehensive evaluations of cardiac chamber dimensions were conducted

ABBREVIATIONS

A	Late diastolic transmitral velocity
BMI	Body mass index
CVD	Cardiovascular disease
DT	Deceleration time
E	Early diastolic transmitral velocity
ECG	Electrocardiogram
ED	Erectile dysfunction
HFpEF	Heart failure with preserved ejection fraction
IIEF	International Index of Erectile Function
IVRT	Isovolumic relaxation time
LA	Left atrial
LAAP	Left atrial anteroposterior diameter
LV DD	Left ventricular diastolic dysfunction
PASP	Pulmonary artery systolic pressure
RA	Right atrial
RV	Right ventricular
TR	Tricuspid regurgitation
TTE	Transthoracic echocardiography

Table 1. Demographic characteristics of the study cohort

	ED (+) (n = 87)	ED (–) (n = 53)	P
ED Grade 1	15 (17.24%)		
ED Grade 2	50 (57.47%)		
ED Grade 3	22 (25.29%)		
Age, years, kg/m^2	52 (50.7 ± 10.7)	54 (52.7 ± 10.7)	0.349
Body mass index	27.1 (28.5 ± 4.8)	26.8 (27.6 ± 4.8)	0.166
Systolic blood pressure, mmHg	130 (129 ± 15.3)	124 (127 ± 15.3)	0.394
Diastolic blood pressure, mmHg	80 (95.1 ± 93.7)	75 (74 ± 93.7)	0.005
Current smokers	56 (64.3%)	1 (2%)	< 0.001
Hypertension	46 (52.8%)	20 (37.7%)	0.082
Diabetes mellitus	29 (33.3%)	6 (11.3%)	1.000
Hyperlipidemia	24 (27.5%)	10 (18.8%)	0.243

ED, Erectile dysfunction.

through standard two-dimensional imaging. Key measurements included left atrial (LA) diameter, area, and volume; right atrial (RA) diameter, area, and volume; and pulmonary artery systolic pressure (PASP). Additionally, Tricuspid Annular Plane Systolic Excursion (TAPSE), as well as left ventricular and right ventricular (RV) diameters, volumes, and global function, along with tricuspid regurgitation (TR) jet velocity, were assessed in strict accordance with the guidelines of the American Society of Echocardiography.[10]

Early (E) and late (A) diastolic mitral inflow velocities were meticulously recorded using pulsed-wave Doppler imaging from the apical four-chamber view. To accurately assess diastolic performance, tissue Doppler imaging was employed to quantify early diastolic (e′) velocities at both the lateral and septal regions of the mitral annulus. The e′ velocity, along with the E/e′ ratio, served as powerful indicators of diastolic function, offering critical insights into left ventricular filling pressures and overall diastolic efficiency.[11]

Statistical Analysis

All statistical analyses were conducted using Jamovi software (version 2.6.22; The Jamovi Project, Sydney, Australia), with a two-tailed significance level set at $P < 0.05$. The Kolmogorov-Smirnov test was used to evaluate the normal distribution of continuous data. As the data were not normally distributed, the Mann-Whitney U test was applied to compare two independent groups. For comparisons involving more than two groups, the Kruskal-Wallis test was used, followed by the Dwass-Steel-Critchlow-Fligner procedure for post hoc analysis. Spearman correlation analysis was performed to examine relationships between continuous variables. Additionally, logistic regression analysis was employed to assess the impact of independent variables on categorical outcomes, ensuring a comprehensive understanding of the underlying dynamics.

Results

The study included a total of 140 participants, divided into a control group with 53 individuals (37.9%) and a patient group with 87 individuals (62.1%) experiencing ED, classified into grades 1, 2, and 3. Specifically, 15 individuals (17.24%) had ED grade 1, 50 individuals (57.47%) had ED grade 2, and 22 individuals (25.29%) had ED grade 3. A summary of the demographic and clinical characteristics of the participants is presented in Table 1. Statistical analysis revealed no significant differences between the ED and control groups regarding age, BMI, or systolic blood pressure ($P > 0.05$). Regarding comorbidities, hypertension was present in 52.8% of patients with ED and 37.7% of controls ($P = 0.082$). Diabetes mellitus was observed in 33.3% of the ED group compared to 11.3% in the control group ($P = 1.000$), and hyperlipidemia in 27.5% versus 18.8%, respectively ($P = 0.243$). Notably, smoking was significantly more common in the ED group (64.3%) than in controls (2%), with a statistically significant difference ($P < 0.001$). While the prevalence of traditional cardiovascular risk factors was numerically higher in the ED group, only smoking reached statistical significance (Table 1).

Patients with ED exhibited significantly increased LA and RA volumes when compared to the control group ($P < 0.05$) (Table 2). Additionally, they showed higher values in LV end-systolic diameter, LA size, deceleration time (DT), and E. Conversely, the patient group demonstrated marked reductions in E/A ratio, e′, RV E, and RV e′, while A′ was significantly elevated compared to controls. Importantly, isovolumic relaxation time (IVRT) was also significantly prolonged in the patient group, suggesting impaired DD ($P < 0.05$) (Table 2). There was no statistically significant difference in PASP between the ED group (29.2 ± 1.6 mmHg) and the control group (28.5 ± 1.4 mmHg; $P = 0.088$). Tricuspid regurgitation jet velocity was evaluated in both groups, with a mean velocity of 2.65 ± 0.35 m/s in the ED group and 2.59 ± 0.31 m/s in the control group ($P = 0.148$). Although the difference was not statistically significant, the trend toward higher TR velocities in the ED group may reflect subtle elevations in right-sided pressures associated with early DD, as shown in Table 2.

Differences between the ED grades (ED1, ED2, and ED3) were analyzed, revealing statistically significant differences in E, E/A ratio, RV E, RV e′, and the RV E/RV e′ ratio ($P < 0.05$) (Table 3). Significant variations in E values were noted between ED1 and ED3; in RV E between the control group and ED1 and ED2; in RV e′ between ED1 and ED2 and ED3; and in the RV E/RV e′ ratio between ED1 and ED2 and ED3 ($P < 0.05$).

Table 2. Comparative analysis of echocardiographic variables between study groups

	ED (+) (n = 87)		ED (-) (n = 53)		P
	Mean ± SD	Median (IQR)	Mean ± SD	Median (IQR)	
LVEF, %	61.13 ± 2.98	60 (3)	60.69 ± 4.48	60 (3)	0.554
LVEDD, mm	47.48 ± 4.33	47 (6.5)	47.81 ± 5.24	47 (8)	0.532
LVESD, mm	35.61 ± 21.39	33 (5)	31.6 ± 5.57	30 (8)	0.023*
IVSD, mm	10.97 ± 1.88	11 (2.5)	10.47 ± 1.64	10 (1)	0.155
LVPWD, mm	10.24 ± 1.27	10 (2)	10 ± 1.48	10 (2)	0.144
LAAP, mm	42.11 ± 6.89	40 (11)	37.99 ± 5.13	38 (5)	0.001*
LAA, cm^2	16.67 ± 3.2	16.6 (3.6)	17.84 ± 3.97	17.2 (3.7)	0.099
LAV, mL	40.68 ± 4.48	40 (5)	37.4 ± 5.34	37 (6.5)	0.0001*
LAVI, mL/m^2	21.99 ± 2.42	21.5 (2.7)	20.22 ± 2.89	20 (3.5)	0.0002*
E, cm/s	60.92 ± 16.27	59 (21.5)	78.53 ± 21.54	78 (24)	0.0001*
A, cm/s	67.61 ± 15.89	66 (22)	66.94 ± 17.24	64 (23)	0.747
RV MID, mm	29.76 ± 4.63	30 (6)	29.79 ± 3.85	31 (5)	0.680
RV annular, cm^2	35.78 ± 5.04	36 (7)	37.3 ± 3.21	37 (3)	0.063
RAA, cm^2	15.01 ± 3.43	14.7 (4.85)	15.7 ± 3.24	15.2 (4.1)	0.146
RAV, mL	40.06 ± 3.85	39 (6)	35.77 ± 4.79	35 (7)	0.0001*
RA, mm	47.17 ± 5.08	47 (7)	48.62 ± 4.67	49 (7)	0.059
TAPSE, mm	23.46 ± 3.56	23 (5)	24 ± 3.25	24 (4)	0.337
PASB, mmHg	29.2 ± 1.6	29 (3)	28.5 ± 1.4	28 (2.5)	0.088
TR, m/s	2.65 ± 0.35	2.60	2.59 ± 0.31	2.55	0.148
Aortic flow, m/s	1.14 ± 0.24	1.1 (0.32)	1.15 ± 0.21	1.08 (0.26)	0.942
IVRT, ms	110.28 ± 47.83	100 (65.5)	73.49 ± 23.42	69 (33)	0.0001*
DT, ms	125.46 ± 56.36	115 (57)	123.79 ± 42.33	114 (57)	0.737
E/A ratio	0.94 ± 0.31	0.86 (0.49)	1.28 ± 0.54	1.34 (0.91)	0.0001*
e’	13.7 ± 10.5	11 (5.5)	14.11 ± 5.83	14 (8)	0.022*
a'	10.51 ± 3.35	11 (4)	9.19 ± 2.95	9 (5)	0.008*
E/e’ ratio	5.75 ± 3.03	5.2 (1.93)	6.34 ± 3.4	5.33 (1.96)	0.457
RV E	52.29 ± 33.17	50 (15)	60.26 ± 15.68	60 (17)	0.0001*
RV e’	13.49 ± 7.73	12 (5.5)	13.91 ± 3.4	15 (4)	0.003*
RV E/RV e’ ratio	4.81 ± 3.14	4.25 (2.46)	4.53 ± 1.38	4.18 (1.15)	0.997
RV A	50.52 ± 17.18	48 (17)	47.94 ± 15.91	42 (24)	0.343
RV a'	14.31 ± 4.04	14 (4)	14.23 ± 3.95	13 (5)	0.749

*Statistically significant (Mann-Whitney U test). A (cm/s), Late Diastolic Mitral Inflow Velocity; a', Late Diastolic Mitral Annular Velocity (Tissue Doppler); DT, Deceleration Time; E (cm/s), Early Diastolic Mitral Inflow Velocity; é, Early Diastolic Mitral Annular Velocity (Tissue Doppler); EF, Ejection Fraction; IVRT, Isovolumetric Relaxation Time; IVSD, Interventricular Septal Diameter; LAA, Left Atrial Area; LAAP, Left Atrial Anteroposterior Diameter; LAV (mL), Left Atrial Volume; LAVI (mL/m2), Left Atrial Volume Index; LVEDD, Left Ventricular End-Diastolic Diameter; LVESD, Left Ventricular End-Systolic Diameter; LVPWD, Left Ventricular Posterior Wall Diameter; PASB (mmHg), Pulmonary Artery Systolic Pressure; RA, Right Atrial Diameter; RAA, Right Atrial Area; RAV (mL), Right Atrial Volume; RV A, Right Ventricular Late Diastolic Inflow Velocity; RV a', Right Ventricular Late Diastolic Annular Velocity; RV Annular, Right Ventricular Annular Diameter; RV E, Right Ventricular Early Diastolic Inflow Velocity; RV é, Right Ventricular Early Diastolic Annular Velocity; RV E/RV é, Ratio of RV Early Diastolic Inflow to Annular Velocity; RV Mid, Right Ventricular Mid-Cavity Diameter; TAPSE, Tricuspid Annular Plane Systolic Excursion; TR (m/s), Tricuspid Regurgitation Jet Velocity.

To evaluate the impact of variables that showed significant differences between groups, logistic regression analysis was conducted. In the univariate logistic regression, left atrial anteroposterior diameter (LAAP), LA and RA volumes, and IVRT demonstrated a statistically significant positive association with the patient group compared to controls ($P < 0.05$). In the multivariate logistic regression analysis, LAAP and IVRT remained significantly associated with the patient group ($P < 0.05$) (Table 4).

Figure 1 illustrates representative echocardiographic findings from a patient with erectile dysfunction. Panel A, obtained in the parasternal long-axis view, reveals left atrial enlargement. Panel B shows a reduced E/A ratio on mitral inflow Doppler, consistent with impaired diastolic filling. Tissue Doppler imaging of the mitral annulus in Panel C (lateral) and Panel D (septal) demonstrates reduced e’ velocities, further indicating diastolic dysfunction. Panels E and F present left atrial area and volume measurements,

Table 3. Comparative analysis of echocardiographic parameters based on erectile dysfunction severity

	Mild ED (n = 15)		Moderate ED (n = 50)		Severe ED (n = 22)		P*	P**
	Mean ± SD	Median (IQR)	Mean ± SD	Median (IQR)	Mean ± SD	Median (IQR)		
E	55.27 ± 13.2	52 (21)	61.56 ± 18.09	18.09 (25.5)	63.32 ± 13.26	59 (14)	< 0.001*	01; 03
A	64.6 ± 16.15	64 (25)	68.3 ± 16.61	16.61 (19.8)	68.09 ± 14.44	67.5 (19)	0.838	
E/A ratio	0.9 ± 0.31	0.75 (0.26)	0.94 ± 0.33	0.33 (0.52)	0.97 ± 0.26	0.89 (0.48)	0.005*	02
e′	19.4 ± 17.8	11 (6.5)	13.06 ± 9.27	9.27 (6)	11.27 ± 3.06	11 (2.75)	0.099	
a'	10.93 ± 2.31	11 (2.5)	10.18 ± 3.58	3.58 (5)	10.95 ± 3.46	11 (5)	0.055	
E/é ratio	4.44 ± 1.99	5.11 (1.4)	6.01 ± 3.41	3.41 (2.28)	6.05 ± 2.51	5.38 (1.6)	0.384	
RV E	37.87 ± 15.83	39 (16.5)	53.08 ± 31.55	31.55 (16)	60.32 ± 42.4	52 (16.8)	< 0.001*	01; 02; 12; 13
RV e′	18.4 ± 11.41	14 (7.5)	13.12 ± 7.31	7.31 (5.75)	10.97 ± 3.04	10 (4)	0.001*	02; 03
RV E/RV e′ ratio	2.9 ± 1.59	2.79 (1.45)	4.96 ± 3.01	3.01 (2.34)	5.78 ± 3.72	4.42 (3.08)	0.004*	01; 12; 13
RV A	52.6 ± 13.6	48 (16.5)	51.2 ± 20.2	20.2 (18.8)	47.55 ± 10.98	48 (12)	0.644	
RV a'	14.6 ± 2.23	14 (2)	14.28 ± 4.97	4.97 (6)	14.19 ± 2.37	14.5 (2.75)	0.961	

*Kruskal–Wallis Test; **Dwass–Steel–Critchlow–Fligner pairwise comparisons. A, Peak Late Diastolic Transmitral Flow Velocity; a′, Peak Late Diastolic Mitral Annular Velocity Measured by Tissue Doppler; E, Peak Early Diastolic Transmitral Flow Velocity; e′, Peak Early Diastolic Mitral Annular Velocity Measured by Tissue Doppler; e/a, Ratio of Early to Late Diastolic Transmitral Flow Velocities; E/e′, Ratio of Early Transmitral Flow Velocity to Early Diastolic Mitral Annular Velocity; RV A, Peak Late Diastolic Tricuspid Inflow Velocity; RV a′, Peak Late Diastolic Tricuspid Annular Velocity Measured by Tissue Doppler; RV E, Peak Early Diastolic Tricuspid Inflow Velocity; RV e′, Peak Early Diastolic Tricuspid Annular Velocity Measured by Tissue Doppler; RV E/RV e′, Ratio of Early Tricuspid Inflow to Early Diastolic Tricuspid Annular Velocity.

Table 4. Univariate and multivariate logistic regression analysis

	Univariate analysis		Multivariate analysis	
	Exp (B) (95% CI)	P	Exp (B) (95% CI)	P
LVESD	1.07 (1.00–1.15)	0.050	–	–
LAAP	1.12 (1.05–1.19)	0.001*	1.16 (1.04–1.29)	0.006*
LAV	0.88 (0.81–0.95)	0.001*	–	–
e′	0.99 (0.96–1.03)	0.792	–	–
a'	1.14 (1.02–1.27)	0.022	–	–
RV E	0.99 (0.98–1)	0.138	–	–
RV e′	0.99 (0.94–1.04)	0.708	–	–
RAV	0.93 (0.9–0.95)	0.0001*	–	–
IVRT	1.03 (1.02–1.05)	0.0001*	1.04 (1.01–1.06)	0.002*

*Statistically significant (P < 0.05). a′, Peak Late Diastolic Mitral Annular Velocity Measured by Tissue Doppler; e, Peak Early Diastolic Transmitral Flow Velocity; e/a, Ratio of Early to Late Diastolic Transmitral Flow Velocities; é, Peak Early Diastolic Mitral Annular Velocity Measured by Tissue Doppler; IVRT, Isovolumetric Relaxation Time; LAAP, Left Atrial Anteroposterior Diameter; LAV, Left Atrial Volume; LVESD, Left Ventricular End-Systolic Diameter; RAV, Right Atrial Volume; RV e, Peak Early Diastolic Tricuspid Inflow Velocity; RV é, Peak Early Diastolic Tricuspid Annular Velocity Measured by Tissue Doppler.

respectively, highlighting atrial remodeling. These findings reflect structural and functional left heart changes in ED patients, even in the absence of overt cardiovascular disease.

Discussion

In this study, we found significant impairments in LV DD and elevated RV filling pressures in patients with ED. Echocardiographic evaluations of individuals without known CVD, who presented to the urology outpatient clinic with complaints of ED, revealed that LA dilatation, increased LA volume, prolonged IVRT, and a reduced E/A ratio were significantly more prevalent in the ED group compared to the control group. These echocardiographic changes indicate potential subclinical DD in patients with ED. Furthermore, a subgroup analysis within the ED cohort showed that greater severity of ED was associated with a significant decrease in RV é velocity and an increase in the RV E/é ratio. The observed increase in RA volume further supports the relationship between higher ED severity and elevated subclinical RV diastolic dysfunction.

Erectile dysfunction is increasingly recognized as a manifestation of systemic vascular disease, particularly atherosclerosis.[12,13] Compelling evidence indicates that ED frequently precedes overt cardiovascular events, reflecting underlying endothelial dysfunction and arterial insufficiency.[14] The unique characteristics of the penile arteries, due to their smaller caliber, allow atherosclerotic changes to manifest earlier than in larger vessels such as the coronary arteries, positioning ED as a potentially significant early marker of subclinical CVD. Atherosclerosis is fundamentally a systemic condition that affects multiple vascular territories and is characterized by similar pathological alterations

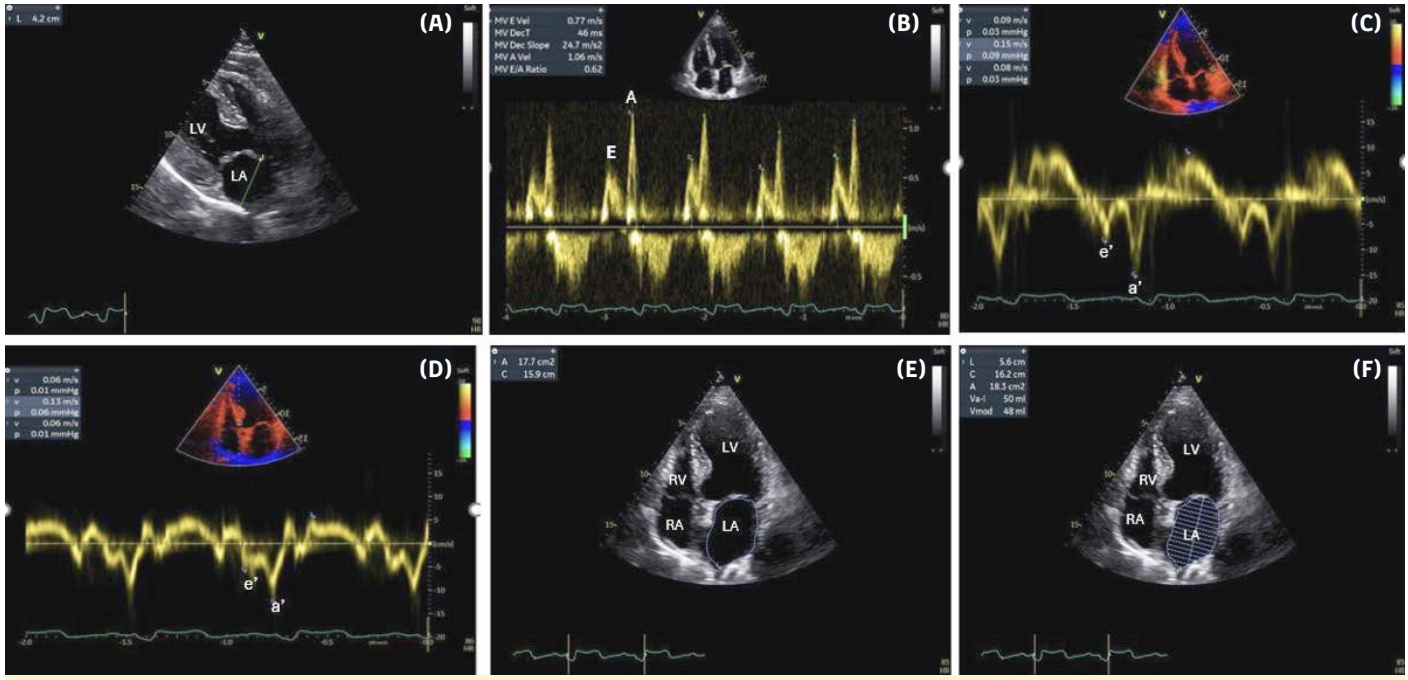

Figure 1. Echocardiographic assessment of left atrial size and left ventricular diastolic function in a patient with erectile dysfunction. *(A) Parasternal long-axis view showing marked left atrial (LA) enlargement posterior to the left ventricle (LV). (B) Pulsed-wave Doppler recording of mitral inflow velocities demonstrating reduced early (E) to late (A) diastolic filling ratio (E/A = 0.62), indicating impaired diastolic relaxation. (C) Tissue Doppler imaging (TDI) from the lateral mitral annulus showing reduced early diastolic annular velocity (e′) and relatively preserved late diastolic velocity (a′), consistent with abnormal LV relaxation. (D) TDI from the septal mitral annulus showing similarly decreased e′ and elevated a′, reinforcing evidence of diastolic dysfunction. (E) Apical four-chamber view with manual planimetry outlining left atrial area (LA), alongside right atrium (RA), right ventricle (RV), and left ventricle (LV). (F) Biplane method of disks for left atrial volume quantification, highlighting enlarged LA dimensions.

LA, Left atrium; LV, Left ventricle; RA, Right atrium; RV, Right ventricle; E, Peak early diastolic mitral inflow velocity; A, Peak late diastolic mitral inflow velocity; e′, Peak early diastolic mitral annular velocity (TDI); a′, Peak late diastolic mitral annular velocity (TDI); TDI, Tissue Doppler imaging.

in the arterial wall.[15] As a result, individuals with arterial ED often face an elevated risk of CVD, even in the absence of overt cardiac symptoms. It is important to note that the clinical onset of symptoms across different vascular beds may not occur simultaneously, primarily due to variations in arterial diameter. Larger arteries tend to be more resilient to comparable degrees of vascular wall thickening than their smaller counterparts.

Moreover, increased arterial stiffness—a common consequence of atherosclerosis—leads to elevated systolic blood pressure and widened pulse pressure.[16] These hemodynamic changes raise myocardial oxygen demand, contribute to LV hypertrophy, and increase myocardial wall stress, ultimately resulting in myocardial ischemia. Collectively, these processes severely impair LV diastolic function, which plays a central role in the development of heart failure with preserved ejection fraction (HFpEF). Impaired myocardial relaxation and increased ventricular stiffness elevate filling pressures, leading to clinical manifestations such as dyspnea, reduced exercise capacity, higher risk of hospitalization, and decreased overall survival. This highlights the importance of early identification of DD, which is vital for effective risk stratification and clinical management.[17,18]

In this study, we systematically assessed diastolic function using key echocardiographic parameters, specifically the E/A ratio and IVRT. Our findings reveal significant impairments in these measures among patients with ED. The E/A ratio, which reflects the dynamic interplay between E and A ventricular filling, serves as a crucial indicator of diastolic function that evolves with disease progression.[19] In the early stages, delayed relaxation presents as a decreased E/A ratio. As DD progresses, pseudo-normalization or even an elevated ratio may occur, resulting from reduced ventricular compliance and altered filling mechanics.

In our patient cohort, we observed a statistically significant reduction in the E/A ratio among those with ED, indicating early-stage DD—even in the absence of overt CVD. Furthermore, IVRT—though influenced by factors such as heart rate and blood pressure—provides valuable insights into myocardial relaxation.[20] It becomes especially informative when interpreted alongside other diastolic indices. In our findings, patients with ED exhibited a notably prolonged IVRT, further reinforcing the presence of subclinical DD.

Moreover, it is essential to recognize that myocardial relaxation and compliance can be significantly influenced by age and metabolic comorbidities, including obesity, insulin resistance, and hypertension—all of which frequently coexist in individuals with ED.[21-23] Importantly, IVRT emerged as an independent variable in our logistic regression analysis of patients with ED, reinforcing its importance in understanding the complex relationship between erectile dysfunction and DD.

Left atrial enlargement also emerged as a robust and independent predictor of ED in this study, underscoring its significance in cardiovascular health. LA enlargement is a clear indicator of atrial remodeling—a phenomenon closely associated with CVD and structural changes in the LV, as supported by previous research.[24] Numerous studies have demonstrated the strong interplay between elevated blood pressure, LV hypertrophy, and atrial size. Conditions such as hypertension and LV hypertrophy can accelerate the onset of diastolic dysfunction, leading to increased filling pressures and subsequent atrial remodeling due to persistent pressure overload. Importantly, LA volume has been proposed as a key marker of both the severity and chronicity of DD, offering a potentially simpler alternative to more complex Doppler echocardiographic assessments of diastolic function and filling pressures. The size of the LA provides valuable insights into the long-term effects of elevated filling pressures. Notably, Nishimura et al.[25] established a direct correlation between elevated LA pressure and the presence of DD, while Matsuda et al.[26] demonstrated that maximal LA volume increases in parallel with the severity of DD, as measured through invasive hemodynamic techniques. Moreover, the progression of DD is linked to increased filling pressures, which not only lead to LA enlargement but also to notable increases in both LA and RA volumes. In our cohort, the statistically significant increases in both LA and RA volumes among patients with ED strongly support the presence of subclinical DD within this population.[27] Despite observing significant differences in individual diastolic parameters such as e′ velocity and IVRT, our study did not demonstrate a statistically significant difference in the E/e′ ratio between the ED and control groups. One plausible explanation is that E/e′, although widely used as a surrogate marker for estimating left ventricular filling pressures, may have limited sensitivity in detecting early stages of DD.[28] This finding is consistent with the results of Hyun et al.,[27] who also reported no statistically significant difference in E/e′ values between ED and control groups in a cardiovascular disease-free population. Similarly, von Bibra et al.[22] showed that in non-diabetic individuals, E/e′ did not increase significantly despite impaired myocardial relaxation, possibly due to early compensatory mechanisms such as volume adaptation that mask elevations in filling pressure. These findings highlight the potential limitations of using E/e′ in isolation to detect early diastolic dysfunction in asymptomatic populations. Instead, parameters such as IVRT, LA volume, and e′ velocity—each of which demonstrated significant differences in our ED group—may serve as more sensitive indicators of subclinical myocardial involvement.[16,27]

Our study underscores the clinical relevance of diastolic function in patients with ED, complementing the more widely recognized LV involvement. Subgroup analysis revealed a significant decline in RV e′ velocity and a rise in the RV E/e′ ratio with increasing ED severity, indicating impaired RV relaxation and elevated filling pressures. These findings were consistent across all ED severity groups and emphasize the utility of tissue Doppler imaging (TDI) in evaluating subclinical RV dysfunction. Prior studies have similarly noted that systemic endothelial dysfunction and vascular pathology can extend to the right heart, supporting a biventricular pathophysiological continuum.[29,30] Our findings suggest a progressive deterioration in RV diastolic function with increasing ED severity, supporting the integration of RV assessment into cardiovascular evaluation in this population.

Our findings support and expand upon prior research linking ED to early DD. Consistent with the studies by Hyun et al.[27] and von Bibra et al.,[22] we found no significant difference in E/e′ ratios between ED and control groups without overt cardiovascular disease, underscoring the limited sensitivity of this index in detecting early-stage DD. In contrast, prolonged IVRT, reduced e′ velocity, and increased LA volume were strongly associated with ED. These results align with the observations of Matsuda et al.[26] and Nishimura et al.,[25] who identified LA enlargement as a marker of chronically elevated filling pressures. The independent predictive value of IVRT and LA size in our multivariate analysis further reinforces their clinical relevance. Furthermore, we observed a significant decline in RV e′ and a rise in RV E/e′ with increasing ED severity, indicating RV diastolic impairment. These findings are consistent with those of Mukhaini et al.[29] and Arısoy et al.,[30] who demonstrated the utility of RV tissue Doppler indices in identifying subclinical RV dysfunction. Collectively, our results highlight the presence of biventricular DD in ED and support the use of echocardiography for early cardiovascular risk assessment in this population.

Given the demonstrated association between ED and subclinical biventricular DD, our findings support the consideration of echocardiographic evaluation in men presenting with ED, particularly when traditional cardiovascular risk factors such as hypertension, diabetes mellitus, obesity, or smoking are present—even in the absence of overt cardiac symptoms. Routine assessment using transthoracic echocardiography, including tissue Doppler parameters and LA volume measurements, may enable early identification of diastolic abnormalities. This proactive approach could facilitate timely cardiovascular risk stratification, guide preventive strategies, and potentially alter the clinical course for individuals at heightened risk of future cardiac events.

It is important to note that smoking, a well-established contributor to endothelial dysfunction and atherosclerosis, was significantly more prevalent in the ED group compared to controls (64.3% vs. 2%, $P < 0.001$). Cigarette smoking impairs endothelial nitric oxide production, increases oxidative stress, and promotes systemic inflammation, all of which contribute to vascular stiffness and microvascular dysfunction.[1,15] These pathophysiological mechanisms underlie both ED and early DD. Moreover, smoking has been independently associated with LV hypertrophy, increased myocardial wall stress, and left atrial remodeling—key contributors to DD.[16] Therefore, while our findings demonstrate a clear association between ED and subclinical biventricular DD, smoking may act as a common upstream pathological factor. This reinforces the importance of controlling for smoking status in future studies evaluating cardiac function in men with ED.

Limitations

This study has several limitations. First, it was conducted at a single center with a relatively small sample size, which may limit the generalizability of the findings. Although patients with known cardiovascular disease were excluded, the presence of undiagnosed or subclinical disease cannot be ruled out. The cross-sectional design also precludes causal inferences regarding the relationship between ED and DD. Advanced imaging techniques, such as LA strain or RV strain analysis, were not employed, which may have reduced the sensitivity for detecting early myocardial dysfunction. Additionally, RV diastolic assessment

was based solely on tissue Doppler imaging, which may not fully capture the complex geometry and function of the RV. While individuals with known hormonal disorders were excluded, serum testosterone and other endocrine parameters were not routinely measured, potentially overlooking hormonal contributions. Lastly, variables such as physical activity, psychological status, and lifestyle factors were not systematically recorded and may have influenced both erectile and cardiac function.

Conclusion

This study reveals a connection between ED and echocardiographic markers of LV DD in individuals without established CVD. Notably, the severity of ED is significantly associated with RV DD in this population. These findings underscore the critical importance of evaluating ED as a potential early indicator of subclinical cardiac dysfunction, suggesting that it may serve as a vital precursor to future cardiovascular disease.

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