Hindawi
Andrologia
Volume 2024, Article ID 6151669, 10 pages
https://doi.org/10.1155/2024/6151669

Research Article

Prevalence of Erectile Dysfunction among Patients with Coronary Artery Disease

Islam Mohamed Abd Elsamie,[1] **Hassan Abou Khodair**,[1] **Mohamed Sayed Bashandy**,[2] **and Hany Aboelwafa**[1]

[1]*Faculty of Medicine, Department of Dermatology and Venereology, Al-Azhar University, Damietta, Egypt*
[2]*Faculty of Medicine, Department of Cardiology, Al-Azhar University, Damietta, Egypt*

Correspondence should be addressed to Islam Mohamed Abd Elsamie; abdelsamiemohamed379@gmail.com

Received 17 June 2023; Revised 8 October 2023; Accepted 21 December 2023; Published 25 March 2024

Academic Editor: Raul Sanchez

Both coronary artery disease (CAD) and erectile dysfunction (ED) are widespread illnesses that frequently coexist. Numerous triggering risk factors and pathophysiological mechanisms contribute to both diseases. Penile color Doppler ultrasound can serve as a crucial tool to evaluate penile arterial blood flow. Therefore, this study aims to assess the ED prevalence in CAD patients, to determine the socioeconomic and clinical profiles of the population, and to define risk factors of their development. The cross-sectional study included 100 male patients who were admitted to the coronary angiography (CAG) unit. Patients were asked if they had ED, and if present, it was graded using the International Index of Erectile Function 5. Right and left cavernosal arteries were examined using color Doppler and erection hardness to assess the peak systolic velocity (PSV) and end-diastolic velocity. Among the 52 cases that suffered from ED, most of the cases had mild grades (38.5%). All cases without ED (100%) had penile artery Doppler PSV findings ≥30 (cm/s) with an average Doppler PSV of 48.02 ± 7.49 cm/s for the right cavernosal arteries and 47.4 ± 7.17 cm/s for the left cavernosal arteries. Yet, 71.2% of cases with ED had penile artery Doppler PSV findings <30 (cm/s) with an average Doppler PSV of 29.1 ± 7.57 cm/s for the right cavernosal arteries and 28.69 ± 7.11 cm/s for the left cavernosal arteries. Furthermore, by CAG, 50% of cases without ED had blockage not necessitating a coronary intervention (no significant disease) compared to 25% of cases with ED. There was positive correlation between advanced grade of ED and age ($r = 0.327$, $P < 0.001$), hypertension ($r = 0.213$, $P = 0.033$), DM ($r = 0.253$, $P = 0.011$), smoking ($r = 0.202$, $P = 0.044$), and severity of CAD ($r = 0.332$, $P < 0.001$). Patients with multivessel CAD have a significantly higher incidence and more severity of ED. Moreover, pathological penile vessels PSV (<25 or <30 cm/s) via Doppler ultrasound is mostly valuable as a noninvasive tool to define ED attributable to vascular impairment that may be antecedent to atherosclerosis in the coronary vessels.

1. Introduction

Erectile dysfunction (ED) is outlined as the consistent or recurrent inability of the male to achieve or maintain a penile erection adequate for satisfactory sexual performance. Sexual intimacy withdrawal, less productivity at work, and a lower quality of life are all associated with ED. Employers are negatively impacted because males with ED have higher absenteeism rates that can be attributed to psychosocial factors, which have an adverse effect on job productivity [1].

Aberrant sexual function has been described in vascular disease patients, for instance, hypertension, coronary artery disease (CAD), diabetes, cerebrovascular disease, as well as peripheral arterial disease. ED is similar to vascular diseases in the pathophysiology, enlightened by anatomical or functional abnormalities "Endothelial dysfunction" [2].

CAD and ED are widespread diseases that frequently coexist. Numerous triggering risk factors may lead to ED and CAD, including age, smoking, dyslipidemia, obesity, hypertension, diabetes, and a sedentary lifestyle [3].

The idea that ED is a sentinel indication of vascular disease has been put forth in numerous ways. The artery size theory maintains that ED is a former symptom of systemic atherosclerosis. Assuming that atherosclerosis develops in all

main circulatory beds at roughly the same rate, symptoms would appear sooner in smaller arterial branches, such as the penile artery, than they would in the heart and limbs, which are capable of resisting the same atherosclerosis or blockage grade [4].

Furthermore, normal male erection is a vascular event that depends on endothelial-derived nitric oxide to promote vasodilation. Given that atherosclerosis is primarily a systemic disease, it is expected that CAD patients will develop penile atherosclerosis and ED eventually [5].

There is no clinical sign, laboratory test, or noninvasive procedure efficient for distinguishing all patients who will have CAD. Nonetheless, ED can serve as an early clinical indicator for CAD occurrence. Moreover, the grade of ED can be allied to the severity of CAD in the abundant proportion of males [6].

Although ED and CVD frequently co-occur and have similar pathophysiological mechanisms, there is some consensus over whether the presence of ED should require further cardiovascular testing or improve CVD prediction beyond the standard risk variables. However, the application of ED as an innovative indicator of CAD is still investigated. ED likewise may serve as a marker of CVD severity [4].

Furthermore, penile color Doppler ultrasound (PCDU) represents the first-line investigation for ED diagnosis. In fact, PCDU is a crucial test to evaluate arterial blood flow of the penis and veno-occlusive mechanisms. Moreover, it can reveal structural changes in the cavernous artery walls, for example, the existence of atherosclerotic plaques or increased intima-media thickness [7].

Therefore, this study aims to (1) assess the ED prevalence in CAD patients, (2) determine the socioeconomic and clinical profiles of the population, and (3) define risk factors of their development.

2. Patients and Methods

2.1. Study Protocol. Our cross-sectional study included 100 married male patients aged 18 years or older with active sexual life potential who were submitted to the coronary angiography (CAG) unit with a history of chest pain during the preceding 24 hr prior to admission and ECG changes indicating coronary artery ischemia plus or minus positive cardiac enzymes at Department of Cardiology, Faculty of Medicine, Al Azhar University, Damietta, Egypt from October 1, 2021 till the end of July 2022. Informed consent was obtained from each participating case in this research after giving them the full information about the study. Exclusion criteria included patients with (1) neurological, psychiatric, or endocrinological diseases; (2) history of prostatic, urethral, or pelvic surgeries; (3) history of revascularization in the myocardium or lower limb; (4) therapy for ED, peripheral artery disease, or the aorta; and (5) severe blood dyscrasia.

2.2. Study Methodology. A thorough history, including age, diabetes, cigarette smoking, hypertension, dyslipidemia, and drug history, was taken from each included patient with stress on both cardiovascular and ED risk factors, as well as complete general, chest, and abdominal examination.

2.2.1. CAG Analysis. It was carried out by the cardiologist, who was unaware of the patient's ED status. According to the outcome of CAG cases were categorized as follows:

(i) No disease: normal or nonsignificant CAD with a blockage that does not require a coronary intervention.
(ii) Single-vessel disease (SVD).
(iii) Double-vessel disease (DVD).
(iv) Triple-vessel disease (TVD).

2.2.2. Erectile Function Assessment. The patients received explanations regarding the similarities in the risk factors between CVD and ED, as well as the function of ED as a surrogate marker of the severity of endothelial dysfunction.

Then, the presence of ED was assessed and graded by means of an Arabic-translated International Index of Erectile Function 5 (IIEF-5) Questionnaire. The questionnaire includes five questions with a score from 0 to 5 for a total score of 5–25 [8]. The assessment focused on sexual activity over the previous 6 months without the use of any medicines to enhance sexual performance. The cut point of ED diagnosis was set to an overall score was $\leq$21. ED was graded into five severity levels according to IIEF-5 score: none: 22–25, mild: 17–21, mild-moderate: 12–16, and moderate: 8–11.

(1) Penile Doppler Procedure for Cases with ED [9]. All examinations were carried out through the GE Voluson E6 device (GE Medical Systems, Milwaukee, WI, USA) and VINNO X1 (Suzhou) Co., Ltd. China, using a color Doppler ultrasound and a high-frequency linear transducer (7.5–18 MHz) probe. The transducer was positioned using transmitter gel on the penis base. The examination by means of a high-frequency linear transducer (7.5–18 MHz) started with a flaccid penis. The whole penis was scanned over cross-sectional and longitudinal images. At the starting point, Doppler mode was turned on, and the angle ($<60°$), the peak systolic velocity (PSV), and end-diastolic velocity (EDV) of the right and the left cavernosal arteries were recorded before injection. The injection site was squeezed for 10 s to prevent agent reflux to surface planes before administering an intracavernosal injection of prostaglandin E1 (PGE1) using a syringe (0.5–3 mL) with an insulin needle (27–30 gauge, 0.5-in). The medication injection time was then recorded. Regarding the medication dosage, 12 μg prostaglandin E1 was injected into cases younger than 30 years, where 16 μg were administered to patients 60 years or older, and patients in-between these mentioned ages were administered 20 μg of PGE1.

After that, the PSV and EDV at the peak of the erection were measured at 5, 10, 15, and 20 min, given that the hardness is evaluated at each subsequent innovative assessment time. The case was requested to wait for between 30 and 60 min after the examination to assess potential side effects brought on by the medication, such as discomfort, prolonged, painful erection, and priapism in the injection site. As mentioned by literature, following the appropriate pharmacological stimulus, a PSV greater than 30 cm/s indicates

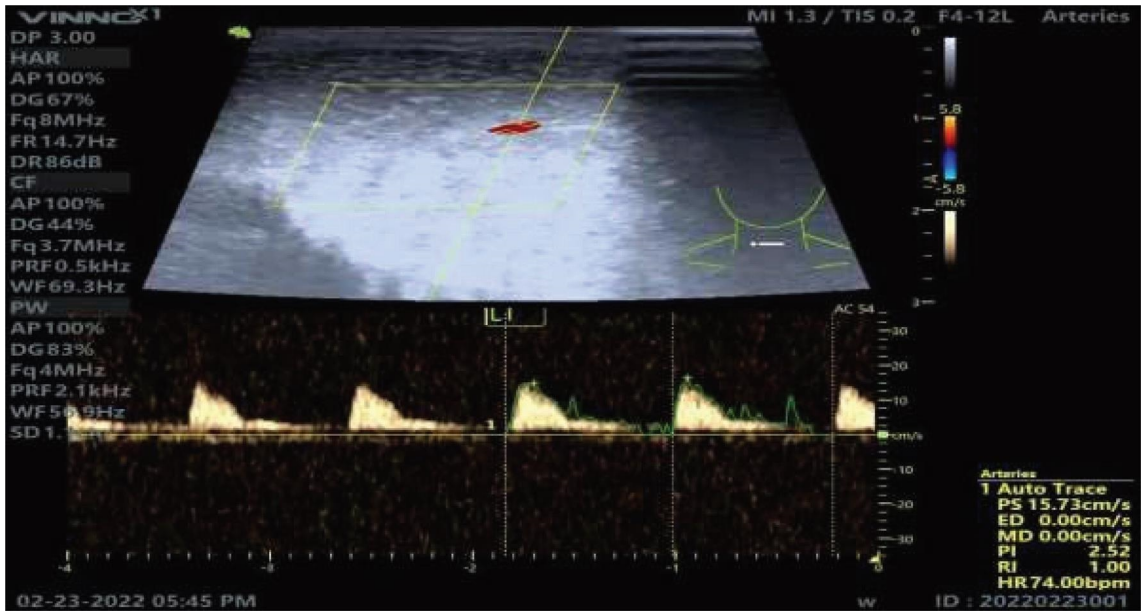

Figure 1: Penile artery Doppler for male case aged 65 years old hypertensive for 7 yr. Coronary angiography showed severe double vessel affection. ILFE-5 score was 10. On penile artery Doppler; PSV was 15.73 cm/s.

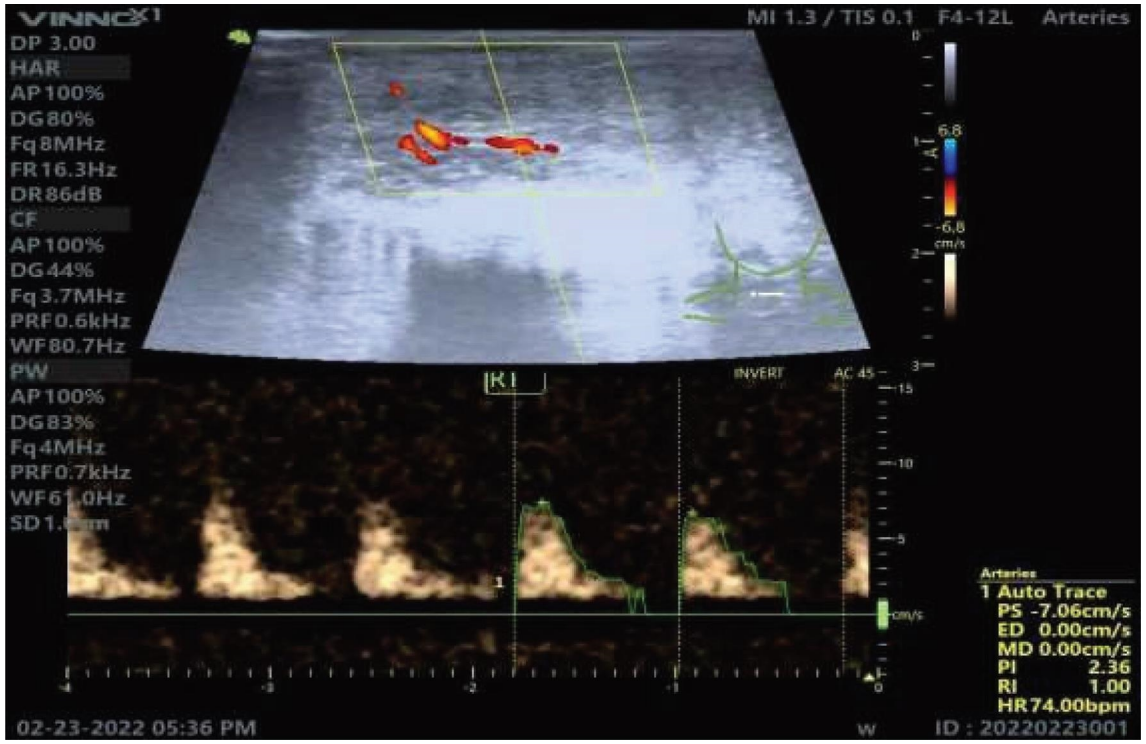

Figure 2: Penile artery Doppler for male case aged 68 years old diabetic for 6 yr and hypertensive for 10 yr. Coronary angiography showed severe double vessel affection. ILFE-5 score was 7. On penile artery Doppler; PSV was 7.06 cm/s.

normal arterial flow; however, a PSV lower than 25 cm/s indicates arterial insufficiency (Figures 1 and 2), operating 30 cm/s as a standard PSV cutoff [7].

(2) Erection Grading of the Penis. Using the erection hardness grading score, penile erections were graded at 10 min intervals [10]. 0: Penis does not enlarge; 1: increasing penis size without hardness; 2: mild hard erection but is not sufficient to penetrate; 3: incomplete hardness but sufficient for penetration; 4: complete rigid and hard erection.

2.3. Sample Size Justification. Based on previous studies [11], we calculated the sample size using PASS software (PASS 11. NCSS, LLC. Kaysville, Utah, USA) by a statistical calculator based on a 95% confidence interval and 80% power with α

error 5%. Based on this assumption, a minimal sample size of 92 cases was enough to assess the difference. The sample was increased to 100 cases to cover the drop-out.

2.4. Statistical Analysis. Statistical analysis was carried out via SPSS version 23. Shapiro–Wilk test was used for normal distribution variables. Numerical data were presented as median and range or mean and standard deviation. Categorical data were expressed as percentages. The two-tailed Student's *t*-test was used to examine the significance of the difference between the groups, whereas the chi-squared (χ^2) test was used to analyze qualitative variables. The *P*-values ≤ 0.05 were considered statistically significant. Mann–Whitney test was used to compare normally distributed data. Moreover, correlations between ED grade and other studied factors were evaluated using Pearson and Spearman's correlation coefficient.

3. Results

3.1. Prevalence of ED in the Study Population. One hundred male patients with acute coronary syndrome (ACS) who underwent CAG at the CAG unit were included. Patients were divided into two groups according to the presence of ED: 52 cases with ED and 48 cases free from ED.

3.2. Risk Factors of Both Diseases. The present study showed that the mean age was significantly higher in cases with ED (54.52 ± 8.65 years) compared to those who do not suffer from ED (49.83 ± 6.24 years) ($P = 0.002$). However, BMI showed no difference between ED cases and those without ED (24.77 ± 2.6 vs. 24.29 ± 2.39 kg/m^2) ($P = 0.339$).

According to risk factors of ED, the present study showed there was a significant difference between cases with ED and those without ED with regard to hypertension as 22.9% of cases with no ED ($n = 11$) compared to 44.2% ($n = 23$) were hypertensive ($P = 0.025$). However, there was no difference in the percentage of diabetic cases between both studied groups ($P = 0.074$) (Table 1).

In addition, the present study indicated that 17 of the cases without ED (35.4%) and 28 of the cases with ED (53.8%) were smokers. There is no statistically significant difference between the two groups regarding the number of smokers in each group ($P = 0.064$) (Table 1). Moreover, hypercholesterolemia was detected in 37.5% of patients without ED and 46.2% of cases with ED, with no statistically significant difference between cases in both studied groups ($P = 0.264$) (Table 1). The prevalence of patients with a family history of cardiovascular disease in the studied cases was 32%. The percentage of cases with family history of cardiovascular disease among cases without ED is higher than that in cases with ED, but without significant difference (35.4% vs. 28.8%, $P = 0.356$) (Table 1).

Furthermore, the present study showed that there was a significant increase in the number of risk factors (older age, obesity, hypertension, diabetes, hypercholesterolemia, and smoking) in cases with ED compared to those without ED (0.046) (Figure 3).

3.3. Results of CAG. Results of CAG showed that 50% of cases without ED had blockage not necessitating a coronary intervention (no significant disease) compared to 25% of cases with ED. Meanwhile, there were 27 patients who suffered from SVD, 14 (29.2%) among cases who did not suffer from ED, and 13 (25%) among ED cases. However, a higher percentage (30.8%) of cases with DVD were present in cases with ED ($n = 16$) compared to 16.7% in cases without ED ($n = 8$). In addition, there were 10 cases with ED who suffered from TVD (19.2%) compared to two cases in those who did not suffer from ED (4.2%). Comparison between cases with ED and those without ED with regard to CAD showed that a higher percentage of cases with ED have moderate CAD (43.3%) compared to 18.8% in cases without ED (Table 1).

3.4. ED Dysfunction Grade. In the present study, there were 52 patients who suffered from ED. Most of the cases with ED were in mild grade 38.5%. In addition, 23.1% of cases had mild-to-moderate grade ED, and 21.1% of cases had ED in moderate grade. Finally, seven cases have a severe grade (13.5%) (Table 2).

The mean age was significantly different between the patients without ED and different ED severity groups: the mean age of the patients without ED was 49.83 ± 6.24 years, whereas the patients with any different degrees of ED showed increasing age ($P < 0.001$) (Figure 4).

3.5. Penile Artery Doppler Results. All cases without ED (100%) had penile artery Doppler PSV findings ≥ 30 (cm/s) with an average Doppler PSV of 48.02 ± 7.49 cm/s for the right cavernosal arteries and 47.4 ± 7.17 cm/s for the left cavernosal arteries. Yet, 71.2% of cases with ED had penile artery Doppler PSV findings <30 (cm/s) with an average Doppler PSV of 29.1 ± 7.57 cm/s for the right cavernosal arteries and 28.69 ± 7.11 cm/s for the left cavernosal arteries. There was a statistically significant difference between cases with and without ED regarding PSV on Doppler penile artery results (Table 3).

3.6. Erection Hardness Score. All cases without ED (100%) had full hard and rigid erections, while a higher percentage of cases with ED showed tumescence and rigidity not sufficient for sexual intercourse (52.7%), 32.7% had tumescence and rigidity sufficient for sexual intercourse, while 7.7% showed full erection and the least percentage cases (1.9%) sowed tumescence alone (Table 4).

3.7. Correlation between Grade of ED and Other Studied Factors. Correlation between grade of ED and other studied factors showed that there was positive correlation between advanced grade of ED and age ($r = 0.327$, P0.05), hypertension ($r = 0.213$, $P = 0.033$), DM ($r = 0.253$, $P = 0.011$), smoking ($r = 0.202$, $P = 0.044$), and severity of CAD ($r = 0.332$, $P < 0.001$). (Table 5). However, there was no correlation with BMI, duration of DM, Hypercholesterolemia, and family history of CAD ($P > 0.05$) (Table 5).

Also, the present study showed that more coronary vessel affection is linked to severe forms of ED (Table 6).

4. Discussion

Patients were divided into two groups according to the presence of ED: 52 cases with any degree of ED and 48 cases free from ED, so the prevalence of ED among CHD-complaining cases was 52%.

Table 1: Demographic and clinical data of the studied cases.

Variable	Groups			*P*-value
	Total cases ($n = 100$)	Cases without ED group ($n = 48$)	Cases with ED group ($n = 52$)	
Age (years)	52.27 ± 7.9	49.83 ± 6.24	54.52 ± 8.65	0.002*
35–<50	42 (42%)	25 (52.1%)	17 (32.7%)	
50–<60	40 (40%)	19 (39.6%)	21 (40.4%)	
60–<70	16 (16%)	4 (8.3%)	12 (23.1%)	0.049*
70–80	2 (2%)	0 (0%)	2 (3.8%)	
BMI at enrollment	24.53 ± 2.5	24.29 ± 2.39	24.77 ± 2.6	0.339
Normal: 18.5–24.9	78 (78%)	37 (77.1%)	41 (78.8%)	
Overweight: 25.0–29.9	19 (19%)	10 (20.8%)	9 (17.3%)	0.921
Obese: ≥30.0	3 (3%)	1 (2.1%)	2 (3.8%)	
Hypertension				
No	66 (66%)	37 (77.1%)	29 (55.8%)	0.025*
Yes	34 (34%)	11 (22.9%)	23 (44.2%)	
Duration of hypertension (years)	6.68 ± 3.38	6.36 ± 2.6	6.83 ± 3.75	0.715
Diabetes mellitus				
No	64 (64%)	35 (72.9%)	29 (55.8%)	0.074
Yes	36 (36%)	13 (27.1%)	23 (44.2%)	
Duration of diabetes (years)	5.69 ± 2.97	5.23 ± 2.7	5.96 ± 3.2	0.490
HbA1c (per g%)	5.95 ± 1.16	5.68 ± 1.16	6.2 ± 1.12	0.024*
Smoking				
No	55 (55%)	31 (64.6%)	24 (46.2%)	0.064
Yes	45 (45%)	17 (35.4%)	28 (53.8%)	
Hypercholesterolemia				
No	58 (58%)	30 (62.5%)	28 (53.8%)	0.264
Yes	42 (42%)	18 (37.5%)	24 (46.2%)	
Family history of cardiovascular disease				
No	68 (68%)	31 (64.6%)	37 (71.2%)	0.356
Yes	32 (32%)	17 (35.4%)	15 (28.8%)	
Different patterns of coronary vessel involvement				
No	37 (37%)	24 (50%)	13 (25%)	0.011
SVD	27 (27%)	14 (29.2%)	13 (25%)	—
DVD	24 (24%)	8 (16.7%)	16 (30.8%)	—
TVD	12 (12%)	2 (4.2%)	10 (19.2%)	—
Degree of coronary vessel involvement				
No	37 (37%)	24 (50%)	13 (25%)	—
Mild	25 (25%)	13 (27%)	12 (23.1%)	0.046*
Moderate	31 (31%)	9 (18.8%)	22 (43.3%)	—
Severe	7 (7%)	2 (4.2%)	5 (9.6%)	—

Values are expressed as mean ± standard deviation or *n* (%). BMI, body mass index; No, normal or nonsignificant CAD, SVD, single-vessel disease; DVD, double-vessel disease; TVD, triple-vessel disease. *Indicates the *P* value > 0.05.

In prior literature, in the patients with ED, the prevalence of CAD has been reported as 5%–56% in the literature. In contrast, ED prevalence in symptomatic patients with CAD has been reported as 44%–75% [12]. In concordance with our results, Montorsi et al. [13] concluded that the prevalence of ED was 42%–57% in CVD patients. Rinkūnienė et al. [14], in a recent work, found that ED was found among 62% of the cardiovascular patients.

In Egypt, Mittawae et al. [15], in 2006, reported that the ED prevalence was 43.2%, and Atta et al. [16] found that this problem prevailed in 36 cases out of 100 who underwent CAG.

The fact that different research covered different groups, described ED differently and used distinct methodologies to diagnose CVD helps to explain this disparity in prevalence. Additionally, the included patients have a wide range of comorbidities, and several medications are administered, each of which may have varied effects on erectile function [14].

As regards risk factors of both diseases, the current study showed that the mean ± SD of cases age was 52.27 ± 7.9 years. Most patients had age less than 60 years (82%). The mean age was significantly higher in ED cases (54.52 ± 8.65 years) compared to those who are free from ED (49.83 ± 6.24 years) ($P = 0.002$). The mean age was significantly different

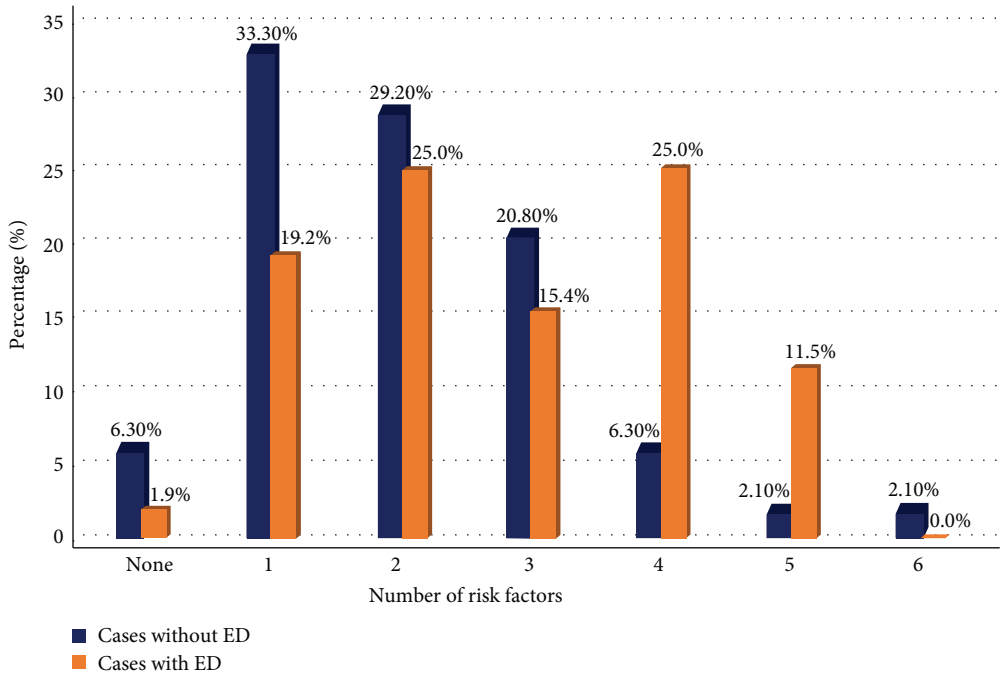

Figure 3: Comparison between cases with and without ED according to number of risk factors.

Table 2: Frequencies and percentages of cases with different grades of ED disease among studied cases.

Grade of ED	Frequency ($n = 52$)	Percentage (%)
Mild	22	42.3
Mild-moderate	12	23.1
Moderate	11	21.1
Severe	7	13.5

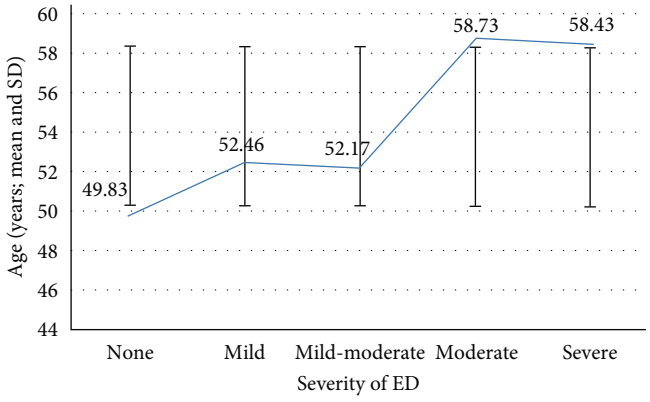

Figure 4: Association between ED severity and patient's age.

between the patients without ED and different ED severity groups ($P < 0.001$). The prevalence of ED increases with age as well as the severity degree of ED.

In line with our results, Rinkūnienė et al. [14] reported that the mean patients' age was 57.6 ± 8.8 years, and most of their included cases aged less than 60 years (60.32%), like our results (82%). The mean age in Tabosa et al. [17] study was 57.0 ± 9.9 years.

Atta et al. [16], also in another Egyptian study, reported that the age of ED patients ranged from 45 to 71 years, with increasing mean age in cases with increasing severity of CAD and subsequent increasing frequency and severity of ED.

Salama et al. [18], in concordance with our findings, reported that the ED prevalence fluctuates according to age, from 1% to 10% in men aged less than 49 years, 20%–40% among men aged between 60 and 69 years, and 50%–100% in men older than 70 years.

There is a consensus that age is a robust determinant of ED happening, with prior studies denoting a strong association between ED and progressing age [19, 20].

Age-related hormonal, inflammatory as well as metabolic, together with the increased prevalence of additional risk factors for ED in those elderly people, may be accountable for this link [21]. Furthermore, aging is related to atherosclerosis risk with the development of arteriopathy in both coronary and penile vasculature. Moreover, aging process also instigates penile tissue degeneration, disturbing the function of the central and peripheral nervous system. Finally, elder males have lesser free testosterone levels in their blood [22].

Regarding other risk factors, most patients (78%) in our study had a BMI ranged between 18.5 and 24.9 kg/m^2 and a 24.53 ± 2.5 kg/m^2 mean. However, we could not find any significant difference between cases with ED and those without ED regarding BMI ($P = 0.339$). Hypertension showed a significant difference between the two groups ($P = 0.025$), but the diabetic percentage had no significant difference between both studied groups ($P = 0.074$). Hypercholesterolemia, family history of CVD, and smoking were more in ED cases but without a statistically significant difference from the non-ED counterparts ($P = 0.264$, $P = 0.356$, and $P = 0.064$, respectively). Additionally, a significant increase in risk factors, such as older

Table 3: Penile artery Doppler PSV.

Penile artery Doppler PSV	Cases without ED ($n=48$)	Cases with ED ($n=52$)	P-value
<30 (cm/s)	0 0%	52 100%	$P<0.001$
≥30 (cm/s)	48 100%	0 0%	
Right CA PSV (cm/s) mean ± SD	48.02 ± 7.49	29.1 ± 7.57	$P<0.001$
Left CA PSV (cm/s) mean ± SD	47.4 ± 7.17	28.69 ± 7.11	$P<0.001$

PSV, peak systolic velocity; CA, cavernosal arteries.

Table 4: Distribution of studied cases according to erection hardness grading score.

Erection hardness grading score	Cases with ED ($n=52$)	Cases without ED ($n=48$)	P-value
Full hard and rigid erection	0 (0%)	100%	
Tumescence and rigidity sufficient for sexual intercourse (sufficiently hard erection for penetration but not fully hard)	17 (32.7%)	0 (0%)	
Tumescence and rigidity are not sufficient for sexual intercourse (slightly hard erection)	30 (57.7%)	0 (0%)	<0.001
Tumescence alone (increase in the penis size but no hardness)	1 (1.9%)	0 (0%)	
Penis does not enlarge	4 (7.7%)	0 (0%)	

Table 5: Correlation between grade of ED and other studied factors.

Parameters	Concentration of grade of ED	
	r	P-value
Age (years)	0.327	<0.001*
BMI (kg/m^2)	−0.014	0.890
Hypertension	0.213	0.033*
DM	0.253	0.011*
Duration of DM	0.076	0.661
Smoking	0.202	0.044*
Hypercholesterolemia	0.110	0.277
Family history of CAD	−0.081	0.425
Penile artery duplex	0.767	0.001*
Severity of CAD	0.332	<0.001*

*Indicates the P value > 0.05.

Table 6: Degree of coronary vessel involvement and different grades of ED.

Total 52 ED cases	No	SVD	DVD	TVD
Mild ED				
N (22)	8	8	6	0
%	36.36%	36.36%	27.27%	0%
Mild-moderate ED				
N (12)	3	3	2	4
%	25%	25%	16.7%	33.3%
Moderate ED				
N (11)	2	1	7	1
%	18.18%	9.09%	63.63%	9.09%
Severe ED				
N (7)	0	1	1	5
%	14.28%	14.28%	14.28%	71.42%

age, obesity, diabetes, hypertension, hypercholesterolemia, and smoking, showed up in cases with ED in comparison to non-ED patients ($P=0.046$).

In agreement with our finding, Rinkūnienė et al. [14], in 2021, reported that hypertension was reported in 38% of their patients. They also reported the presence of hyperglycemia in 32.2%. Moreover, 52.2% of their included cases were smokers, 100% had dyslipidemia, and 32.4% had positive familial history of CVD. They also reported that CVD risk factors (obesity, smoking, dyslipidemia, DM, and positive family history) in their cases appeared more in cases with ED than those without ED; however, statically significance is not available in difference ($P>0.05$). However, hypertension was significantly more common in the ED patients than in their non-ED counterparts ($P=0.02$), like our results.

A prior meta-analysis conducted by Wang et al. [23] proved that hypertensive males experienced severe ED at a higher rate than the general population. In line with all these results, hypertension has been established as a key predictor of ED [24, 25].

Memon et al. [3], in 2022, also reported that 91.7% of their included cases had one or more risk factors for CVD development: 35.97% were diabetic, 55.78% had hypertension, 59.41% were hyperlipidemic, smoking was present in 20.13% of people, and CAD ran in 38.61% of patients' family history. Atta et al. [16] also reported that cases with more severe CVD and more severe ED degrees were more obese; the majority of them were smokers, hypertensive, diabetic, and hyperlipidemic.

Moreover, in agreement with our findings, Seid et al. [26] revealed that 69.9% of diabetic patients in their study group had ED.

Additionally, it is clear from the literature that smoking-related atherosclerosis accumulation in the pudendal artery results in arteriogenic ED. Hallanzy et al. [27] stated that in

their study, smokers had 1.2 times the odds of developing ED as nonsmokers. Moreover, endothelial damage and inflammation driven by hyperlipidemia increase the risk of ED [28]. Nevertheless, literature is restricted concerning the direct association between ED and a family history of CAD. Some research has revealed an indirect relationship between having a positive family history of CAD and developing ED [29].

As regards the presence and severity of ED, the International Index of Erectile Function IIEF-5 questionnaire was used to assess the prevalence of ED, and our results showed that among the 52 cases that suffered from ED. Most cases with ED had a mild grade of 38.5%. In addition, 23.1% of cases had mild-to-moderate grade ED in, and 21.1% of cases had ED in moderate grade. Finally, seven cases have severe grades (13.5%) using the IIEF-5 score.

Nearer to our results, Rinkūnienė et al. [14] reported that mild ED represented the most common grade (37.4%), followed by mild-to-moderate grade (15.2%) of cases and 5.3% of cases had ED in moderate grade, and the remaining 4.1% had severe grade while somewhat more than a third of their cases(38%) did not report ED.

Closer to our results also, Tabosa et al. [17] concluded that 76.3% of cases had ED, with 16.1% having severe ED, 15.2% having moderate ED, and 37.4% having mild grade ED.

Memon et al. [3] reported that ED was present in 74.3% of patients who underwent CAG: 19.5% of patients had mild ED, 38.28% suffered from moderate ED, and 16.5% suffered from severe ED.

Kumar et al. [30] showed that 70% of the male patients who underwent CAG had any degree of ED, but unlike our results, most cases had a severe degree (39.2%), moderate in 23.5%, mild-to-moderate in 22.7%, and mild in 14.6%.

Regarding results of penile artery Doppler findings, all cases without ED (100%) had penile artery Doppler PSV findings ≥ 30 (cm/s) with an average of 48.02 ± 7.49 cm/s in the right cavernosal arteries and 47.4 ± 7.17 cm/s in the left cavernosal arteries. Nonetheless, 71.2% of cases with ED had penile artery Doppler PSV findings <30 (cm/s) with an average Doppler PSV of 29.1 ± 7.57 cm/s for the right cavernosal arteries and 28.69 ± 7.11 cm/s for the left cavernosal arteries. There was a statistically significant difference between cases with and without ED regarding PSV on Doppler penile artery results; all cases without ED (100%) had full hard and rigid erections, while a higher percentage of cases with ED showed rigidity and tumescence insufficient for sexual performance (52.7%), 32.7% had adequate tumescence and rigidity for sexual intercourse while 7.7% showed full erection and the least percentage cases (1.9%) showed tumescence alone.

EDV generally represents the venous function of the corpus cavernosum of the penis, while PSV typically represents the blood supply function of the penile cavernosal artery [31].

There are limited data in the literature regarding the results of penile artery Doppler findings and their relation to coronary vessel involvement.

Even though it is now familiar that ED, and specifically vasculogenic ED, might herald clinical symptoms of CVD, the precise role of penile artery Doppler measures for predicting the risk of CVD in the future is still not well-defined [32].

Atta et al. [16], in line with our results, also reported that on penile artery Doppler cases with no or minor coronary affection had all full and hard erections and cases with more involvement of coronary vessels had worsening of erectile hardness scores.

It is generally accepted that PSV is diagnostic for penile vascular impairment when PSV is less than 30 cm/s and has the ability to identify patients at risk for peripheral vascular damage or CVD [7].

A major strength of the current study is assessing the PSV that is commonly utilized to define ED by reason of vascular impairment. Ma et al. [33] reported that the PSV had a 100% sensitivity and 95% specificity in detecting arterial dysfunction, which was confirmed by pudendal arteriography.

According to Corona et al. [34], It has been shown that the impairment on penile Doppler ultrasound is a separate risk factor for CVD.

Some previous researchers have already described the association between penile Doppler ultrasound examination and cardiovascular disease. For instance, cavernosal artery insufficiency, defined as PSV < 30 cm/s, was a predictor of abnormal stress echocardiography in an earlier prospective research involving 49 men who complained of vascular ED [35]. Another study indicated that 1,687 of the 2,396 patients who were a part of it and had long-term follow-up experienced vascular ED [36].

Furthermore, our results revealed that by CAG, 50% of cases without ED had blockage not necessitating a coronary intervention (no significant disease) compared to 25% of cases with ED. Meanwhile, there were 27 patients who suffered from SVD, 14 (29.2%) among cases who did not suffer from ED, and 13 (25%) among ED cases. However, a higher percentage (30.8%) of cases with DVD were present in cases with ED ($n = 16$) compared to 16.7% in cases without ED ($n = 8$). In addition, there were ten cases with ED who suffered from TVD (19.2%) compared to two cases in those who did not suffer from ED (4.2%). The present study showed that more coronary vessel affection is linked to a severe form of ED.

Memon et al. [3] reported that 8 (13.8%) of the subjects who had CAG had normal coronary angiograms, 15 (24.9%) had single-vessel disease (SVD), 17 (29.5%) had DVD, and 18 (31%), were labeled as s TVD and like our results the more coronary involvement, the more cases had ED with increasing grade.

In concordance with our findings, in the Al-Daydamony et al. [37] study, moderate or severe ED patients displayed increasing angiographic severity of CAD with three vessels involvement than patients with mild or no ED.

Shanker et al. [38] also found that severe ED had repeated in multivessel-affected patients when paralleled to solitary-vessel disease. They recommended that the presence of severe ED with a well-controlled CAD may raise the suspicion about the involvement of multiple coronary vessels.

In agreement with this also, Kumar et al. [30] stated that ED patients had elevated incidence of multiple and diffuse coronary vessels involved contrasted to those without ED. Moreover, severe ED patients in their study had a higher number of mean coronary vasculature affected vs. ED patients with milder grades.

Moreover, the current study demonstrates a positive correlation between advanced grade of ED and age ($r = 0.327$, $P < 0.001$), hypertension ($r = 0.213$, $P = 0.033$), DM ($r = 0.253$, $P = 0.011$), smoking ($r = 0.202$, $P = 0.044$), and severity of CAD ($r = 0.332$, $P < 0.001$). However, no correlation was found between BMI, duration of DM, Hypercholesterolemia, and family history of CAD ($P > 0.05$).

Tabosa et al. [17], in line with our findings, reported that there was a correlation between IIEF-5 score values and the more advanced age ($r = -0.224$). The correlation between total ED scores and patients' age ($r = -0.308$ and $P < 0.001$) was also described in Rinkūnienė et al. [14] study.

From all, we can conclude that ED could be deliberated as a part of systemic atherosclerosis indicators. Both CAD and ED share mutual risk factors and pathology described as endothelial dysfunction.

5. Conclusion

More than half of cases with CAD frequently have ED with varying grades. Both the severity and incidence of ED are significantly more common in cases with multivessel CAD. The prevalence of ED among CAD patients shows that erectile function may have already been compromised before heart vessel damage reveals symptoms. Physicians should put into consideration the strong association between the two conditions, as ED may become apparent before angina symptoms. Subsequently, patients with ED can have optimum risk stratification for concurrent systemic atherosclerosis and CAD. Also, pathological penile vessels PSV (<25 or <30 cm/s) via Doppler ultrasound is mostly valuable as a noninvasive tool to define ED attributable to vascular impairment that may be antecedent to atherosclerosis in the coronary vessels.

Data Availability

The data are available from the corresponding author upon reasonable request.

Ethical Approval

The Quality Assurance Unit at the Faculty of Medicine, Al-Azhar University, Egypt, approved the study.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Acknowledgments

The research was funded by the authors.

References

[1] D. A. Abuhay, Y. Y. Gela, and A. A. Getu, "Prevalence of erectile dysfunction and associated factors among hypertensive patients attending governmental health institutions in Gondar City, Northwest Ethiopia: a cross-sectional study," *International Journal of Hypertension*, vol. 2021, Article ID 1482500, 10 pages, 2021.

[2] A. Nehra, "Erectile dysfunction and cardiovascular disease: efficacy and safety of phosphodiesterase type 5 inhibitors in men with both conditions," *Mayo Clinic Proceedings*, vol. 84, no. 2, pp. 139–148, 2009.

[3] S. A. Memon, M. Adil, F. R. Khan, S. Ullah, S. Rehmat, and N. Z. Gul, "Association between erectile dysfunction, cardiovascular risk factors, and coronary artery disease: role of exercise stress testing and international index of erectile function (IIEF-5) questionnaire," *IJC Heart & Vasculature*, vol. 40, Article ID 101033, 2022.

[4] O. A. Raheem, J. J. Su, J. R. Wilson, and T. C. Hsieh, "The association of erectile dysfunction and cardiovascular disease: a systematic critical review," *American Journal of Men's Health*, vol. 11, no. 3, pp. 552–563, 2017.

[5] S. R. Chowdhury, M. Karim, S. M. A. Ullah, and M. A. Bakar, "Association between erectile dysfunction and cardiovascular disease: a systematic review," *Chattagram Maa-O-Shishu Hospital Medical College Journal*, vol. 66, no. 2, pp. 59–66, 2020.

[6] M. Desai, G. Naik, U. S. Kamat, and J. A. Cacodcar, "Concurrence of erectile dysfunction and coronary artery disease among patients undergoing coronary angiography at a tertiary medical college hospital in Goa," *Indian Heart Journal*, vol. 72, no. 2, pp. 123–125, 2020.

[7] M. D. R. Ponce, M. Vecchiato, D. Neunhaeuserer et al., "Association between penile color Doppler ultrasonography and cardiorespiratory fitness in patients with vascular erectile dysfunction," *Sexual Medicine*, vol. 9, no. 3, Article ID 100347, 2021.

[8] R. C. Rosen, J. C. Cappelleri, M. D. Smith, J. Lipsky, and B. M. Peña, "Development and evaluation of an abridged, 5-item version of the International Index of Erectile Function (IIEF-5) as a diagnostic tool for erectile dysfunction," *International Journal of Impotence Research*, vol. 11, no. 6, pp. 319–326, 1999.

[9] F. Carneiro, O. C. Saito, and E. P. Miranda, "Standardization of penile hemodynamic evaluation through color duplex-doppler ultrasound," *Revista da Associação Médica Brasileira*, vol. 66, no. 9, pp. 1180–1186, 2020.

[10] U. Khanzada, S. A. Khan, M. Hussain et al., "Evaluation of the causes of erectile dysfunction in patients undergoing penile doppler ultrasonography in Pakistan," *The World Journal of Men's Health*, vol. 35, no. 1, pp. 22–27, 2017.

[11] L. Canat, G. Cicek, G. Atis, C. Gurbuz, and T. Caskurlu, "Is there a relationship between severity of coronary artery disease and severity of erectile dysfunction?" *International Brazilian Journal of Urology*, vol. 39, no. 4, pp. 465–473, 2013.

[12] O. Doluoglu, C. Tatar, G. Kutucularoglu et al., "Erectile dysfunction as a risk factor predicting coronary artery disease," *Nephro-Urology Monthly*, vol. 2, no. 3, pp. 462–468, 2010.

[13] P. Montorsi, P. M. Ravagnani, S. Galli et al., "Common grounds for erectile dysfunction and coronary artery disease," *Current Opinion in Urology*, vol. 14, pp. 361–365, 2004.

[14] E. Rinkūnienė, S. Gimžauskaitė, J. Badarienė, V. Dženkevičiūtė, M. Kovaitė, and A. Čypienė, "The prevalence of erectile dysfunction and its association with cardiovascular risk factors in patients after myocardial infarction," *Medicina*, vol. 57, no. 10, pp. 1103–1114, 2021.

[15] B. Mittawae, A. R. El-Nashaar, A. Fouda, M. Magdy, and R. Shamloul, "Incidence of erectile dysfunction in 800 hypertensive patients: a multicenter Egyptian national study," *Urology*, vol. 67, no. 3, pp. 575–578, 2006.

[16] A. Atta, M. Salama, M. Sallam, and A. El-Shahed, "Association between erectile dysfunction and severity of coronary artery

disease," *Al-Azhar Medical Journal*, vol. 49, no. 3, pp. 871–880, 2020.

[17] A. Tabosa, D. C. D. Oliveira, V. H. Stangler et al., "Association between erectile dysfunction and quality of life in patients with coronary artery disease," *International Journal of Cardiovascular Sciences*, vol. 30, no. 3, pp. 219–226, 2017.

[18] M. N. Salama, A. A. Eid, A. Hatem, and A. K. Swidan, "Prevalence of erectile dysfunction in Egyptian males with metabolic syndrome," *The Aging Male*, vol. 23, no. 4, pp. 257–263, 2020.

[19] J. S. Bu, "Vascular endothelial dysfunction and pharmacological treatment," *World Journal of Cardiology*, vol. 7, no. 11, pp. 719–741, 2015.

[20] S. Çayan, M. Kendirci, Ö. Yaman et al., "Prevalence of erectile dysfunction in men over 40 years of age in Turkey: results from the Turkish Society of Andrology Male Sexual Health Study Group," *Turkish Journal of Urology*, vol. 43, pp. 122–129, 2017.

[21] R. Kapoor and A. Kapoor, "Erectile dysfunction: a present day coronary disease risk equivalent," *Indian Journal of Medical Research*, vol. 144, no. 3, pp. 307–310, 2016.

[22] T. Head, S. Daunert, and P. J. Goldschmidt-Clermont, "The aging risk and atherosclerosis: a fresh look atarterial homeostasis," *Frontiers in Genetic*, vol. 8, pp. 216–226, 2017.

[23] X. Y. Wang, W. Huang, and Y. Zhang, "Relation between hypertension and erectile dysfunction: a meta-analysis of cross-section studies," *International Journal of Impotence Research*, vol. 30, no. 3, pp. 141–146, 2018.

[24] A. V. Sarma, J. M. Hotaling, I. H. de Boer et al., "Blood pressure, antihypertensive medication use, and risk of erectile dysfunction in men with type I diabetes," *Journal of Hypertension*, vol. 37, no. 5, pp. 1070–1076, 2019.

[25] A. Adeba Garcia, R. Alvarez Velasco, M. Vigil-Escalera Diaz et al., "Predictive factors of erectile dysfunction in a cardiac rehabilitation programme. What can we improve?" *European Journal of Preventive Cardiology*, vol. 28, no. Supplement_1, Article ID zwab061.315, 2021.

[26] A. Seid, H. Gerensea, S. Tarko, Y. Zenebe, and R. Mezemir, "Prevalence and determinants of erectile dysfunction among diabetic patients attending in hospitals of central and northwestern zone of Tigray, northern Ethiopia: a cross-sectional study," *BMC Endocrine Disorders*, vol. 17, no. 1, pp. 1–7, 2017.

[27] J. Hallanzy, M. Kron, V. E. Goethe et al., "Erectile dysfunction in 45-year-old heterosexual German men and associated lifestyle risk factors and comorbidities: results from the German male sex study," *Sexual Medicine*, vol. 7, no. 1, pp. 26–34, 2019.

[28] J. B. Kostis and J. M. Dobrzynski, "Statins and erectile dysfunction," *The World Journal of Men's Health*, vol. 37, no. 1, pp. 1–3, 2019.

[29] G. Sangiorgi, A. Cereda, D. Benedetto et al., "Anatomy, pathophysiology, molecular mechanisms, and clinical management of erectile dysfunction in patients affected by coronary artery disease: a review," *Biomedicines*, vol. 9, no. 4, pp. 432–451, 2021.

[30] J. Kumar, T. Bhatia, A. Kapoor et al., "Association between erectile dysfunction and severity of coronary artery disease: observations from a coronary angiographic study in Asian Indians," *Heart*, vol. 98, no. Suppl 2, pp. E317-E318, 2012.

[31] X. J. Xuan, G. Bai, C. X. Zhang et al., "The application of color doppler flow imaging in the diagnosis and therapeutic effect evaluation of erectile dysfunction," *Asian Journal of Andrology*, vol. 18, no. 1, pp. 118–122, 2016.

[32] N. Caretta, M. De Rocco Ponce, N. Minicuci et al., "Penile Doppler ultrasound predicts cardiovascular events in men with erectile dysfunction," *Andrology*, vol. 7, no. 1, pp. 82–87, 2019.

[33] M. Ma, B. Yu, F. Qin, and J. Yuan, "Current approaches to the diagnosis of vascular erectile dysfunction," *Translational Andrology and Urology*, vol. 9, no. 2, , pp. 709–721, 2020.

[34] G. Corona, G. Fagioli, E. Mannucci et al., "Original research—erectile dysfunction: penile doppler ultrasound in patients with erectile dysfunction (ED): role of peak systolic velocity measured in the flaccid state in predicting arteriogenic ED and silent coronary artery disease," *The Journal of Sexual Medicine*, vol. 5, no. 11, pp. 2623–2634, 2008.

[35] J. Mulhall, P. Teloken, and J. Barnas, "Vasculogenic erectile dysfunction is a predictor of abnormal stress echocardiography," *The Journal of Sexual Medicine*, vol. 6, no. 3, pp. 820–825, 2009.

[36] G. Rastrelli, G. Corona, M. Monami et al., "Poor response to alprostadil ICI test is associated with arteriogenic erectile dysfunction and higher risk of major adverse cardiovascular events," *The Journal of Sexual Medicine*, vol. 8, no. 12, pp. 3433–3445, 2011.

[37] M. M. Al-Daydamony, A. Shawky, and A. Tharwat, "Erectile dysfunction severity as a predictor of left main and/or three-vessel disease in acute coronary syndrome patients," *Indian Heart Journal*, vol. 70, pp. S56–S59, 2018.

[38] A. S. R. Shanker, B. Phanikrishna, and C. B. V. Reddy, "Association between erectile dysfunction and coronary artery disease and it's severity," *Indian Heart Journal*, vol. 65, no. 2, pp. 180–186, 2013.