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ORIGINAL RESEARCH

Erectile dysfunction and angiographic correlation between coronary artery stenosis and internal iliac-internal pudendal artery stenosis in patients with suspected coronary artery disease: a retrospective study

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Abstract

This study aimed to assess the angiographic correlation between coronary artery stenosis and internal iliac-internal pudendal artery (II-IPA) stenosis and evaluate its association with erectile dysfunction (ED). We reviewed the data of 91 patients who had undergone pelvic angiography (PA) to evaluate II-IPA stenosis and coronary angiography (CAG) due to suspected coronary artery disease. Erectile function (EF) was evaluated using the International Index of Erectile Function before CAG and PA. CAG was performed first, followed by PA of the bilateral II-IPA. Regardless of the location and number of stenosis sites, based on CAG, we categorized the patients into two groups. Patients with a maximum stenosis <50% and ≥50% on CAG were assigned to Group I and Group II, respectively. Then, the EF domain score and the diameter stenosis (DS) of II-IPA were evaluated and compared. Overall, 55 patients comprised Group I, while 36 patients comprised Group II. ED was present in 96.3% and 97.2% of the patients in Group I and II, respectively. There was no statistical difference between the groups in the severity of ED (p = 0.457). PA revealed that 14.5% and 36.1% of patients in Groups I and II had ≥50% stenosis of the II-IPA. The mean DS of II-IPA in Group II was greater than that in Group I (p = 0.017). There was a statistically significant correlation between the degree of coronary artery stenosis and the degree of II-IPA stenosis (r = 0.295, p = 0.005). This study revealed that coronary artery stenosis correlates with II-IPA stenosis. The presence and degree of coronary artery stenosis or II-IPA stenosis indicate the necessity for more active treatment in patients with ED.

Keywords

Angiography; Coronary stenosis; Coronary artery disease; Erectile dysfunction

1. Introduction

Erectile dysfunction (ED) and coronary artery disease (CAD) share several risk factors such as alcohol abuse, diabetes, hypertension, hypercholesterolemia, obesity, tobacco smoking, and low testosterone; additionally, vascular endothelial dysfunction underlies both these conditions [1–3]. Several systemic meta-analyses showed that ED is considered a sign of a clinical spectrum of illness, which may progress to CAD in 5 years [4–6]. Therefore, some researchers advocate the need to evaluate the risk of CAD through ED screening [7–9].

In the pathophysiology of CAD, prolonged exposure to several risk factors damages endothelial cells and induces vascular endothelial dysfunction. This leads reduced vasodilation and a proinflammatory and prothrombic state due to impairment of nitric oxide (NO) production or inactivation of NO by reactive oxygen species, eventually leading to the development of atherosclerotic lesions [10–12]. Persistence of dysfunction reduces the coronary artery's inner diameter, decrease blood flow, resulting in ischemic damage to the cardiac muscle [11–13]. Although vascular endothelial dysfunction is a major contributor to ED, decreased blood flow to the penis cannot be considered as the cause of ED because erectile function (EF) is affected by endothelial dysfunction prior to atherosclerotic plaque formation. Nevertheless, as vascular endothelial dysfunction results in blood vessels stenosis, patients with ED may show significant internal iliac-internal pudendal arteries (II-IPA) stenosis, which are the primary sources of blood to the penis [14–16]. Additionally, it was estimated that coronary artery stenosis correlates with II-IPA stenosis.

Few studies have revealed an angiographic correlation between coronary artery stenosis and II-IPA stenosis. Performing

coronary angiography (CAG) and pelvic angiography (PA) would be essential to reveal the correlation between coronary artery stenosis and II-IPA stenosis; however, angiography, including CAG and PA, is an invasive procedure. PA performed alone or concurrently with CAG for research or diagnostic purposes without a specific therapeutic approach can be harmful to patients and be subject to moral condemnation. Therefore, there is currently no evidence of a correlation between coronary artery stenosis and II-IPA stenosis.

In this study, we evaluated the correlation between coronary artery stenosis and II-IPA stenosis by comparing and analyzing the angiography results of patients who had undergone CAG and PA. Furthermore, we assessed the association of coronary artery stenosis and II-IPA stenosis with ED.

2. Materials and methods

2.1 Study population

This retrospective study analyzed the data of patients who had undergone CAG due to exercise-induced ischemia during treadmill/cardiac perfusion testing or symptoms of angina consistent with coronary computed tomography findings as well as PA to evaluate II-IPA stenosis between January 2014 and December 2015. This study was conducted at Daejoen St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Republic of Korea. Overall, 91 patients were enrolled. The exclusion criteria were as follows: acute myocardial infarction within 1 month; autoimmune disease; alcohol or substance abuse; cardiogenic shock; generalized anxiety disorder; history of pelvic radiation therapy; high prostate specific antigen (PSA) levels; low serum testosterone level; malignancy; mood or panic disorders; prior penile trauma; prior radical prostatectomy; recent infectious disease; serum creatinine levels >2.5 mg/dL; social phobia; medication for anxiety or depression. A baseline assessment included blood tests, comprehensive health interview, echocardiography, and questionnaires of medical history and social psychology. Blood tests included complete blood count, hemoglobin A1c levels, routine blood chemistry profiles, testosterone levels, PSA levels.

2.2 Assessment of EF

Prior to CAG and PA, sexual function was evaluated using the International Index of EF-15 (IIEF-15), which includes EF, intercourse satisfaction, sexual desire, overall satisfaction, and orgasmic function. The diagnosis and severity of ED were based on EF score as follows: 1–10, severe ED; 11–16, moderate ED; 17–21, mild-moderate ED; 22–25, mild ED; and 26–30, no ED.

2.3 Assessment of coronary artery stenosis

According to the current standard guideline, CAG was done through the radial artery approach. Patients with a maximum stenosis of <50% and ≥50% were allocated to Group I and Group II, respectively, regardless of the location and number of stenotic sites. When coronary artery stenosis was observed, percutaneous coronary intervention (PCI), selection of PCI

strategy, adjunctive antithrombotic therapy, stents (type, diameter, and length), and adjunctive devices were left to the surgeon's discretion.

2.4 Assessment of II-IPA stenosis

II-IPA stenosis assessment was done as described in our previous study [17]. Using quantitative CAG, the vessel sizes of both II arteries and the IPA were assessed. The IPA was assessed in three segments (proximal, middle, and distal). The following parameters were assessed: calculated interpolated normal diameter; lesion length; minimal luminal diameter; proximal and distal reference diameters, and percentage of diameter stenosis (DS).

2.5 Statistical analysis

Student's t-test or Mann-Whitney U test was used to compare continuous variables, expressed as mean $\pm$ standard deviation. Chi-square and Fisher's exact tests were used to compare categorical variables, expressed as count (percentage). Correlation coefficients were determined using Pearson's method. Statistical analyses were done using Statistical Analysis System (SAS) 9.4 (SAS Institute, Cary, NC, USA), and $p < 0.05$ was considered statistically significant.

3. Results

3.1 Baseline clinical characteristics

Patients' baseline clinical characteristics are presented in Table 1. Except for statin use, there was no statistical difference in the clinical characteristics between the two groups. Stain use was significantly higher in Group II compared to Group I. ($p = 0.022$).

3.2 Angiographic assessment of coronary artery stenosis

Coronary artery stenosis <50% and ≥50% in at least one or more areas on CAG was observed in 55 (Group I) (Fig. 1A–B) and 36 (Group II) patients (Fig. 1E–F), respectively. Of the 36 patients in Group II, one-vessel, two-vessel, and three-vessel diseases were observed in 31, 3, and 2 patients, respectively (Table 2). PCI was performed at 11 sites in 10 patients in Group II.

3.3 EF Assessment

Excluding 3 patients (Group I = 2 and Group II = 1), 88 patients were diagnosed with ED. The EF domain score ($p = 0.901$) and the distribution of ED severity ($p = 0.457$) were not statistically different between the two groups. Additionally, there were no statistical differences in orgasmic function ($p = 0.648$), sexual desire ($p = 0.532$), intercourse satisfaction ($p = 0.911$), or overall satisfaction ($p = 0.957$) in the IIEF-15 scores between the two groups (Table 1).

T A B L E 1. Baseline clinical characteristics.

	Group I (DS <50% on CAG)	Group II (DS ≥50% on CAG)	p-value
Number of patients	55	36	
Age (years)	58.44 ± 7.84	60.39 ± 8.48	0.264
BMI (kg/m^2)	24.67 ± 2.71	25.21 ± 3.12	0.388
DM (%)	8 (14.6)	10 (27.8)	0.121
HBP (%)	28 (50.9)	18 (50.0)	0.932
Hyperlipidemia (%)	7 (12.7)	2 (5.6)	0.310
Smoking (%)	23 (41.8)	14 (38.9)	0.781
Previous CVA (%)	1 (1.8)	2 (5.6)	0.560
Previous MI (%)	13 (23.6)	12 (33.3)	0.311
Previous PCI (%)	24 (43.6)	20 (55.6)	0.266
Medication History			
Aspirin (%)	31 (56.4)	26 (72.2)	0.126
Statin (%)	28 (50.9)	27 (75.0)	0.022*
Hormonal status			
PSA (ng/mL)	0.76 ± 0.65	0.93 ± 0.71	0.246
Testosterone (ng/mL)	4.13 ± 1.31	4.32 ± 1.77	0.574
Prolactin (ng/mL)	10.14 ± 7.59	7.88 ± 4.01	0.104
IIEF questionnaire			
Erectile function	14.29 ± 6.73	14.47 ± 6.88	0.901
Orgasmic function	2.93 ± 2.57	3.19 ± 2.94	0.648
Sexual desire	5.84 ± 1.97	6.11 ± 2.15	0.532
Intercourse satisfaction	4.47 ± 3.50	4.39 ± 3.51	0.911
Overall satisfaction	6.58 ± 2.34	6.61 ± 2.78	0.957
Severity of ED			
Normal (26–30)	2 (3.6%)	1 (2.8%)	
Mild ED (22–25)	8 (14.6%)	7 (19.4 %)	
Mild to Moderate ED (17–21)	6 (10.9%)	8 (22.2%)	0.457
Moderate ED (11–16)	24 (43.6%)	10 (27.8%)	
Severe ED (1–10)	15 (27.3%)	10 (27.8%)	

BMI: body mass index; CAG: coronary angiography; CVA: cerebrovascular accident; DM: diabetes mellitus; DS: diameter stenosis; ED: erectile dysfunction; HBP: high blood pressure; IIEF: International Index of erectile function; MI: myocardial infarction; PCI: percutaneous coronary intervention; PSA: prostate specific antigen.
* p < 0.05 (statistically significant difference).

TABLE 2. Angiography results of the coronary arteries and II-IPA.

Coronary angiography						*p* value
	Group I			Group II		
No. of patients						
		DS = 0	18		One-vessel disease	31
	55			36	Two-vessel disease	3
		0< DS <50	37		Three-vessel disease	2
Pelvic angiography						
	Rt. II-IPA			Lt. II-IPA		0.425
No. of patients						
	DS = 0	75 (82.4%)		DS = 0	71 (78.0%)	
	1≤ DS <50	3 (3.3%)		1≤ DS <50	7 (7.7%)	
	DS ≥50	13 (14.3%)		DS ≥50	13 (14.3%)	
	Group I (n = 55)			Group II (n = 36)		
No. of patients						
	DS <50	47 (85.5%)		DS <50	23 (63.9%)	0.017*
	DS ≥50	8 (14.6%)		DS ≥50	13 (36.1%)	
DS (%)	12.93 ± 23.85			29.98 ± 36.57		0.017*
Rt, II	6.45 ± 18.24			19.30 ± 38.45		
Rt. IPA proximal	0.00 ± 0.00			26.41 ± 39.01		
Rt. IPA middle	16.32 ± 30.34			37.05 ± 41.17		
Rt. IPA distal	15.32 ± 28.44			32.10 ± 43.71		
Lt. II	12.32 ± 22.99			17.58 ± 27.64		
Lt. IPA proximal	0.00 ± 0.00			29.41 ± 34.08		
Lt. IPA middle	28.75 ± 30.85			17.33 ± 28.75		
Lt. IPA distal	7.83 ± 22.16			12.91 ± 24.85		

Data are expressed as mean ± standard deviation.
DS: diameter stenosis; II: internal iliac artery; IPA: internal pudendal artery, Lt.: left; Rt.: right.
* p < 0.05 (statistically significant difference).

3.4 Correlation between the severity of ED and the degree of DS in coronary arteries

Fig. 2 shows the distribution of EF domain score and the degree of DS in the coronary arteries. NO statistical correlation was observed between the severity of ED (EF domain score) and the extent of the coronary artery stenosis (r = −0.014, *p* = 0.899).

3.5 Angiographical evaluation of stenosis in bilateral II-IPA

The degree of DS in the II-IPA was similar in the right and left sides, with no statistical difference between the two sides (*p* = 0.425) (Table 2). Among the 91 patients, 70 and 21 patients had a stenosis of <50% and ≥50%, respectively, in at least one or more areas in the bilateral II-IPA. In Groups I and II, 47 (85.5%) (Fig. 1C–D) and 8 (14.5%) patients, and 23 (63.9%) and 13 (36.1%) patients (Fig. 1G–H) had a stenosis of <50%

and ≥50%, respectively. The incidence of stenosis ≥50% in the bilateral II-IPA was significantly higher in Group II than in Group I (*p* = 0.017). Additionally, the total mean DS and mean DS at each measurement site were greater in Group II compared to Group I, except for the Lt. middle IPA (Table 2).

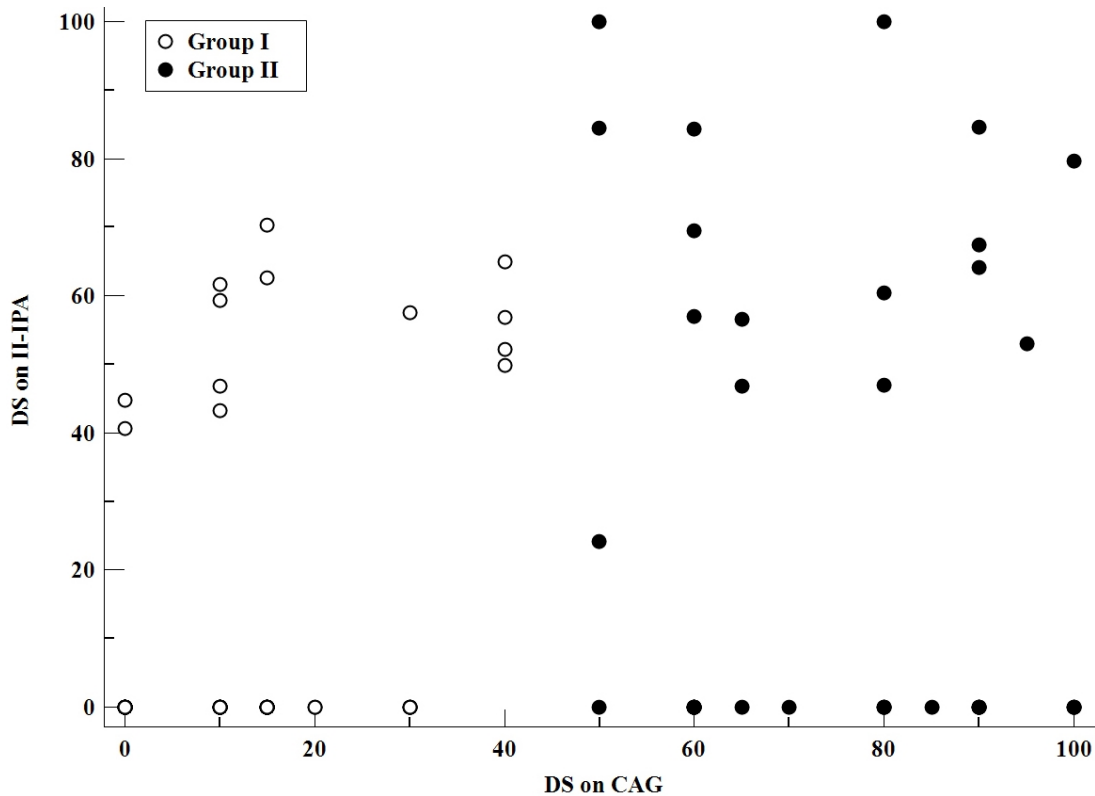### 3.6 Correlation between the DS of coronary artery and the DS of II-IPA

The distribution according to the degree of coronary artery stenosis and II-IPA stenosis in both groups is shown in Fig. 3. A significant correlation was observed between the degree of coronary artery stenosis and the degree of II-IPA stenosis (r = 0.295, *p* = 0.005).
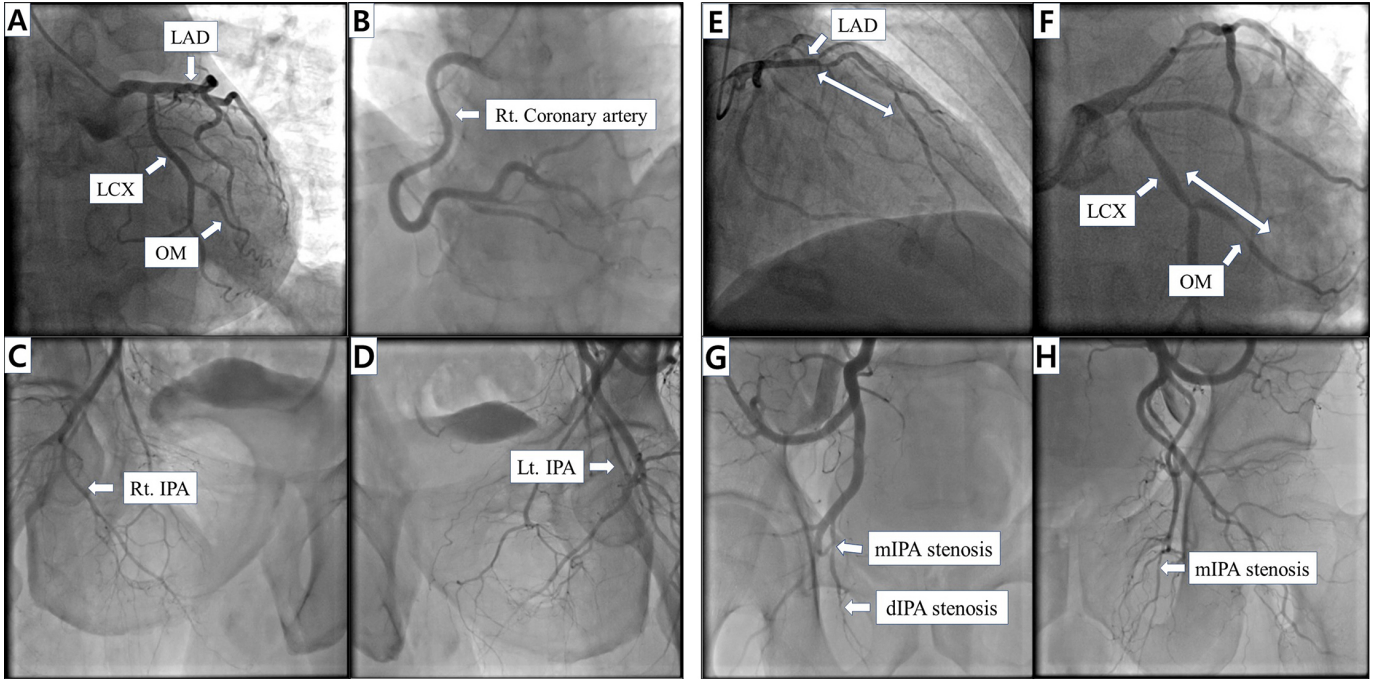
F I G U R E 1. Angiographic findings in coronary arteries and II-IPA. A–D is from one patient in Group I, and E–H is from one patient in Group II. The two-way arrow indicates the location and length of the stenosis. A: normal CAG showing Lt. coronary artery, B: normal CAG showing Rt. coronary artery, C: normal PA showing Rt. II-IPA, D: normal PA showing Lt. II-IPA: E, stenosis of LAD (DS = 85) in CAG: F, stenosis of LCX OM branch (DS = 90) in CAG: G, stenosis of mIPA (DS = 54) and dIPA (DS = 85) in Rt. IPA: H, stenosis of mIPA (DS = 19) in Lt. IPA. The DS was measured by the QCA method. CAG: coronary angiography; dIPA: distal internal pudendal artery; DS: diameter stenosis; II-IPA: internal iliac-internal pudendal artery; LAD: left anterior descending artery; Lt.: left; LCX: left circumflex artery; mIPA: middle internal pudendal artery; OM: obtuse marginal artery; QCA method: quantitative coronary angiographic method; Rt.: right.
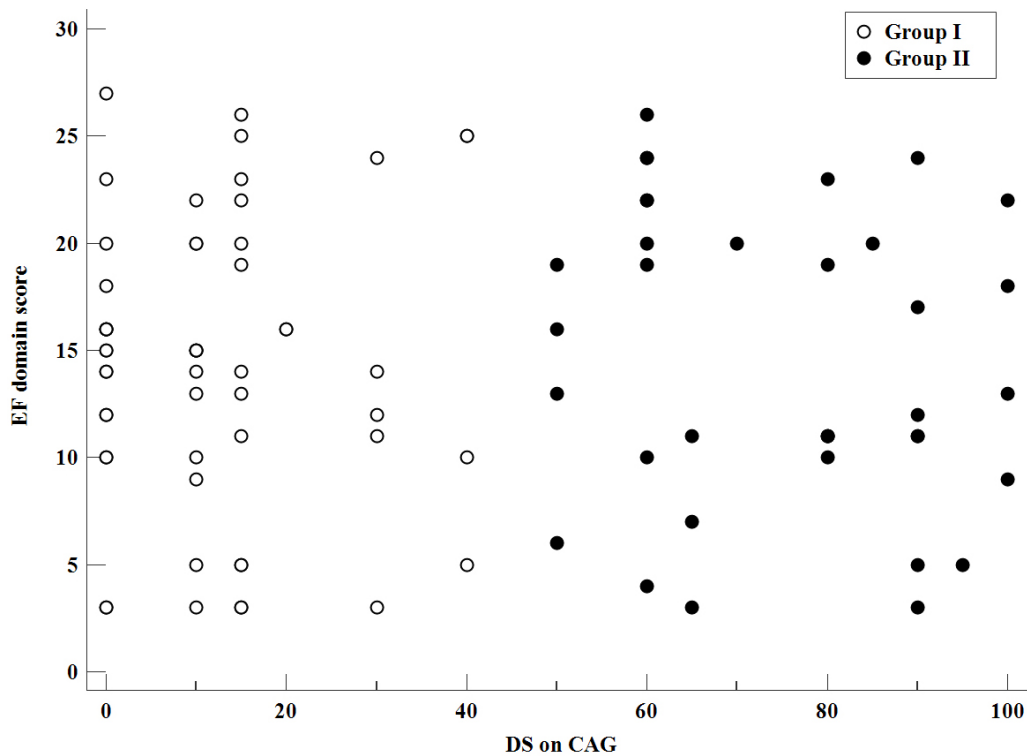
F I G U R E 2. Patient distribution based on the DS on CAG and EF domain score. CAG: coronary angiography; DS: diameter stenosis; EF: erectile function; p: probability value; r: correlation coefficient (r = −0.014, p = 0.899).

F I G U R E 3. Patients' categorization according to the degree of coronary artery stenosis and II-IPA stenosis. CAG: coronary angiography; DS: diameter stenosis; II-IPA: internal iliac-internal pudendal artery; *p*: probability value; r: correlation coefficient (r = 0.295, *p* = 0.005).

4. Discussion

Few studies have revealed an angiographic correlation between coronary artery stenosis and II-IPA stenosis. Rogers *et al*. [18] performed CAG and PA in 10 patients with suspected CAD and ED who were nonresponsive to phosphodiesterase-5 inhibitor (PDE5i) and reported a correlation between CAD and IPA disease in patients with ED. Sanad *et al*. [19] performed CAG and PA to examine the relationship between angiographically verified CAD and aorto-ilio-pudendal (A-I-P) artery disease in patients with ischemic heart disease-associated vascular ED (n = 60). They reported that ED correlates with A-I-P vascular lesions than with CAD alone, and that there was a significant association between the severity of CAD and A-I-P artery disease in patients with CAD associated with ED. While these studies suggested that coronary artery stenosis and IPA stenosis may be correlated, they were limited by their small study populations.

Our study showed that most patients with clinically suspected CAD exhibited ED. Furthermore, the study results revealed no correlation between the degree of coronary artery stenosis and ED severity. However, a significant correlation was observed between the degree of coronary artery stenosis and degree of II-IPA stenosis.

The Massachusetts Male Aging Study and subsequent studies reported that the ED rates in patients with CAD are as high as 75% [5, 20, 21]. In our study, 97% of the patients diagnosed angiographically with CAD were confirmed to have ED, similar to the previously reported ED prevalence rate.

Additionally, 96% of patients suspected of having CAD but were angiographically confirmed to have no CAD had ED. ED can manifest even in the early stages of endothelial dysfunction prior to plaque formation. Sanad *et al*. [19] reported that angiographically confirmed CAD was present in 10% of patients complaining of ED but without clinical symptoms of CAD. Our study results support the opinion that ED precedes CAD by 5 years.

Greenstein *et al*. [22] evaluated IIEF scores in 40 patients scheduled for CAG for ischemic heart disease and reported that the ED worsened with increased number of stenotic coronary arteries. In our study, only 5 of the 36 patients in Group II had two or three-vessel disease on CAG. Therefore, ED severity cannot be compared in relation to the number of afflicted coronary arteries. Instead, we evaluated whether the degree of coronary artery stenosis correlates to the severity of ED and found that there was no correlation between the two; This agrees with our previous study results, where no correlation was observed between the EF domain score and the DS of II-IPA [17]. However, a significant number of patients confirmed to have no CAD by CAG presented with symptoms of ED; therefore, we believe that the degree and presence of atherosclerotic plaque formation in the coronary arteries and II-IPA indicate that the anatomical structure of the arteries has been altered due to endothelial dysfunction, suggesting the need for more active treatment of ED rather than being an indicator of ED severity.

Our results showed that the degree of coronary artery stenosis correlates with the degree of II-IPA stenosis, which agrees

with the findings of Rogers *et al.* [18] and Sanad *et al.* [19]. We believe that these findings confirm the reasonable assumption that there is a correlation between coronary artery stenosis and II-IPA stenosis because CAD and ED are linked to the common pathophysiology of vascular endothelial dysfunction.

The above correlation has several clinical implications. First, as PCI is performed in patients with CAD, it is pertinent to consider whether percutaneous intervention is required in patients with II-IPA stenosis to improve ED. Several researchers have attempted percutaneous intervention, including percutaneous transluminal angioplasty (PTA) or stent placement, in patients with II-IPA stenosis during PA. Valji and Bookstein performed PTA in arteriogenic patients with ED and IPA stenosis, and Roger *et al.* [18] performed percutaneous stent revascularization for the first time in 30 patients with ED who had suboptimal responses to PDE5i. Additionally, Diehm *et al.* [25] performed endovascular therapy in 50 patients with atherosclerotic ED who had a poor response or contraindication to PDE5i [23–25]. These studies reported that the expansion of stenosis and improvement of blood flow were achieved in most patients without adverse effects during the procedure, with the obtained favorable treatment results as follows: the IIEF score improved by ≥ 4 points in comparison to the pre-procedure score in 60–70% of the patients; furthermore, the improved score was maintained over 3 weeks to 12 months follow-up period. However, Von Allmen *et al.* [26] study showed modest results upon endovascular revascularization and no significant improvement in the IIEF score.

Although most previous studies have shown favorable results, the percutaneous intervention has not been adopted as the standard treatment in patients with ED and II-IPA stenosis because these studies have been limited by their small sample sizes, and the safety and efficacy of this intervention have not been established. However, the percutaneous intervention has shown sufficient therapeutic potential in this patient population, and we believe that a long-term, multi-center, well-designed prospective study on this topic is necessary. It will be important to pay close attention to patient selection and the proficiency of the operator in these future studies.

Second, there is a need for closer cooperation between urologists and cardiologists who treat patients with CAD and ED. Patients with ED who do not have CAD on CAG but manifest symptoms of CAD should be regarded as high-risk patients who may develop CAD in the future. Therefore, urologists should recognize and explain the correlation between ED and CAD to patients and closely assess a patient's overall health and behavior in addition to performing conventional ED tests. Additionally, to minimize the effects of various risk factors, detailed interviews, behavioral therapy, and drug treatments, such as statin treatment, should be included as part of ED treatment. Cardiologists should also be aware of the correlation between CAD and ED and recommend that patients be asked about ED to receive urological treatment.

The study had several limitations. First, coronary artery stenosis and II-IPA stenosis were observed in a relatively small proportion of the total study population. This may be because the study participants were patients who had undergone both CAG and PA for suspected CAD without considering ED's presence or its severity. However, through the selection of study participants, we believe that we revealed the correlation between coronary artery stenosis and II-IPA stenosis more empirically. Second, venous leakage was not excluded as a confounding cause of ED using penile vascular duplex imaging. Therefore, we were unable to determine that ED in the study patients was entirely due to vascular endothelial dysfunction. Third, we did not assess endothelial cell dysfunction and did not analyze the classification and effects of hypertensive medication that can induce ED in this study. A larger-scale, well-designed, prospective study using a valid endothelial biomarker will provide an opportunity to clearly identify the association and correlation between coronary artery stenosis, II-IPA stenosis, ED, and vascular endothelial dysfunction.

5. Conclusions

This study clearly demonstrated an angiographic correlation between coronary artery stenosis and II-IPA stenosis. We believe that patients with ED and clinically suspected CAD should be comprehensively evaluated with not only conventional ED tests but also through overall health and behavior assessment. Furthermore, we believe that the presence and extent of coronary artery stenosis or II-IPA stenosis is an indicator of more active medical or interventional treatment rather than an indicator of ED severity.

ABBREVIATIONS

ED: erectile dysfunction; CAD: coronary artery disease; NO: nitric oxide; EF: erectile function; II-IPA: internal iliac-internal pudendal arteries; PA: pelvic angiography; CAG: coronary angiography; PSA: prostate specific antigen; IIEF-15: International Index of Erectile Function-15; PCI: percutaneous coronary intervention; PDE5i: phosphodiesterase-5 inhibitor; A-I-P: aorto-ilio-pudendal.

AVAILABILITY OF DATA AND MATERIALS

The data presented in this study are available on reasonable request from the corresponding author.

AUTHOR CONTRIBUTIONS

BHP—designed the study, analyzed the data, and drafted the paper. SHH—performed coronary and pelvic angiography, and supervised the study. DSH—participated in literature survey and analysis of data. SMY—participated in literature survey and writing manuscript. DWK—participated in literature survey and performed coronary and pelvic angiography. CSY—participated in literature survey and analysis of data. HJ—designed the study, supervised all research steps, and drafted the paper.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The present study protocol was reviewed and approved by the institutional review board of The Catholic University of Korea, Daejeon St. Mary's Hospital (IRB No. DC21IRIS0067). As this is a retrospective study, patient consent was not required and it was approved by the IRB.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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