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RESEARCH ARTICLE

Testosterone Deficiency Does Not Predict Penile Curvature or Plaque Size in Men with Peyronie's Disease

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Abstract

Objective: To investigate the association between serum testosterone levels and the severity of penile curvature and plaque size in men with Peyronie's disease (PD).

Materials and Methods: This retrospective cross-sectional study included 108 men diagnosed with Peyronie's disease who presented to our urology outpatient clinic between January 2022 and July 2025. Patients with prior testosterone replacement therapy, pelvic surgery, intralesional treatment, or systemic conditions affecting testosterone metabolism were excluded. Demographic and clinical data—including disease duration, penile pain, erectile dysfunction (assessed by IIEF-5), and disease phase—were recorded. Penile curvature was assessed via self-photographs during natural or pharmacologically induced erections, and plaque size was measured ultrasonographically. Fasting morning serum samples were analyzed for total testosterone (TT), free testosterone (FT), sex hormone-binding globulin (SHBG), luteinizing hormone (LH), follicle-stimulating hormone (FSH), estradiol, and metabolic parameters.

Results: Mean age was 59.6 ± 7.5 years, and mean disease duration was 22.7 ± 29.4 months. Mean horizontal and vertical curvatures were 12.1 ± 19° and 28.5 ± 19.8°, respectively. Mean plaque size was 8.9 ± 7 mm. No significant differences were observed between hypogonadal and eugonadal groups in curvature severity ($p>0.05$), plaque size ($p=0.54$), or disease phase. Hypogonadal men had significantly lower SHBG and estradiol levels and higher HbA1c and triglyceride values (all $p<0.05$). No correlations were found between testosterone levels and curvature degree or plaque dimensions.

Conclusion: Serum testosterone levels are not associated with PD severity. Hypogonadism appears to be a comorbidity rather than a determinant of disease severity, suggesting routine testosterone evaluation may not be necessary in PD management.

Keywords: Peyronie's disease, penile curvature, penile plaque, testosterone levels

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INTRODUCTION

Peyronie's disease (PD) is a connective tissue disorder characterized by the presence of a fibrous inelastic scar involving the tunica albuginea of the penis, which can cause penile curvature, pain, palpable plaque, and erectile dysfunction (1). Reported prevalence varies widely, from well below 1% to over 20%, depending on population and methodology, but most series agree that PD predominantly affects middle-aged and older men, typically between the fifth and sixth decades of life (2-5). Beyond the physical deformity, PD can substantially impair sexual function and intimate relationships, is associated with considerable psychological distress, and negatively affects overall quality of life (5). Although its precise etiology remains uncertain, repeated microvascular trauma during intercourse, followed by abnormal wound healing and excessive collagen deposition, is believed to play a central role (6).

In recent years, attention has turned to possible hormonal influences on the development and severity of PD, particularly testosterone. Androgens affect connective tissue metabolism, endothelial function, and nitric oxide activity, all of which contribute to wound healing and fibrosis regulation (7-9). Some reports have found lower serum testosterone levels among men with PD and suggested an association with greater curvature or plaque burden (7,10), whereas other studies found no significant correlation (9,11,12). The inconsistent evidence leaves unclear whether hypogonadism contributes to PD pathogenesis or merely coexists due to shared risk factors such as age and metabolic comorbidities. This study aimed to determine whether serum testosterone levels are associated with plaque size or the severity of penile curvature in patients with Peyronie's disease.

MATERIALS AND METHODS

Study Design and Population

In this retrospective cross-sectional study, a total of 108 men diagnosed with Peyronie's disease (PD) who presented to our urology outpatient clinic between January 2022 and July 2025 and fulfilled the study inclusion criteria were included. The study was approved by the Scientific Research Ethics Committee of Antalya Training and Research Hospital on 2025-05-08 (Approval number: 2025-172-8/17). Full compliance with the Declaration of Helsinki was ensured, and informed consent was obtained from all patients enrolled in the study. Additionally, all patient data were anonymized, and no personally identifiable information was disclosed. The diagnosis of PD was based on the presence of palpable penile plaques and/or penile deformity confirmed by ultrasonography, physical examination, and clinical history. Patients with a history of pelvic surgery, ongoing testosterone replacement therapy, recent testosterone replacement therapy within the past year, previous intralesional therapy, or systemic conditions known to interfere with testosterone metabolism (such as pituitary or testicular disorders, chronic corticosteroid use, or androgen therapy) were excluded from the study.

Clinical Evaluation

Demographic and clinical characteristics were recorded for all participants, including age, medical comorbidities (hypertension, diabetes mellitus, coronary artery disease, chronic obstructive pulmonary disease), smoking status, and medication history. Disease-specific parameters such as disease duration, presence of penile pain, erectile dysfunction (ED), penile trauma history, and disease phase (active or chronic) were documented. Erectile function was evaluated using the International Index of Erectile Function-5 questionnaire (IIEF-5). The onset of PD was determined by the first instance of pain at erection or the initial finding of a penile plaque or deformity. The direction and degree of penile curvature were assessed from two-axis self-photographs obtained during either natural erection or artificial erection induced by intracavernosal injection of vasoactive agents. The maximal degree of curvature was measured using a goniometer placed at the point of maximal angulation. Both horizontal (lateral) and vertical (dorsal or ventral) curvature angles were recorded. Penile length was measured on stretch from the pubic bone to the tip of the glans. Plaque size was determined by penile ultrasonography. Disease phase was categorized as active (presence of pain and/or deformity progression within the last 3 months) or stable/chronic (≥ 6-12 months after onset of PD or once the deformity has remained stable and painless for ≥ 3 months) (13).

Laboratory Evaluation

Fasting morning venous blood samples were drawn between 07:00 and 11:00 am after overnight fasting. Laboratory assessments included total testosterone (TT), free testosterone (FT), sex hormone-binding globulin (SHBG),

luteinizing hormone (LH), follicle-stimulating hormone (FSH), estradiol (E2), prolactin, and basic metabolic and lipid parameters (hemoglobin, HbA1c, total cholesterol, LDL, HDL, triglycerides). Complete blood count analyses were performed on a Sysmex XN-1000 hematology analyzer (Sysmex Corp., Kobe, Japan). Biochemical parameter analyses were conducted using an enzymatic colorimetric method on an AU5800 analyzer (Beckman Coulter, Florida, USA). Follicle-stimulating hormone (FSH), luteinizing hormone (LH), and total testosterone were measured using a chemiluminescent immunoassay (CLIA) on a UniCel DxI 800 Access immunoassay system (Beckman Coulter, Florida, USA). Free testosterone was measured by a chemiluminescent immunoassay (CLIA) on the Snibe Maglumi X3 immunoassay system (Snibe Company, Shenzhen, China).

In this study, hypogonadism was defined as total testosterone (TT) < 12 nmol/L (≈ 3.5 ng/mL), according to current EAU guidelines (14,15).

Statistical Analysis

Statistical analyses were performed using a commercially available software (SPSS version 25.0, Chicago, IL). The Shapiro-Wilk test was used to determine whether the distribution of continuous variables was normal. The continuous data were presented as mean ± standard deviation (SD). The data that were not normally distributed were given as median and interquartile range, and the Mann-Whitney U test was used to compare. In addition to frequency and percentage distributions of the data, the Student t test was used in group comparisons, and the chi-square test was used for variables between categorical data. P<0.05 was considered to be statistically significant.

RESULTS

A total of 108 men diagnosed with Peyronie's disease were included in the study. The mean age was 59.6 ± 7.5 years, and the mean disease duration was 22.7 ± 29.4 months. The mean IIEF-5 score was 16.1 ± 4.6. Horizontal and vertical curvature degrees were 12.1 ± 19° and 28.5 ± 19.8°, respectively. Mean plaque size, as measured by ultrasonography, was 8.9 ± 7 mm. The mean total testosterone level was 3.88 ± 1.16 ng/mL, and 38.9% (n=42) of the patients were classified as hypogonadal based on EAU guidelines (TT < 12 nmol/L or ≈3.5 ng/mL) (14,15). Demographic, clinical, and laboratory data of the study population are summarized in Tables 1 and 2. In 91 patients, penile curvature was uniplanar, whereas 14 patients had multiplanar curvature. In 3 patients, no curvature was observed; however, these individuals presented with hourglass or notching deformities only.

Table 1. Disease-specific and hematological, biochemical, hormonal parameters of the study group

Parameters	Mean ± SD (min–max)
Age (years)	59.62 ± 7.5 (39-74)
Disease duration (months)	22.66 ± 29.4 (2-120)
ED duration (months)	14.35 ± 18.3(0-120)
IIEF-5 score	16.09 ± 4.6 (6-24)
Horizontal curvature degree (Right/Left)	12.13 ± 19 (0-60)
Vertical curvature degree (Dorsal/Ventral)	28.52 ± 19.8 (0-75)
Penile length (cm)	13.105 ± 0.9 (11-18)
Plaque size on USG (mm)	8.93 ± 7 (2-30)
Hemoglobin (g/dL)	14.705 ± 1.42 (10.8-18.7)
HbA1C (%)	6.169 ± 1.33 (4.5-13.6)
Neutrophil count (/µL)	4603.33 ± 1650.64 (2120-10410)
Lymphocyte count (/µL)	2521.30 ± 833.94 (890-4990)
Platelet count (/µL)	253268.52 ± 65119.1 (125000-432000)
NLR	1.98 ± .87 (0.62-4.78)
PLR	108.39 ± 38.15 (35.07-216.44)
FSH (U/L)	7.52 ± 5.74 (1.96-40.89)
LH (U/L)	5.11 ± 2.49 (1.60-15.18)
Prolactin (µg/L)	8.9671 ± 7.64 (0.23-55.65)
SHBG (nmol/L)	40.237 ± 15.77 (0.94-82.1)
Estradiol (ng/L)	28.95 ± 8.06 (15-60)
Total testosterone (ng/mL)	3.88 ± 1.16 (1.93-7.78)
Free testosterone (pg/mL)	9.31 ± 4.71 (3.36-25.40)
Total cholesterol (mg/dL)	200.94 ± 39.73 (117-362)
Triglyceride (mg/dL)	166.19 ± 99.48 (37-583)
LDL (mg/dL)	124.41 ± 43.04 (51-400)
HDL (mg/dL)	48.42 ± 10.02 (35-85)
Albumin (g/L)	43.630 ± 2.9 (33.7-51.7)

Values are mean ± SD**,** Abbreviations: ED, erectile dysfunction; IIEF-5, International Index of Erectile Function; USG, ultrasonography; HbA1c, hemoglobin A1c; NLR, Neutrophil to Lymphocyte ratio; PLR, Platelet to Lymphocyte ratio; FSH, follicle-stimulating hormone; LH, luteinizing hormone; SHBG, sex hormone binding globulin; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

When comparing hypogonadal (n=42) and eugonadal (n=66) patients, there were no significant differences in age, disease duration, erectile function scores, severity of curvature (horizontal: 13.6 ± 18.9° vs 11.2 ± 19.1°, p=0.53; vertical: 29.4 ± 19.0° vs 27.9 ± 20.4°, p=0.71), or plaque size (8.4 ± 5.8 mm vs 9.3 ± 7.7 mm, p=0.54). Hypogonadal men showed significantly lower SHBG (32.27 ± 12.59 vs 45.30 ± 15.58 nmol/L, p<0.001) and estradiol levels (25.81 ± 6.55 vs 30.95 ± 8.33 pg/mL, p<0.001), and higher HbA1c (6.1 [1] vs 5.7 [0.7] %, p=0.001) and triglyceride values (168 [72] vs 140 [97] mg/dL, p=0.008) than eugonadal men. Detailed comparative data are presented in Table 3.

There were no significant differences between hypogonadal and eugonadal groups with respect to the prevalence of hypertension, diabetes mellitus, coronary artery disease, chronic obstructive pulmonary disease, smoking status, distribution of active versus chronic disease phase, presence of penile pain, or rate of calcified plaques. Detailed comparisons of these clinical characteristics between the two groups are presented in Table 4.

No significant correlations were found between total or free testosterone levels and either the degree of penile curvature or plaque size (all p>0.05).

DISCUSSION

Peyronie's disease is a benign acquired fibrotic condition of the tunica albuginea that leads to penile curvature, pain, palpable plaque, and erectile dysfunction, with prevalence increasing with age (1,2,5). Overall, penile deformity represents the most common initial symptom of Peyronie's disease, occurring in 52–94% of patients. Penile pain is the second most frequent symptom, reported by approximately 20–70% of patients during the early phase of the disease (16). Consistent with the literature, penile deformity was the most common initial symptom in our study (65.7%), followed by pain (20.4%) and erectile dysfunction (13.9%) as the second and third most frequent presenting symptoms, respectively. Kadioglu et al., in a population-based study, reported dorsal and lateral curvatures as the most common deformities, with dorsal curvature observed in 45.6% and lateral curvature in 29.3% of patients (17). Similarly, Moreno and Morgentaler found that the primary direction of curvature was dorsal in 66.9% of cases, followed by ventral in 12.4% and lateral in 8.3% (7). In line with these findings, the most frequent curvature pattern in our cohort was also dorsal curvature, which was identified in 56.5% of patients. (Table 2).

Table 2. Comorbidities and general characteristics of Peyronie's population sample

Characteristics	n (%)
Hypertension	32 (29.6)
Diabetes	35 (32.4)
Coronary artery disease	17 (15.7)
COPD	7 (6.5)
Antiplatelet use	23 (21.3)
Active smoking	43 (39.8)
PDE-5 inhibitor use	47 (43.5)
Disease phase (Active/Chronic)	28(25.9) / 80(74.1)
Penile pain	25 (23.1)
History of penile trauma	8 (7.4)
Horizontal curvature (Right/Left)	7(6.5) / 28(25.9)
Vertical curvature (Dorsal/Ventral)	61(56.5) / 23(21.3)
Curvature interfering with intercourse	41 (38)
Concomitant LUTS	25 (23.1)
Initial symptom (Pain/Deformity/ED)	22(20.4) / 71(65.7) / 15(13.9)
Notching/ Hourglass deformity	20 (18.5)
Calcified plaque	44 (40.7)
Hypogonadism	42 (38.9)

Abbreviations: COPD, Chronic obstructive pulmonary disease; PDE-5 inh, Phosphodiesterase type 5 inhibitor; LUTS, Lower urinary tract symptoms; ED, Erectile dysfunction.

Table 3. Comparison between hypogonadal and eugonadal patients according to Disease-specific and Hematological, biochemical, hormonal parameters

Parameters	Hypogonadal (n=42)	Eugonadal (n=66)	p
Normally distributed variables (mean ± SD)			
Age (years)	59.86 ± 6.0	59.47 ± 8.3	0.795
IIEF-5 score	16.12 ± 4.7	16.08 ± 4.6	0.962
Horizontal curvature degree (Right/Left)	13.57 ± 18.9	11.21 ± 19.1	0.532
Vertical curvature degree (Dorsal/Ventral)	29.40 ± 19.0	27.95 ± 20.4	0.712
Plaque size on USG (mm)	8.40 ± 5.8	9.26 ± 7.7	0.541
Hemoglobin (g/dL)	14.60 ± 1.7	14.76 ± 1.2	0.572
Neutrophil count (/μL)	4978.33 ± 1652.36	4364.70 ± 1616.86	0.059
Lymphocyte count (/μL)	2529.29 ± 802.78	2516.21 ± 859.21	0.937
NLR	2.14 ± 0.92	1.87 ± 0.82	0.115
PLR	108.16 ± 34.26	108.54 ± 40.69	0.959
SHBG (nmol/L)	32.27 ± 12.59	45.30 ± 15.58	**<0.001**
Estradiol (ng/L)	25.81 ± 6.55	30.95 ± 8.33	**<0.001**
Total testosterone (ng/mL)	2.81 ± 0.40	4.56 ± 0.96	**<0.001**
Free testosterone (pg/mL)	6.82 ± 3.63	10.89 ± 4.66	**<0.001**
Total cholesterol (mg/dL)	204.62 ± 30.67	198.61 ± 44.62	0.445
Albumin (g/L)	43.73 ± 3.04	43.56 ± 2.83	0.768
Non-normally distributed variables (median [IQR])			
Disease duration (months)	12 [18]	12 [11]	0.207
ED duration (months)	12 [16]	12 [15]	0.801
Penile length (cm)	13 [1]	13 [0.5]	0.054
HbA1C (%)	6.1 [1]	5.7 [0.7]	**0.001**
Platelet count (/μL)	247500 [88250]	249000 [100500]	0.816
FSH (U/L)	6.52 [4.72]	6.3 [3.9]	0.733
LH (U/L)	4.5 [1.92]	4.5 [3.1]	0.398
Prolactin (μg/L)	7.3 [5.4]	7.8 [6]	0.712
Triglyceride (mg/dL)	168 [72]	140 [97]	**0.008**
LDL (mg/dL)	128.5 [42]	116 [51]	0.298
HDL (mg/dL)	46 [11]	47 [13]	0.661

Data are presented as mean ± SD for normally distributed variables and as median [IQR] for non-normally distributed variables. Normality was assessed using the Shapiro–Wilk test. Abbreviations: IQR, interquartile range; ED, erectile dysfunction; IIEF-5, International Index of Erectile Function; USG, ultrasonography; HbA1c, hemoglobin A1c; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; FSH, follicle-stimulating hormone; LH, luteinizing hormone; SHBG, sex hormone-binding globulin; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

Table 4. Comparison between hypogonadal and eugonadal patients according to comorbidities and general characteristics of PD

Characteristics	Hypogonadal (n=42)	Eugonadal (n=66)	p
Hypertension	14 (33.3%)	18 (27.3%)	0.523
Diabetes	17 (40.5%)	18 (27.3%)	0.206
Coronary artery disease	6 (14.3%)	11 (16.7%)	0.793
COPD	4 (9.5%)	3 (4.5%)	0.427
Antiplatelet use	11 (26.2%)	12 (18.2%)	0.344
Active smoking	16 (38.1%)	27 (40.9%)	0.842
PDE-5 inhibitor use	17 (40.5%)	30 (45.5%)	0.692
Disease phase (Active/Chronic)	7 (16.7%) / 35 (83.3%)	21 (31.8%) / 45 (68.2%)	0.114
Penile pain	7 (16.7%)	18 (27.3%)	0.247
History of penile trauma	2 (4.8%)	6 (9.1%)	0.479
Horizontal curvature (Right/Left)	3 (7.1%) / 12 (28.6%)	4 (6.1%) / 16 (24.2%)	0.842
Vertical curvature (Dorsal/Ventral)	25 (59.5%) / 8 (19)	36 (54.5%) / 15 (22.7%)	0.863
Curvature interfering with intercourse	16 (38.1%)	25 (37.9%)	0.982
Concomitant LUTS	13 (31%)	12 (18.2%)	0.161
İnitial symptom (Pain/Deformity/ED)	7 (16.7%) / 31 (73.8%) / 4 (9.5%)	15 (22.7%) / 40 (60.6%) / 11 (16.7%)	0.352
Notching/ Hourglass deformity	9 (21.4%)	11 (16.7%)	0.353
Calcified plaque	18 (42.9%)	26 (39.4%)	0.437

Abbreviations: COPD, Chronic obstructive pulmonary disease; PDE-5 inh, Phosphodiesterase type 5 inhibitor; LUTS, Lower urinary tract symptoms; ED, Erectile dysfunction.

It is widely recognized that PD plaque development may originate from penile trauma or repeated microvascular injury during erection (6,18), leading to an imbalance between profibrotic and antifibrotic pathways. This dysregulation—particularly involving transforming growth factor-β1 (TGF-β1)—promotes excessive collagen deposition and subsequent plaque formation (19); however, the pathogenesis of PD is likely multifactorial, involving an interplay among genetic predisposition, mechanical injury, local inflammatory processes, and dysregulated wound healing (13). Clinical studies have identified several comorbid conditions that cluster with PD, including diabetes mellitus, hypertension, dyslipidemia, hypogonadism, smoking, Dupuytren's contracture, and other systemic fibrotic or autoimmune disorders, suggesting that local penile pathology develops on a background of systemic vascular and connective tissue susceptibility (8,20,21).

The relationship between testosterone and PD has attracted particular interest over the past decade. Androgens are known to influence tissue repair and collagen metabolism, with experimental and clinical data linking androgen deficiency to impaired wound healing and increased fibrosis (22,23). These findings support a theoretical basis for the hypothesis that low testosterone may predispose to fibrotic tissue remodeling and PD development. Moreover, testosterone deficiency may contribute indirectly to PD via diminished erectile rigidity, thereby increasing susceptibility to repetitive penile trauma during sexual activity (24). Additionally, androgens have been shown to positively regulate nitric oxide (a potent anti-fibrotic mediator), which further supports the protective role of testosterone against fibrotic processes (25).

However, the clinical evidence regarding the association between testosterone levels and PD has been conflicting. Moreno and Morgentaler reported that 74% of men with

PD had low testosterone (defined by either total or free testosterone), with a significant correlation between low free testosterone and penile curvature severity (54.3° vs. 37.1°, p = 0.006) (7). Similarly, Nam et al. found that patients with testosterone deficiency had a significantly greater mean degree of penile curvature than those with normal testosterone levels (32.0 ± 16.9° vs. 21.8 ± 15.4°, p = 0.033), and a higher prevalence of moderate to severe curvature (40% vs. 23.7%, p = 0.015), proposing mechanisms such as reduced nitric oxide activity and upregulated TGF-β1 expression in hypogonadal states (26). Cavallini et al. reported that PD patients had lower bioavailable testosterone compared to controls, with larger plaque size in hypogonadal men, while penile curvature did not differ significantly. They also reported improved treatment outcomes with combined testosterone replacement and intralesional verapamil (8).

In contrast, more recent studies have largely failed to confirm these associations, aligning closely with our findings. Kirby et al. found no difference in curvature severity between hypogonadal and eugonadal PD patients (35.4° vs. 34.0°, p = 0.70) and comparable testosterone levels between men with PD and age-matched men with isolated erectile dysfunction (328 vs. 332 ng/dL, p = 0.98), suggesting that low testosterone may represent a common feature of sexual dysfunction rather than a PD-specific phenomenon (11). Mulhall et al., in a rigorous analysis of 184 men with PD undergoing intracavernosal injection-induced erections, found no association between total or free testosterone levels and the magnitude of penile deformity (r = −0.01, p = 0.95) (12). Similarly, Candela et al. analyzed 149 men with chronic-phase PD and observed no correlation between testosterone levels and penile curvature across testosterone quartiles (p = 0.31), with only disease duration independently predicting deformity severity (9). Can et al. also found no significant association between testosterone and plaque dimensions or curvature degree in 147 PD patients, despite lower mean testosterone levels compared to controls (3.9 ± 1.1 vs. 4.2 ± 1.7 ng/mL, p = 0.062) (27). In a recent study, Schneider et al. evaluated the impact of testosterone on collagenase clostridium histolyticum (CCH) treatment outcomes in 36 men with PD and found that neither baseline testosterone levels nor hypogonadal status (<300 ng/dL) predicted treatment response, with no significant difference in curvature improvement between hypogonadal and eugonadal groups (p = 0.41) (28). Our study, consistent with these studies, reinforces the prevailing evidence that serum testosterone levels are not associated with objective measures of PD severity. In our cohort of 108 men, 38.9% were classified as hypogonadal according to EAU guidelines (total testosterone <12 nmol/L or approximately 3.5 ng/mL). Despite this substantial prevalence of low testosterone, we observed no significant differences between hypogonadal and eugonadal groups in horizontal curvature (13.6° vs. 11.2°, p = 0.53), vertical curvature (29.4° vs. 27.9°, p = 0.71), plaque size (8.4 mm vs. 9.3 mm, p = 0.54), or disease phase distribution. Furthermore, correlation analysis revealed no association between total or free testosterone levels and either the degree of penile curvature or plaque dimensions, suggesting that hormonal status does not influence the anatomical manifestations of PD.

In the literature, several studies have investigated comorbidities associated with PD (17,29), including hyperlipidemia as a potential risk factor, though findings remain inconsistent. While Rhoden et al. found no correlation between serum lipid profiles and PD, Can et al. reported significantly elevated LDL levels in PD patients (27,30). In our study, no significant differences were observed between eugonadal and hypogonadal groups regarding hypertension, diabetes, coronary artery disease, or COPD. However, hypogonadal men exhibited significantly lower SHBG and estradiol levels, along with higher HbA1c and triglyceride values. These findings suggest that hypogonadism may be associated with a distinct metabolic profile characterized by impaired glucose regulation and dyslipidemia, potentially contributing to PD pathophysiology through altered tissue repair and increased fibrotic remodeling (29).

Our study has several limitations. First, the retrospective design may have introduced selection bias. Second, the absence of a healthy control group and comparisons exclusively between PD subgroups may limit the generalizability of our findings. Third, plaque measurements by multiple radiologists may have introduced inter-observer variability, potentially affecting the consistency of radiological assessments.

CONCLUSION

In conclusion, our findings suggest that serum testosterone levels are not associated with the severity of penile curvature or plaque dimensions in men with Peyronie's disease.

Hypogonadism appears to represent a comorbid condition rather than a determinant factor in disease severity. These results have important clinical implications, as they suggest that testosterone evaluation and supplementation may not be necessary as part of routine PD management unless indicated for other reasons. Further prospective, controlled studies are warranted to definitively clarify the relationship between testosterone and PD pathophysiology.

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Conflict of Interest: The authors report no conflict of interest.

Informed Consent: All patients participating in the study were informed about the study, and their informed consent was obtained.

Ethical Approval: The study was approved by the Clinical Research Ethics Committee of Antalya Training and Research Hospital (Approval No: 2025-172-8/17, Date: 2025-05-08).

Author Contributions:

- Concept and Design: MŞ, ST
- Supervision: ST, ŞK, AE
- Data Collection and/or Analysis: BA, MRİ, AE
- Analysis and/or Interpretation: MŞ, AE, ÇÖ
- Literature Search: MŞ, BA, ST
- Writing: MŞ
- Critical Review: AE, ÇÖ, ŞK

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