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Does size matter in prostate cancer? A cross-sectional study on genital dimensions in Caucasian men

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Abstract

Background: Testosterone is crucial in male genital development during puberty. This study primarily aimed to compare genital size in Caucasian men with and without clinically significant prostate cancer (csPCa). The secondary aim was to assess genital size differences in csPCa patients stratified by tumor grade. **Methods**: We conducted an observational cross-sectional study on consecutive patients undergoing prostate biopsy. Each patient underwent systematic and cognitive-targeted transrectal biopsy. csPCa was defined as PCa with Gleason Score (GS) $\geq$7, tumor volume $\geq$0.5 cc, or extra-prostatic extension; other PCa and negative biopsies were classified as non-csPCa. Penile length was measured with the stretched test, and testicular volume by ultrasonography. **Results**: A total of 156 patients (64 csPCa and 92 non-csPCa) were enrolled. Median age was comparable between groups (68.9 *vs.* 66.6 years; $p = 0.289$). Median (IQR) stretched penile length (SPL) was 15.0 (14.0–16.3) cm in the csPCa group and 15.0 (13.0–15.2) cm in the non-csPCa group ($p = 0.301$). Median (IQR) right testicular volume was 12 (10–14) cc in both groups ($p = 0.752$), while left testicular volume was 10 (7–13) cc in the csPCa group and 12 (10–14) cc in the non-csPCa group ($p = 0.172$). Among csPCa patients, those with GS >7 had a longer SPL (16.0 (15–17) cm) compared to GS = 7 patients (14.5 (14–15) cm) ($p = 0.012$), with no significant differences in testicular volume. **Conclusions**: In Caucasian men, genital dimensions did not differ between those with and without csPCa. Within the csPCa group, longer SPL was observed in patients with higher-grade tumors. These findings suggest a possible association between genital size and tumor aggressiveness, which warrants further investigation.

Keywords

Androgens; Clinically significant prostate cancer; Penile length; Tumor grade

¿Importa el tamaño en el cáncer de próstata? Un estudio transversal sobre las dimensiones genitales en hombres caucásicos

Resumen

Antecedentes: La testosterona desempeña un papel fundamental en el desarrollo genital masculino durante la pubertad. El objetivo principal de este estudio fue comparar las dimensiones genitales en varones caucásicos con y sin cáncer de próstata clínicamente significativo (CPCs). Como objetivo secundario, se evaluaron las diferencias en las dimensiones genitales de los pacientes con CPCs estratificados según el grado tumoral. **Métodos**: Se llevó a cabo un estudio observacional y transversal en pacientes consecutivos sometidos a biopsia prostática . Todos los pacientes fueron sometidos a biopsia prostática sistemática y dirigida cognitiva por vía transrectal. El CPCs se definió como CP con puntuación de Gleason (GS) ≥7, volumen tumoral ≥0.5 cc o extensión extraprostática; el resto de los casos y las biopsias negativas se clasificaron como no CPCs. La longitud peneana en estiramiento (LPE) se midió clínicamente y el volumen testicular mediante ecografía. **Resultados**: Se incluyeron 156 pacientes (64 CPCs y 92 no CPCs). La mediana de edad fue comparable entre grupos (68.9 *vs.* 66.6 años; $p = 0.289$). La LPE mediana (RIQ) fue de 15.0 (14.0–16.3) cm en el grupo con CPCs y de 15.0 (13.0–15.2) cm en el grupo sin CPCs ($p = 0.301$). El volumen testicular derecho fue de 12 (10–14) cc en ambos grupos ($p = 0.752$), mientras que el izquierdo fue de 10 (7–13) cc en el grupo con CPCs y de 12 (10–14) cc en el grupo sin CPCs ($p = 0.172$). Entre los pacientes con CPCs, aquellos con GS >7 presentaron una LPE mediana (RIQ) de 16.0 (15–17) cm frente a 14.5 (14–15) cm en los pacientes con GS = 7 ($p = 0.012$), sin diferencias significativas en el volumen testicular. **Conclusiones**: En varones caucásicos, las dimensiones genitales no difirieron entre quienes presentaban o no CPCs. Sin embargo, dentro del grupo con CPCs, los pacientes con tumores de mayor grado mostraron una LPE más larga. Estos hallazgos sugieren una posible asociación entre el tamaño genital y la agresividad tumoral, que merece ser explorada en investigaciones futuras.

Palabras Clave

Andrógenos; Cáncer de próstata clínicamente significativo; Longitud peneana; Grado tumoral

1. Introduction

Prostate cancer (PCa) is the most frequently diagnosed non-cutaneous malignancy in men and a leading cause of cancer-related death worldwide, with incidence and mortality varying considerably across regions and ethnic groups [1, 2]. While age, family history, and related genetic factors remain the most established risk determinants, compelling evidence highlights the central role of androgen signaling in both prostate physiology and carcinogenesis [3, 4].

Testosterone and its potent metabolite, dihydrotestosterone (DHT), are critical for prostate growth and the development of male external genitalia during fetal life and puberty [5]. Penile elongation and testicular growth, largely completed by late adolescence, reflect cumulative androgen exposure during key developmental periods [6]. Experimental models have shown that increased early-life androgen stimulation may induce prostatic epithelial proliferation and, in some settings, precancerous changes [7].

Ethnic disparities in both genital dimensions and PCa epidemiology have been consistently documented. Reference values for adult stretched penile length (SPL) and testicular volume have been reported in population studies, showing correlations with androgen exposure during development and adulthood [8]. African men, on average, present with greater SPL, larger testicular volume, higher adolescent free testosterone levels, and earlier pubertal onset compared to Caucasians [9–11]. Notably, these same populations also experience a disproportionately higher incidence of PCa, with a greater proportion of high-grade tumors compared with Caucasians [12]. This overlap suggests a potential biological link between early androgenic imprinting and tumor aggressiveness.

Despite these observations, no study to date has systematically compared genital dimensions potentially serving as surrogate markers of developmental androgen exposure between men with and without clinically significant prostate cancer (csPCa) in a Caucasian population. Addressing this gap may provide novel insights into the hormonal underpinnings of PCa biology and inform future hypotheses on risk stratification.

The primary aim of this study was to compare male genital size, specifically stretched penile length and testicular volume, between Caucasian men with and without csPCa at biopsy. The secondary objective was to compare these measurements between Caucasian patients with csPCa stratified by tumor grade.

2. Materials and methods

2.1 Study design and ethical details

We designed an observational, cross-sectional pilot study involving consecutive Caucasian men undergoing prostate biopsy for suspected PCa at our Institution (University of Campania Luigi Vanvitelli, Naples, Italy) between January 2023 and June 2025. The study was purely observational and non-interventional, and was therefore deemed exempt from formal review by the institutional ethics committee. It was conducted in accordance with the principles of the Declaration of Helsinki and relevant national regulations [13]. Written informed consent was obtained from all participants for inclusion of their data in a dedicated research database and for publication of anonymized results. All patients received appropriate counseling and informed consent about the biopsy procedure. All data were de-identified and stored in a secure institutional database compliant with General Data

Protection and Regulation (GDPR). Written informed consent explicitly covered sensitive genital measurements and the use of anonymized data for research and publication.

2.2 Patient enrollment

Consecutive Caucasian men aged ≥50 years who underwent prostate biopsy for suspected PCa were considered for inclusion. Exclusion criteria included congenital penile curvature, congenital genital malformations, Peyronie's disease, history of priapism, history of penile tumor, prior penile surgery for curvature, tumor, or augmentation, history of cryptorchidism, history of testicular torsion, prior orchidopexy, history of testicular tumor, prior unilateral or bilateral orchiectomy, clinically significant varicocele or prior varicocelectomy, known chromosomal disorders, diagnosed hypogonadism, current or recent (≤12 months) treatment with testosterone therapy, Gonadotropin-Releasing Hormone (GnRH) analogs or antagonists, anti-androgens, estrogens, or use of anabolic-androgenic steroids for doping purposes, and inability or unwillingness to undergo pre-biopsy multiparametric magnetic resonance imaging (MRI). Patients with missing data for primary outcomes or key covariates were also excluded.

2.3 Patient evaluation

Baseline variables collected included age, body mass index (BMI), smoking status, family history of PCa or breast cancer (BCa), serum prostate-specific antigen (PSA), findings on digital rectal examination (DRE), Prostate Imaging Reporting and Data System (PI-RADS) version 2.1 [14] score on pre-biopsy multiparametric magnetic resonance imaging (mpMRI), prostate volume, total testosterone, use of 5α-reductase inhibitors (5-ARI), history of prior benign prostatic hyperplasia (BPH) surgery, and biopsy-naive status. SPL was measured in the supine position using the standard stretched penile length technique, with the penis gently extended to the point of resistance from the pubic bone to the tip of the glans along the dorsal surface. Bilateral testicular volume was assessed by high-resolution scrotal ultrasonography using the ellipsoid formula (length × width × height × 0.52). Scrotal ultrasound was performed using a (1202 Flex Focus 400 Ultrasound digital and multipurpose Scanner (BK Medical, Milan, Italy)), equipped with a (2–8 MHz) linear-array transducer. All genital measurements were performed by a single trained urologist following a standardized protocol to minimize inter-observer variability. The primary outcome was the presence of csPCa defined as Gleason score (GS) ≥7, tumor volume ≥0.5 cc, and/or evidence of extraprostatic extension [14]. PCa lacking these characteristics or negative biopsy findings including cases with prostatic intraepithelial neoplasia (PIN) or atypical small acinar proliferation (ASAP) were classified as non-csPCa.

All biopsies were performed in an outpatient setting by a single experienced urologist using a combined systematic and cognitive-targeted transrectal approach. All histopathological specimens were reviewed by a single dedicated genitourinary pathologist with extensive expertise in PCa diagnosis to ensure uniformity in tumor detection and grading.

2.4 Statistical analysis

Sample size was calculated to detect a clinically meaningful difference in SPL between patients with and without csPCa. Based on published literature, the standard deviation (σ) of SPL in adult men was estimated at 2.0 cm, and a difference of 1.0 cm was considered clinically relevant [15]. Assuming a two-sided α of 0.05, a power of 80%, and an expected 40:60 ratio of csPCa to non-csPCa patients based on previous biopsy cohorts [16], the required sample size was determined using a two-sample *t*-test formula adapted for unequal group sizes. This calculation yielded a minimum of 53 patients with csPCa and 80 patients without csPCa, for a total of 133 participants. Normality of continuous variables was assessed using the Shapiro-Wilk test [17]. Continuous variables were reported as medians and interquartile ranges (IQRs), whereas categorical variables were expressed as absolute frequencies and percentages. Comparisons between groups were performed using the Chi-square test for categorical variables and the Mann-Whitney U test for non-normally distributed continuous variables. For the comparison between patients with and without csPCa, and between patients with GS 7 and GS >7, results were visually summarized using box-and-whisker plots with overlaid individual values, displaying medians, IQR, and whiskers extending to 1.5 × IQR. A p-value < 0.05 was arbitrarily set to indicate statistical significance. RStudio v. 2024.09.0+375 was used for statistical analyses (now Posit, Boston, MA, USA). We prespecified unadjusted analyses, as the sample size did not allow for reliable multivariable modelling; this is recognized as a limitation.

3. Results

A total of 156 patients were enrolled, including 64 with csPCa and 92 with non-csPCa (Table 1). Median (IQR) age did not differ significantly between groups (csPCa: 68.9 [59.6–76.4] *vs.* non-csPCa: 66.6 [60.4–72.3] years; $p = 0.289$). A family history of PCa or BCa was significantly more prevalent in the csPCa group (18.8% *vs.* 4.3%, $p = 0.008$). No significant differences were observed in total testosterone levels ($p = 0.898$) or BMI ($p = 0.152$). Men with csPCa had significantly higher median PSA levels (8.0 [6.1–13.8] *vs.* 5.6 [4.4–7.0] ng/mL, $p < 0.001$) and PI-RADS scores (4 [4–5] *vs.* 3 [2–3], $p < 0.001$) compared with non-csPCa patients. The two groups did not differ in the prevalence of 5-ARI use ($p = 0.976$) or prostate volume ($p = 0.140$) (Table 2).

Median SPL was 15.0 [14.0–16.3] cm in the csPCa group and 15.0 [13.0–15.2] cm in the non-csPCa group ($p = 0.301$). Median right testicular volume was 12.0 [10.0–14.0] cc in both groups ($p = 0.752$), while median left testicular volume was 10.0 [7.0–13.0] cc in the csPCa group and 12.0 [10.0–14.0] cc in the non-csPCa group ($p = 0.172$) (Table 3, Fig. 1). Within the csPCa group, 36 patients had GS 7 and 28 had GS >7. These secondary subgroup analyses are exploratory and underpowered. Within the csPCa subgroup, patients with GS >7 had a significantly greater median SPL compared with those with GS 7 (16.0 [15.0–17.0] *vs.* 14.5 [14.0–15.0] cm, $p = 0.012$). No significant differences were observed between GS categories in right testicular volume (11.0 [9.0–13.0] *vs.*

TABLE 1. Histological findings on prostate biopsy.

	Total (n = 156)	csPCa[a] (n = 64)	non-csPCa[b] (n = 92)
GS 6, n (%)	38 (24.4)	0 (0)	38 (41.3)
GS 7, n (%)	36 (23.1)	36 (56.2)	0 (0)
GS 8–10, n (%)	28 (17.9)	28 (43.8)	0 (0)
Subtotal of PCa, n (%)	102 (65.4)	64 (100)	38 (100)
PIN, n (%)	8 (5.1)	0 (0)	8 (8.7)
ASAP, n (%)	6 (3.8)	0 (0)	6 (6.5)
Chronic inflammation, n (%)	36 (23.1)	0 (0)	36 (39.1)
Others, n (%)	4 (2.6)	0 (0)	4 (4.3)

ASAP: Atypical Small Acinar Proliferation; cs: clinically significant; GS: Gleason Score; PCa: Prostate Cancer; PIN: Prostatic Intraepithelial Neoplasia.
[a] PCa with GS ≥7 and/or volume ≥0.5 cc and/or extraprostatic extension; [b] PCa without features of csPCa and negative biopsies.

TABLE 2. Baseline characteristics of patients.

	Total (n = 156)	csPCa[a] (n = 64)	non-csPCa[b] (n = 92)	*p*-value
Age, yr, Median (IQR)	67.3 (60.1–74.0)	68.9 (59.6–76.4)	66.6 (60.4–72.3)	0.289
Ethnicity (Caucasian), n (%)	156 (100)	64 (100)	92 (100)	/
Family history for PCa or BCa, n (%)	16 (10.3)	12 (18.8)	4 (4.3)	**0.008**
Smoking, n (%)	86 (55.1)	38 (59.4)	48 (52.2)	0.468
BMI, points, Median (IQR)	28.3 (23.4–32.0)	27.5 (22.8–30.3)	28.5 (23.9–32.2)	0.152
Total testosterone, ng/mL, Median (IQR)	5.36 (3.91–6.44)	5.22 (3.86–6.53)	5.41 (4.10–6.23)	0.898
5ARI, n (%)	23 (14.7)	10 (15.6)	13 (14.1)	0.976
PSA, ng/mL, Median (IQR)	6.4 (4.6–8.4)	8.0 (6.1–13.8)	5.6 (4.4–7.0)	**<0.001**
Suspicious DRE, n (%)	63 (40.4)	27 (42.2)	36 (39.1)	0.828
PI-RADS, Median (IQR)	4 (3–4)	4 (4–5)	3 (2–3)	**<0.001**
Prostate volume, cc, Median (IQR)	46 (37.5–78.5)	44.5 (35.3–79.5)	47.0 (40.0–76.5)	0.140
Prostate biopsy naive, n (%)	133 (85.3)	58 (90.6)	75 (81.5)	0.177
Prior BPH surgery, n (%)	13 (8.3)	6 (9.4)	7 (7.6)	0.921

5ARI: 5α-Reductase Inhibitors; BCa: Breast Cancer; BMI: Body Mass Index; BPH: Benign Prostatic Hyperplasia; cs: clinically significant; DRE: Digital Rectal Examination; IQR: Interquartile Range; PCa: Prostate Cancer; PI-RADS: Prostate Imaging Reporting and Data System; PSA: Prostate-Specific Antigen.
[a] PCa with GS ≥7 and/or volume ≥0.5 cc and/or extraprostatic extension; [b] PCa without features of csPCa and negative biopsies; Statistically significant values were reported in bold.
Chi-squared test and Mann-Whitney U Test were used to compare the groups (csPCa vs. non-csPCa).

TABLE 3. Male genital size and csPCa.

	Total (n = 156)	csPCa[a] (n = 64)	non-csPCa[b] (n = 92)	*p*-value
Stretched penis length, cm, Median (IQR)	15 (13.5–16.1)	15 (14.2–16.3)	15 (12.9–15.2)	0.301
Right testicular volume, cc, Median (IQR)	12 (10–14)	12 (10–14)	12 (10–14)	0.752
Left testicular volume, cc, Median (IQR)	11 (8–13)	10 (7–13)	12 (10–14)	0.172

cs: clinically significant; IQR: Interquartile Range; PCa: Prostate Cancer.
[a] PCa with GS ≥7 and/or volume ≥0.5 cc and/or extraprostatic extension; [b] PCa without features of csPCa and negative biopsies.
Mann-Whitney U Test was used to compare the groups (csPCa vs. non-csPCa).

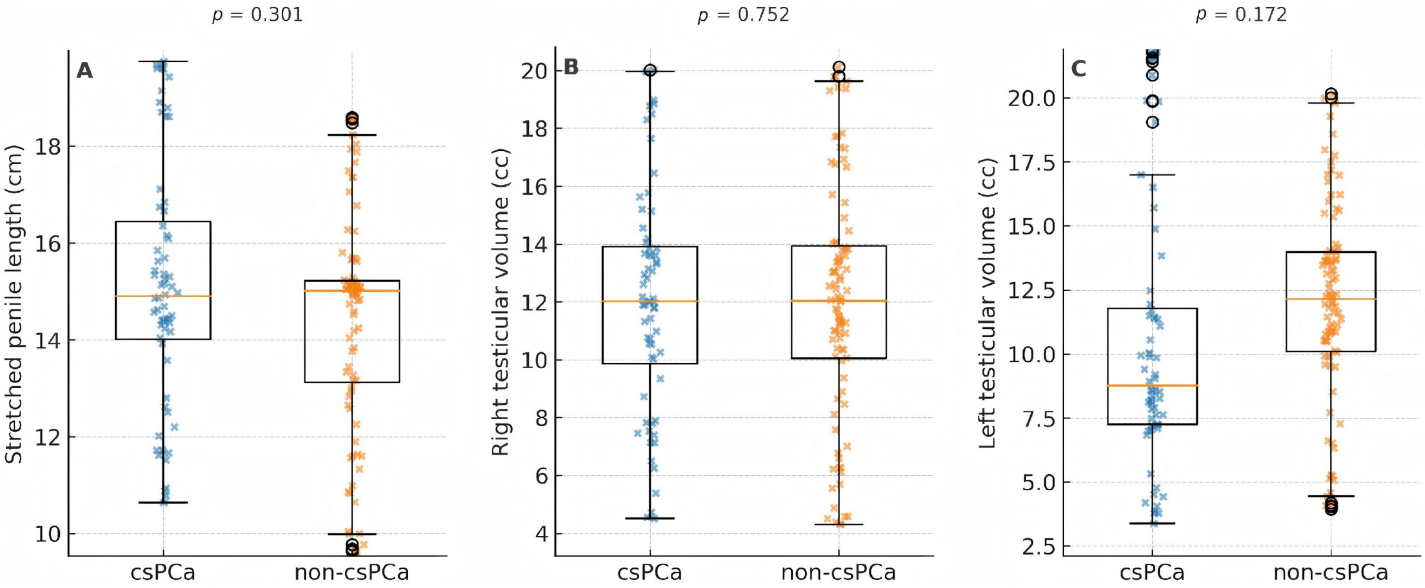

FIGURE 1. Primary outcome—SPL and bilateral testicular volumes in men with and without csPCa. Box-and-whisker plots illustrating SPL (A) and right (B) and left (C) testicular volumes in Caucasian men with and without csPCa on prostate biopsy. Boxes represent the IQR, horizontal lines within boxes indicate the median, and whiskers extend to 1.5 × IQR. Individual patient values are overlaid as jittered points. *p*-values were calculated using the Mann-Whitney U test. cs: clinically significant; PCa: Prostate Cancer.

12.0 [10.0–14.0] cc, $p = 0.128$) or left testicular volume (11.0 [8.0–13.0] *vs.* 11.5 [9.0–13.0] cc, $p = 0.244$) (Fig. 2).

4. Discussion

4.1 Main findings and previous literature

In this cross-sectional study of Caucasian men, genital dimensions—SPL and testicular volume—were not significantly associated with the overall presence of csPCa. However, within the csPCa subgroup, men with high-grade tumors (GS >7) had significantly longer SPL, pointing toward a potential role of developmental androgenic influences on tumor severity. Although ours is the first study to directly examine SPL and testicular volume in relation to both the presence and aggressiveness of csPCa, several lines of evidence support the biological plausibility of our findings. Our findings should be interpreted in light of normative genital dimensions and their known hormonal correlates, as reported in recent systematic reviews. Anogenital distance (AGD) a well-established proxy for prenatal androgen exposure has been inversely related to PCa risk. In a Spanish case-control study, each 5-mm increase in AGD was associated with an approximately 17% reduction in the odds of PCa (OR = 0.83; 95% CI: 0.70–0.99) [18]. Additionally, AGD correlates with genital dimensions in healthy young men, reinforcing its value as a marker of early androgenic programming. Moreover, racial disparities in prostate cancer may reflect differences in pubertal androgen exposure. Seminal studies have shown that African American men have higher circulating testosterone levels in early adulthood approximately 15% greater than white counterparts which may explain their roughly twofold elevated PCa risk [19]. Prospective research also indicates that African American males attain higher peak testosterone earlier and may experience more rapid declines with age compared to white males [20], potentially underpinning the heightened vulnerability to the aggressive disease. In parallel, traditional anthropometric factors have long been linked to PCa prognosis. Data from Swedish cohorts (n ≈ 431,000) indicate that taller stature modestly increases risk for localized disease, while obesity significantly raises the risk of PCa-specific mortality (Hazard Ratio (HR) per 5 kg/m^2 = 1.11–1.22) [21]. Likewise, findings from the European Prospective Investigation into Cancer and Nutrition (EPIC) cohort uphold associations between height, BMI, and aggressive PCa forms [22]. Together, these findings on prenatal androgen exposure (AGD), pubertal testosterone patterns, and systemic growth metrics suggest that genital anthropometry such as SPL may serve as a surrogate for androgen-related developmental imprinting relevant to PCa biology. Our observation that longer SPL is linked solely with higher-grade tumors aligns with this framework. However, these results are exploratory in nature and warrant validation in larger, ethnically diverse, longitudinal studies incorporating endocrine and definitive histological data.

4.2 Strengths and limitations

To our knowledge, while several anthropometric parameters have been investigated in relation to PCa [23], this is the first study to specifically examine SPL and testicular volume in connection with both the presence and pathological aggressiveness of clinically significant prostate cancer within a Caucasian population. The study employed a cross-sectional design, with data collected in real time according to a predefined protocol, thereby reducing the risk of bias associated with retrospective analyses. Consecutive patient enrollment, use of standardized and validated measurement tools (includ-

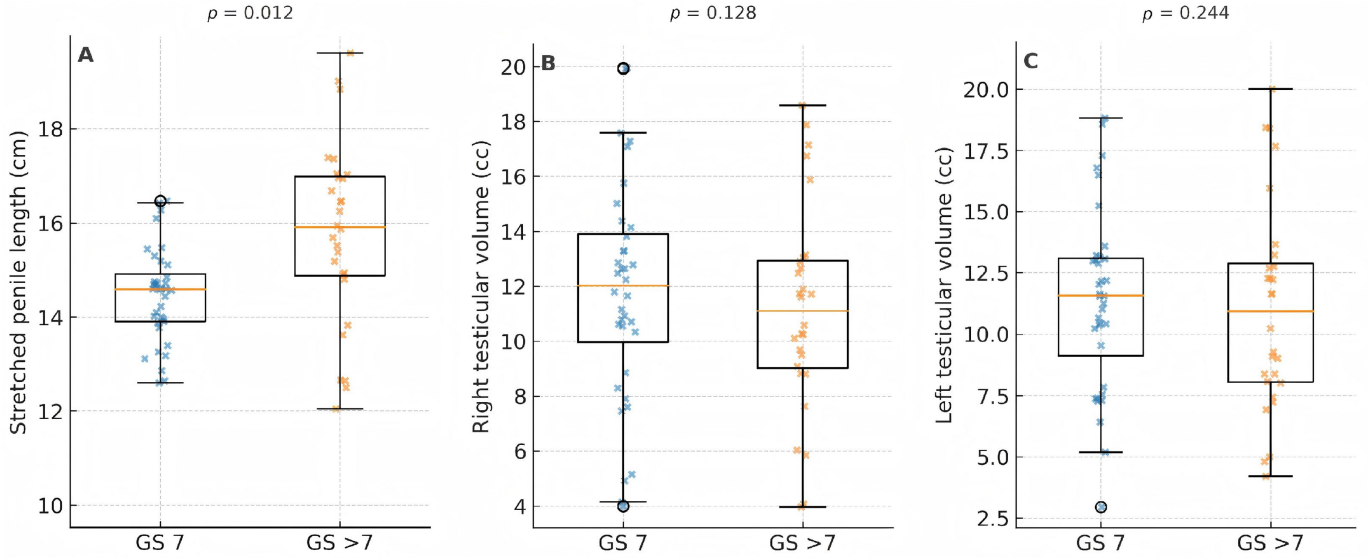

FIGURE 2. Secondary outcome—SPL and bilateral testicular volumes in men with csPCa stratified by tumor grade. Box-and-whisker plots illustrating SPL (A) and right (B) and left (C) testicular volumes in Caucasian men with csPCa on prostate biopsy stratified by GS (GS 7 *vs.* >7). Boxes represent the IQR, horizontal lines within boxes indicate the median, and whiskers extend to 1.5 × IQR. Individual patient values are overlaid as jittered points. *p*-values were calculated using the Mann-Whitney U test. GS: Gleason Score.

ing stretched penile length assessment and ultrasound-derived testicular volume), pre-biopsy mpMRI evaluation with PI-RADS v2.1 scoring, and histopathological review by a single experienced genitourinary pathologist strengthen the internal validity and reproducibility of our findings.

However, several limitations should be acknowledged. First, the single-center setting and ethnically homogeneous sample limit external validity and generalizability to other ethnic groups. Second, although the total sample size (n = 156) met the a priori power calculation for the primary analysis, the subgroup of patients with high-grade tumors (GS >7) was relatively small, potentially reducing the power of secondary analyses. Third, the absence of direct longitudinal data on androgen exposure during critical developmental windows precludes causal inference regarding early-life hormonal imprinting. Fourth, the absence of genetic profiling such as germline mutation testing or androgen receptor polymorphism analysis prevents evaluation of the potential contribution of hereditary factors to both genital development and prostate cancer aggressiveness [24]. This limits the ability to disentangle the relative influence of genetic versus hormonal determinants on the observed findings. Fifth, the cross-sectional design precludes assessment of temporal relationships. The absence of adjusted analyses represents a limitation; larger studies should incorporate multivariable models to account for confounders. Finally, the lack of follow-up precludes both confirmation of biopsy findings with surgical specimens and evaluation of long-term oncologic outcomes such as progression, recurrence, treatment response, and survival. The restrictive eligibility criteria increased internal validity by limiting confounders but may reduced representativeness and external validity.

4.3 Clinical relevance and future perspectives

Current evidence, including the present study, does not support the incorporation of genital dimensions such as stretched penile length and testicular volume into routine PCa risk assessment or diagnostic algorithms. Nonetheless, the observed association between larger penile size and greater tumor grade may reflect androgen-driven developmental imprinting, with potential implications for understanding disease biology. These findings highlight a biologically plausible, albeit indirect, link between early-life hormonal exposure and prostate cancer phenotype. Future research should prioritize large-scale, ethnically diverse, and prospectively designed cohorts, ideally with longitudinal endocrine profiling from adolescence to adulthood and pathological confirmation through surgical specimens confirmation to validate and expand upon these findings. Such approaches could clarify the temporal relationship, validate these preliminary associations, and contribute to the development of biologically informed risk stratification models.

5. Conclusions

In this cross-sectional Caucasian cohort, overall genital size was not associated with PCa presence, whereas men with longer stretched penile length more frequently harbored high-grade tumors. These preliminary findings may reflect lifelong androgen-related developmental influences. Confirmation from larger, multi-ethnic, longitudinal studies with histological verification is warranted to determine the biological basis of this relationship and its potential relevance for risk stratification in selected populations. These cross-sectional findings indicate associations only and cannot establish causation.

AVAILABILITY OF DATA AND MATERIALS

The raw database and the unprocessed statistical analysis files generated during this study are available from the corresponding author upon reasonable request, in compliance with privacy regulations.

AUTHOR CONTRIBUTIONS

CM—conceived the study, performed the statistical analysis, and oversaw overall project administration. SP—played a key role in patient recruitment. ST—contributed substantially to drafting the original manuscript. AL, AR, MO, LR, PC, GDR, FB and CQ—were involved in patient recruitment, clinical assessment, and data acquisition. LS—assisted in manuscript preparation. DA, FF and MDS—critically reviewed the manuscript. All authors have read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study was conducted in accordance with the principles of the Declaration of Helsinki and relevant national regulations. Owing to its purely observational, non-interventional design, it was deemed exempt from formal review by the Ethics Committee University of Campania Luigi Vanvitelli—A.O.R.N. dei Colli. All subjects provided written informed consent for inclusion in the study and for publication of anonymized data.

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CONFLICT OF INTEREST

The authors declare no conflict of interest. Although Celeste Manfredi is currently serving on the Editorial Board of this journal, he was not involved in the peer review process of this article and had no access to any information related to its evaluation. The entire editorial responsibility for this manuscript was delegated to Marco G. Alves.

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