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ORIGINAL PAPER

The relationship of prostate cancer diagnosed by magnetic resonance imaging/transrectal ultrasound prostate fusion biopsy with standard and adjusted anogenital distances

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Summary

Aim: *Several studies have reported contradictory associations between prostate cancer (PCa), tumor grade, and anogenital distances; however, all were based on conventional transrectal ultrasound (TRUS) prostate biopsy. We aimed to investigate the relationship between Anogenital distances (AGDs)/body mass index (BMI)-adjusted AGDs and overall PCa and clinically significant prostate cancer (csPCa) detected by multiparametric magnetic resonance imaging (MRI)/TRUS fusion prostate biopsy.*

Materials and methods: *All mp-MRI scans conducted from July 2020 to May 2024 for suspected PCa were reviewed for the study (n = 10,204). Among these, patients who underwent fusion biopsy due to Prostate Imaging-Reporting and Data System (PIRADS) 3/4/5 lesions were included in the study (n = 675). After exclusion criteria (n = 256), the remaining patients were divided into 3 groups. The study group (group-1, study group, n = 153) consisted of patients with cancer pathology, while the study subgroup (group-2, n = 80) included patients with csPCA. The control group (group-3, n = 266) comprised patients without cancer. The groups were comparatively analyzed with respect to demographic characteristics, clinical parameters, prostate-specific antigen (PSA)-related variables, mpMRI findings, biopsy characteristics, and pathological outcomes, AGDAnus to Penis (AGDAP), adjusted AGDAP, AGDAnus to scrotum (AGDAS), and adjusted AGDAS. Multivariable logistic regression analyses adjusted for age, prostate volume, PSA-related parameters, and PIRADS score were used to evaluate anogenital distance measures and identify independent predictors of csPCa.*

Results: *The study group exhibited a significantly higher age compared to the control group; nevertheless, no differences were seen between the groups for BMI and metabolic syndrome. As expected, there were significant differences between the study and control group in terms of total PSA, PSA density, DRE findings, and prostate volume. There was no significant difference between the study and control groups in terms of AGDAP, adjusted AGDAP, AGDAS, and adjusted AGDAS. When the csPCa subgroup was selected as the study group, no significant difference was observed between the control group in terms of AGDs and adjusted AGDs. Multivariable logistic regression analysis identified age and PIRADS score as independent predictors of csPCa, while prostate volume showed an inverse association. Although standard AGDAP demonstrated an independent association, other AGD parameters were not significant.*

Conclusions: *While standard AGDAP emerged as an independent predictor, BMI-adjusted AGD measures did not provide independent diagnostic value for csPCa in patients undergoing mpMRI/TRUS fusion biopsy.*

KEY WORDS: *Anogenital distances; Image-guided biopsy; Multiparametric magnetic resonance imaging; Prostate; Prostate cancer.*

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INTRODUCTION

Prostate cancer (PCa) is the second most prevalent cancer and the sixth most common cause of cancer-related mortality in males worldwide, with 1.4 million new cases and 375,000 deaths in 2020 (1). The diagnosis of PCa is typically performed with a prostate biopsy, which is recommended based on the results of *prostate-specific antigen* (PSA) testing and/or a *digital rectal examination* (DRE). In recent years, the advancement of *magnetic resonance imaging* (MRI) technologies has significantly contributed to the detection and treatment of prostate cancer. A prostate MRI provides a clearer view of possible suspicious lesions within the prostate tissue that could indicate PCa. By combining MRI with *transrectal ultrasound* (TRUS) images, a more precise method called *multiparametric MRI* (mpMRI)/TRUS fusion prostate biopsy can be used to more accurately detect prostate cancer. The multicenter, randomized PRECISION trial demonstrated that risk assessment with MRI prior to biopsy and MRI-targeted biopsy yielded considerably better results compared to the conventional TRUS prostate biopsy (38% *vs.* 26%, respectively) (2).

Anogenital distances (AGDs), which refer to the distance between the anus verge and the anterior insertion of the penis (*Anogenital DistanceAnus to Penis*; AGDAP) as well as the distance between the anus verge and the rear base of the scrotum (AGDAS), are sexually dimorphic features that can vary between males (3). Studies have demonstrated that AGDs can be determined in the prenatal period and continue to differ in maturity based on various

factors. In rat models, exposure to chemicals that inhibit androgen activity results in shorter AGDs (4). Prenatal exposure to high levels of androgens causes the development of AGDs to be prolonged. Therefore, AGDs can serve as a marker for prenatal androgenic activity (5). Additionally, research has discovered that children who have cryptorchidism or hypospadias exhibit shorter AGDs (6).

Several studies have found a substantial correlation between prostate cancer and AGDs. Elevated levels of AGDAP have been demonstrated to correlate with a reduced likelihood of developing PCa (4). A separate study found a correlation between AGD and PCa severity, and it revealed that those with longer AGDAS had higher Gleason scores and D'Amico risk class. These findings indicate that exposure to androgen hormones during pregnancy may have a significant impact on the development and severity of prostate cancer (7). All of these investigations are conducted utilizing the conventional TRUS prostate biopsy method. Conducting a study using mpMRI-TRUS fusion prostate biopsy data might enhance our understanding of this potential association.

In this study, we hypothesized that there could be a diagnostic connection between prostate cancer, prostatic lesions, and AGDs. In this context, We aimed to investigate the relationship between AGDs/BMI-adjusted AGDs and overall PCa and *clinically significant PCa* (csPCa) detected by mpMRI-TRUS fusion prostate biopsy.

Materials and methods

Ethical standards and approvals

The present study obtained approval from the *Institutional Medical Ethics Committee of Haseki Training and Research Hospita*l (date: May 23, 2024, approval no. 25-2024). Furthermore, the Hospital's Institutional Education Planning Board has granted approval (approval no. 138). Verbal and written informed consent was obtained from all patients before biopsy.

Study design

The study was conducted as a single-center analytical comparative cross-sectional study. Due to the retrospective nature of the study and the aim to improve its statistical power, all patients with PI-RADS 3/4/5 lesions detected on mp-MRI scans between 2020 and 2024 were included in the initial dataset. Our clinic has been conducting fusion biopsies since 2016. However, the data collected before 2020 was not included in the analysis since our radiology department adopted *Prostate Imaging-Reporting and Data System* (PI-RADS) version 2.1 in 2019. All data included in the present study, including AGD measurements, were obtained from the prospectively maintained "*urology department database*" of our institution.

All mpMRI scans conducted at our hospital from July 2020 to May 2024 for suspected PCa were reviewed for the study (n = 10,204). Among these, patients who underwent fusion biopsy due to PIRADS 3/4/5 lesions were included in the study (n = 675). Patients who underwent cognitive fusion biopsies, active surveillance, or had incomplete data were excluded from our study (n = 256). The remaining patients were divided into 3 groups. The study group (study group, n = 153) consisted of patients with cancer pathology, while the study subgroup (study subgroup, n = 80) included patients with csPCA. The control group (control group, n = 266) comprised patients without cancer (benign prostate hyperplasia; BPH, high grade prostatic intraepithelial neoplasia; HGPIN, atypical small acinar proliferation; ASAP, prostatitis, non-neoplasm). The groups were comparatively analyzed in terms of age, BMI, comorbidity, total PSA, PSA density, DRE, prostate volume, PIRADS score, lesion number on mp-MRI, number of cores taken in biopsy, Gleason score, ISUP grade, and cancer stages in pathology, AGDs, and adjusted AGDs.

Metabolic syndrome status was determined retrospectively from the institutional database at the time of biopsy, based on recorded comorbidities (diabetes, hypertension, dyslipidemia) and BMI.

The primary endpoint was to determine the correlation with prostate cancer, while the secondary endpoint was to assess the relationship with csPCa.

MRI scans

All MRI scans were performed using either 1.5 Tesla or 3 Tesla MRI machines. The MRI sequences that were assessed consisted of T1-weighted, T2-weighted, diffusion-weighted, and dynamic gadolinium-contrast images. A proficient radiologist (R.T.) with expertise in prostate MRI analyzed each mpMRI picture based on the specified criteria in PI-RADS v2.1 guideline recommendations (8). The radiologist measured the prostate volumes using axial and sagittal T2-weighted scans, using the formula height x breadth x depth/2.

Histology

The tissues, fixed in formalin and embedded in paraffin, were analyzed for pathological examination. The tissues were evaluated as slices with a thickness of 4 μm and stained with hematoxylin & eosin. The cases were assessed by analyzing the primary and secondary Gleason patterns and classified based on the *International Society of Urological Pathology* (ISUP) 2014 classification (9).

A Gleason score of 3+4 (ISUP grade-2) or higher was considered indicative of csPCa.

Measurements of anogenital distances

The measurements were performed utilizing a digital vernier. AGDAP was defined as the measurement from the upper edge of the anus to the anterior base of the penis, whereas AGDAS was defined as the measurement from the upper edge of the anus to the perineoscrotal junction caliper (Figure 2). Adjusted AGDs were calculated as AGD scores divided by BMI.

Biopsy procedure

Patients who had PI-RADS 3 or higher lesions underwent transrectal mp-MRI/TRUS fusion prostate biopsy (both target and systemic sampling) under local anesthesia. Each patient was given oral fosfomycin as antibiotic prophylaxis, with a dosage of 3 g given orally in two doses – one 24 hours before the biopsy and another 24 hours after. The

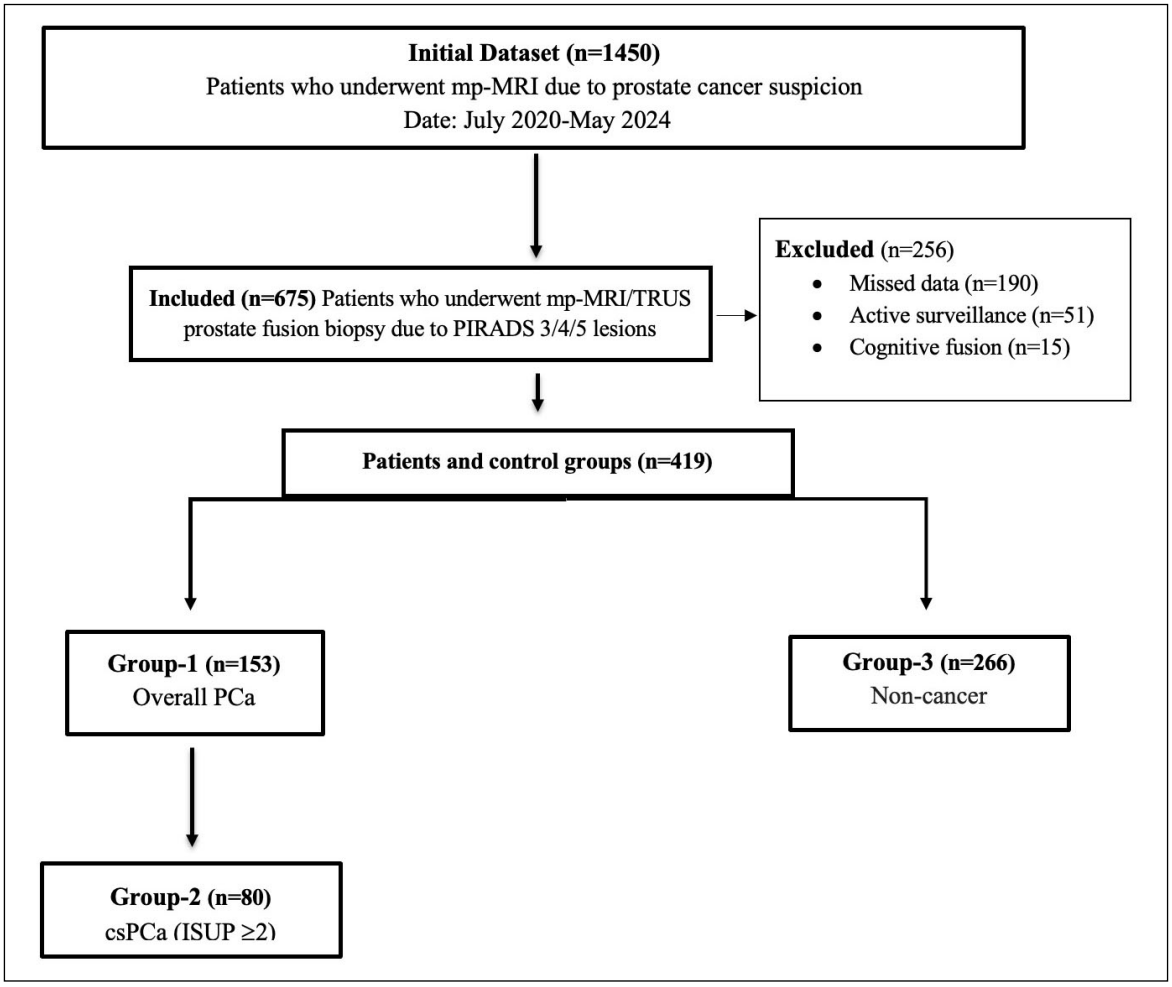
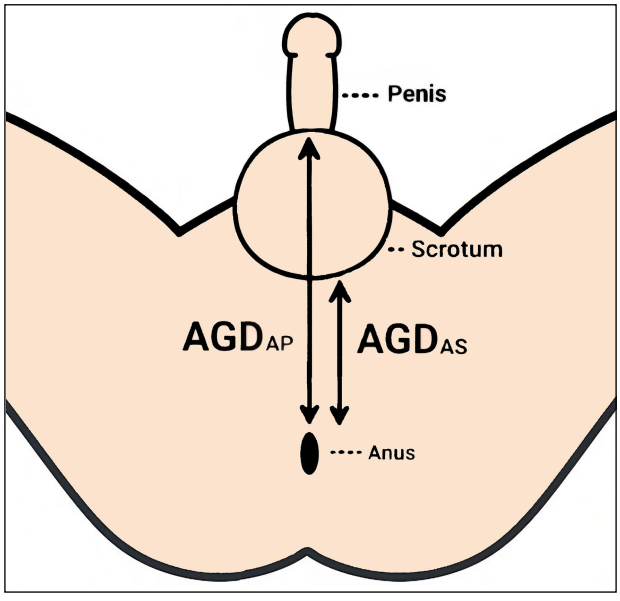


Figure 1.
Flowchart of the study.

biopsies were conducted with the Biojet rigid image-fusion platform (*D&K Technologies GmbH, Barum, Germany*) with the patient positioned in lithotomy. Analgesia was achieved with the administration of pudendal nerve blocks and periprostatic nerve blocks. A biplane probe (*BK-Medical, Herlev, Denmark*) and disposable automated tru-cut prostate biopsy gun with an 18G needle, were used for the sample. A minimum of three core samples were collected from each suspicious lesion, with at least two of them being obtained from the lesion center (Figure 3). The systematic biopsy schedule included 10-12 systematic samples from the prostate's peripheral zone. The samples were collected from the lateral and far-lateral areas of the apical, mid, and base portions of the prostate on both sides.

Figure 2.
Measurement of AGD_S.



Statistical analysis

The study's statistical analysis was conducted using IBM SPSS Statistics® version (last version) 29.0.2 software developed by IBM Corp. in Armonk, NY, USA. Numerical variables that follow a normal distribution were presented using the mean ± *standard deviation* (sd), while numerical variables having a distribution that differs from normality were presented using the median and *minimum* (min.)-*maximum* (max.) values. Categorical variables were expressed as proportions denoted by a percentage symbol (%). The normality of the quantitative variables was evaluated by various methods, including histogram visuals, coefficient of variation, skewness and kurtosis values, normal Q-Q plot and detrended normal Q-Q plot graphics, and the Kolmogorov-Smirnov test.

The Independent-Samples T test was used to compare numerical variables that demonstrated a normal distribution (age, BMI, and adjusted AGDAP) between two distinct groups that were not dependent on each other. Conversely, the Mann-Whitney U test was used to compare variables that did not display a normal distribution, such as total PSA, PSA density, prostate volume, number of significant lesions on the mp-MRI, total number of cores in biopsy, AGDAP, AGDAS, and adjusted AGDAS. The Chi-square test was to compare categorical data (comorbidity, DRE findings, and 5-ARI usage).

To identify independent predictors of csPCa, logistic regression analyses were performed. Multivariable models were adjusted for age, total PSA, prostate volume, and PIRADS score.

Anogenital distance parameters and their BMI-adjusted counterparts were entered into separate models to avoid multicollinearity.

The data were analyzed at a 95% confidence level, and any p-values that were equal to or less than .05 were considered statistically significant.

Results

A total of 419 patients were included in the study, with 153 in the study group and 266 in the control group. Within the study cohort, 80 patients (study group) were diagnosed with csPCa, while 73 patients presented with insignificant PCa. The study group exhibited a significantly higher age compared to the control group; nevertheless, no differences were seen between the groups for BMI and metabolic syndrome (Table 1).

As expected, there were significant differences between the study and control groups in terms of total PSA, PSA density, DRE findings and prostate volume (Table 2).

There was no significant difference between the study and control groups in terms of AGDAP, adjusted AGDAP, AGDAS, adjusted AGDAS (Table 3).

Variables	Study group (Overall PCa) (n = 153)	Control group (Non- cancer) (n = 266)	P value
Age (years), mean ± sd (range)	63,1 ± 5,7 (48-75)	61,1 ± 6,5 (40-77)	,002 [a]
BMI (kg/m²), mean ± sd (range)	27,6 ± 3,4 (19,5-41,2)	27,6 ± 3,7 (16,9-39,4)	,90 [a]
Comorbidity, n (%)			,08 [b]
metabolic syndrome (-)	130 (85%)	241 (90,6%)	
metabolic syndrome (+)	23 (15%)	25 (9,4%)	

PCa: prostate cancer, sd: standard deviation, BMI: Body mass index
[a]: Independents samples T test; [b]: Pearson Chi-square test.

Table 1.
Comparison of demographic characteristics and comorbidities in study and control groups.

Variables	Study group (Overall PCa) (n = 153)	Control group (Non- cancer) (n = 266)	P value
Total PSA, mean ± sd (range)	10,98 ± 22,4 (2,3-270)	7,59 ± 5,36 (0,7-39,2)	,007 [c]
PSA density, mean ± sd (range)	0,25 ± 0,47 (0,04-5,63)	0,12 ± 0,07 (0,03-0,57)	<,001 [c]
DRE findings, n (%)			<,001 [d]
positive	41 (26,8%)	16 (6%)	
negative	112 (73,2%)	250 (94%)	
5-ARI usage, n (%)			,360 [d]
(+)	11 (7,2%)	26 (9,8%)	
(-)	142 (92,8%)	20 (90,2%)	
Prostate volume, mean ± sd (range)	52,1 ± 32,5 (9-275)	64,9 ± 32,3 (12-195)	<,001 [c]
Number of significant lesions,mean ± sd (range)	1,28 ± 0,54 (1-3)	1,30 ± 0,53 (1-3)	,48 [c]
Total number of cores, mean ± sd (range)	17,2 ± 2,4 (8-24)	16,9 ±2,0 (8-22)	,07 [c]
csPCa, n (%)	96 (52,2%)	0	NA

PSA: prostate specific antigen, DRE: digital rectal examination, ARI: alpha reductase inhibitor, csPCa: clinically significant prostate cancer, NA: not available.
[c]: Mann-Whitney U Test.
[d]: Pearson Chi-square test.

Table 2.
Comparison of PSA derivates, lesion characteristics, and biopsy features between the groups.

Variables	Study group (Overall PCa) (n = 153)	Control group (Non- cancer) (n = 266)	P value
AGD_{AP}, mean ± sd (range)	108,3 ± 16,2 (76-180)	108,1 ± 15,9 (66-193)	,903 [e]
Adjusted AGD_{AP}, mean ± sd (range)	3,96 ± 0,59 (2,49-5,37)	3,96 ± 0,65 (2,59-7,46)	,888 [f]
AGD_{AS}, mean ± sd (range)	36,2 ± 14,6 (10-96)	38,0 ± 14,5 (8-97)	,222 [e]
Adjusted AGD_{AS}, mean ± sd (range)	1,32 ± 0,51 (0,42-3)	1,39 ± 0,52 (0,22-3,33)	,241 [e]

AGD_{AP}: Anogenital $Distance_{Anus\ to\ Penis}$; AGD_{AS}: Anogenital $Distance_{Anus\ to\ Scrotum}$.
[e]: Mann-Whitney U Test; [f]: Independents samples T test.

Table 3.
Comparison of AGDs and adjusted AGDs between the groups.

Variables	Study subgroup (csPCa) (n = 80)	Control group (Non- cancer) (n = 266)	P value
AGD_{AP}	109,6 ± 17,8 (78-180)	108,1 ± 15,9 (66-193)	,889 [g]
Adjusted AGD_{AP}	3,98± 0,58 (2,49-5,36)	3,96 ± 0,65 (2,59-7,46)	,887 [h]
AGD_{AS}	36,1 ± 15,9 (12-96)	38,0 ± 14,5 (8-97)	,173 [g]
Adjusted AGD_{AS}	1,30 ± 0,53 (0,42-3)	1,39 ± 0,52 (0,22-3,33)	,199 [g]

AGD_{AP}: Anogenital $Distance_{Anus\ to\ Penis}$; AGD_{AS}: Anogenital $Distance_{Anus\ to\ Scrotum}$.
[g]: Mann-Whitney U Test; [h]: Independents samples T test.

Table 4.
Comparison of AGDs and adjusted AGDs between study subgroup and control group.

When csPCa subgroup was selected as the study group, no significant difference was observed between the control group in terms of AGDs and adjusted AGDs (Table 4).
In multivariable logistic regression analysis for csPCa, age and PIRADS score were identified as robust independent predictors, although prostate volume exhibited an inverse association with csPCa. Total PSA and PSA density did not exhibit an independent association with csPCa following correction. Among the metrics of anogenital distance, only the standard AGDAP exhibited a statistically significant association; however, this was not maintained following BMI correction, and neither AGDAS nor BMI-adjusted AGD measurements were recognized as independent predictors (Table 5).

Table 5.
Multivariable logistic regression analyses for csPCa.

Variables	OR	95% CI	p-value
Age	1.12	1.06-1.18	<0.001
Total PSA	1.03	0.95-1.11	0.509
PSA density	1.80	0.04-89.02	0.767
Prostate volume	0.97	0.95-0.99	0.001
PIRADS score	4.99	3.03-8.21	<0.001
AGD_{AP}	1.03	1.01-1.05	<0.001
Adjusted AGD_{AP}	1.42	0.89-2.28	0.146
AGD_{AS}	1.01	0.99-1.03	0.340
Adjusted AGD_{AS}	1.03	0.58-1.85	0.908

PSA: prostate specific antigen; PIRADS: Prostate Imaging–Reporting and Data System;
AGD_{AP}: Anogenital $Distance_{Anus\ to\ Penis}$; AGD_{AS}: Anogenital $Distance_{Anus\ to\ Scrotum}$.

Discussion

Our investigation did not find any statistically significant association between the measurements of AGDs / adjusted AGDs and the occurrence of PCa, including both overall PCa and csPCa. Our findings add a new perspective to the existing literature on the association between AGD and PCa. In this context, the relationship between AGDS and PCa needs to be questioned.
A recent systematic review study, comprising 47 studies, investigated the association between AGDs and different reproductive disorders. The study concluded that AGDs could serve as a potentially useful non-invasive indicator for predicting the likelihood of developing PCa, depending on hormonal exposure during early life. Reviewed publications have generally demonstrated a positive correlation between elevated AGD levels and a risk of PCa. Despite this positive correlation, this systematic review emphasized that it was not possible to reach a definitive overall conclusion due to variations in study design, patient sample sizes, and demographic characteristics. In addition, all biopsies performed in the studies included in the systematic review relied on conventional systematic TRUS-guided biopsy, a technique known to be limited by sampling error and underdetection of clinically significant prostate cancer, and no study utilizing mpMRI/TRUS fusion biopsy was included (5).
A meta-analysis of 26 studies investigating the correlation between endogenous testosterone and PCa revealed no statistically significant association between PCa and levels of endogenous testosterone. Furthermore, the meta-analysis indicated that, despite the absence of trials with very long follow-up periods, testosterone replacement therapy for symptomatic hypogonadism did not result in elevated PSA levels or an increased risk of developing PCa. This meta-analysis demonstrates that although a relationship can be established between AGDs and androgen exposure at an early age, this androgen exposure may not be sufficient to cause PCa (10).
Similarly, another collaborative analysis of 18 prospective studies, including 3886 PCa and 6438 control patients, found no significant correlation between PCa and endogenous sex hormones (11). Collectively, these two meta-analyses indicate that prenatal androgen exposure alone is unlikely to be sufficient to initiate or drive prostate carcinogenesis. Taken together, these findings suggest that, in contrast to some previously published studies reporting a significant association, the results of our study may more accurately reflect the true clinical relationship between AGDs and PCa. Moreover, among the studies reporting a significant association, conflicting results have been described, with one study showing a decreased risk of PCa with increasing AGDAP (4), another reporting higher AGDAP values in the PCa group (12), and a third demonstrating an association between increased AGDAS and higher Gleason scores (7).
In recent years, the diagnostic paradigm of PCa has undergone a fundamental transformation with the integration of mpMRI, which has reshaped patient selection and risk stratification prior to biopsy. Contemporary evidence demonstrates that mpMRI-based diagnostic pathways significantly reduce unnecessary biopsies, limit the detection of indolent disease, and improve the identification of clinically significant PCa compared with systematic TRUS-guided biopsy alone (13). Within this refined diagnostic framework, biomarkers or anthropo-

metric surrogates such as AGDs are likely to provide limited incremental value once high-quality imaging and targeted biopsy techniques are incorporated into clinical decision-making. Moreover, large population-based studies and guideline-driven diagnostic models increasingly prioritize mpMRI findings, PSA density, and lesion-specific characteristics over indirect developmental markers when predicting cancer presence and aggressiveness (14). Collectively, these considerations support the interpretation that the absence of an observed association between AGDs and PCa in our study reflects the predominance of modern imaging-guided diagnostic strategies rather than a methodological limitation, underscoring the limited clinical utility of AGDs in contemporary PCa diagnostic algorithms. This interpretation is further supported by our multivariable logistic regression analysis, in which age and PIRADS score emerged as robust independent predictors of csPCa, while prostate volume showed an inverse association. In contrast, AGD parameters did not demonstrate a consistent independent association after BMI adjustment, reinforcing their limited incremental value within contemporary MRI-guided diagnostic models.

Interpreting our study, taking into account its limitations and strengths, will provide valuable guidance for comprehension. A most important limitation of the study is that it analyzed data retrospectively from a single center. However, the prospective recording of all data in our study mitigates this limitation. Another limitation is that anogenital distance measurements were obtained at a single time point. In addition, AGDs are considered surrogate markers of prenatal androgen exposure and may not fully reflect lifelong hormonal dynamics or postnatal environmental influences that contribute to prostate carcinogenesis. The use of BMI-adjusted AGDs in the present study was intended to partially mitigate these potential confounding effects of body habitus and adult anthropometric variability.

Despite these limitations, the present study represents one of the largest cohorts assessing the relationship between AGDs and PCa using mpMRI/TRUS fusion biopsy, which provides superior diagnostic accuracy compared to conventional systematic biopsy. The inclusion of csPCa as a separate outcome further strengthens the clinical relevance of our findings.

Conclusions

From a clinical perspective, our results clearly demonstrate that while standard AGDAP emerged as an independent predictor, BMI-adjusted AGD measures did not provide independent diagnostic value for identifying csPCa in patients diagnosed by mpMRI/TRUS fusion biopsy. Risk stratification and biopsy decision-making should thus persist in utilizing established clinical criteria, serum PSA derivatives, and mpMRI-derived lesion features. The routine incorporation of AGDs into contemporary prostate cancer diagnostic algorithms is not supported by our results. Future research should focus on biomarkers that have direct and reproducible clinical relevance inside MRI-guided diagnostic systems.

Declarations

Ethical approval and consent for participate: *The present study obtained approval from the Institutional Medical Ethics Committee of Haseki Training and Research Hospital (date: May 23, 2024, approval no. 25-2024). Furthermore, the Hospital's Institutional Education Planning Board has granted approval (approval no. 138).*

Consent for publication: *Verbal and written informed consent was obtained from all patients before biopsy.*

Availability of data and material: *The data that support the findings of this study are not publicly available due to institutional restrictions but are available from the corresponding author upon reasonable request.*

Competing interests: *The authors declare that they have no conflicts of interest related to this study.*

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Authors' contributions: *F.S.E. and A.E. made substantial contributions to the conception and design of the study; F.S.E. S.S and C.D. were responsible for data acquisition, including clinical data collection and anogenital distance measurements; R.T. contributed to the acquisition and interpretation of mpMRI data; F.S.E. A.E and S.S. performed the statistical analysis, interpreted the results and drafted the manuscript; A.E and R.T. critically revised the manuscript for important intellectual content; All authors contributed to the final approval of the manuscript and agree to be accountable for all aspects of the work.*

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