ORIGINAL ARTICLE

Association between Androgenic Hair Patterns and Prostate Cancer Risk in South Indian Men: A Case-control Study from the Cauvery Delta

Jayaprakash Narayanan[1], V Ezhil Sundar[2], Sri Akshay Mathi[3*]

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Abstract

Background: Prostate cancer (PCa) is one of the most common malignancies in men worldwide. Androgens influence both prostate growth and hair patterns. Androgenic alopecia (male-pattern baldness) and excessive male-pattern body hair (hypertrichosis) have been hypothesized as clinical markers of long-term androgen exposure. Previous Western studies have reported mixed results on whether early-onset or severe androgenic alopecia correlates with increased prostate cancer risk. Data in South Indian (Dravidian) populations is lacking.

Objective: To examine the association between androgenic hypertrichosis, androgenic alopecia, and prostate cancer in Dravidian men from the Cauvery Delta region of Tamil Nadu, India.

Materials and methods: We conducted an age-stratified, population-based case-control study among men in the Cauvery Delta. The cases consisted of 117 men with pathologically confirmed adenocarcinoma of the prostate (diagnosed 2010–2015). Controls were 123 men with benign prostatic hyperplasia (BPH) from the same hospital registries, frequency-matched by age. Individuals with incomplete data or non-Dravidian (North Indian) ancestry were excluded. Trained investigators performed face-to-face interviews, directly observing and recording postpubertal body hair growth (indicative of androgenic hypertrichosis) and scalp hair loss (androgenic alopecia classified by the Norwood scale). Statistical analysis included multivariate discriminant analysis (Wilks' Lambda), one-way ANOVA for continuous variables, chi-square cross-tabulation, and computation of Cramer's V statistic to assess association strength. A two-tailed *p*-value of <0.05 was considered statistically significant.

Results: The age distributions of cases and controls were comparable. The prevalence of androgenic hypertrichosis and alopecia did not differ significantly between prostate cancer cases and BPH controls. Cramer's V analysis showed that prostate cancer status accounted for only 1.1% of the variance in hypertrichosis (Cramer's V ≈ 0.011) and 1.5% of the variance in alopecia (Cramer's V ≈ 0.015).

Conclusion: In this case-control study of Dravidian men from Tamil Nadu, we observed no significant association between androgenic alopecia or hypertrichosis and prostate cancer. These findings contrast with data from Western cohorts, suggesting that interethnic variation in androgen receptor polymorphisms, follicular sensitivity, and environmental exposures may modulate prostate cancer risk differently. Further research is needed to elucidate how androgenic traits influence prostate carcinogenesis across different ethnic groups.

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Introduction

Prostate cancer (PCa) is one of the most frequently diagnosed cancers in men worldwide. Based on the GLOBOCAN 2022 estimates, PCa ranks as the fourth most common cancer overall and the second most common among men, with around 1.47 million new cases and nearly 397,000 deaths globally.[1] More recent analyses report that age-standardized incidence rates (ASIR) vary over 13-fold between regions, ranging from under 10 per 100,000 in South-Central Asia to over 100 per 100,000 in parts of Northern America and Northern Europe.[2] The median age at diagnosis is approximately 67 years, with over 60% of cases occurring in men aged 65 or older. Established risk factors for PCa include advancing age, family history, race/ethnicity, and certain genetic variations.[3] Notably, men of African ancestry have a higher incidence and mortality than those of European descent.

In India, the incidence of prostate cancer is steadily rising, particularly in metropolitan regions. Prostate cancer ranks second among male cancers in India, with an ASIR of 5.5 per 100,000 men—substantially lower than rates in Western countries (e.g., >100 per 100,000 in the United States). However, this likely reflects underreporting, low screening rates, and limited awareness, rather than the true absence of disease.[4]

Among Indian states, South India—particularly urban regions such as Chennai, Bengaluru, and Thiruvananthapuram—has consistently reported higher prostate cancer incidence. Data from the Population-based Cancer Registries (PBCRs) under the Indian Council of Medical Research - National Centre for Disease Informatics and Research (ICMR-NCDIR) showed that the Chennai PBCR reported an ASIR of 10.8 per 100,000, which is nearly double the national average.[5] Similar trends were observed in the Bengaluru and Thiruvananthapuram registries. This regional variation may reflect greater diagnostic access, lifestyle differences, and possibly unique genetic profiles in the Dravidian population.

Notably, Dravidian men in South India may harbor distinct androgen receptor (AR) polymorphisms, such as shorter CAG repeat lengths, which have been associated with both increased prostate cancer risk and variable androgen sensitivity.[6] This underscores the importance of region-specific research into androgenic traits and their potential role in prostate carcinogenesis, as findings from Western populations may not directly apply to Indian cohorts.

The Cauvery Delta lies in the southeastern part of the Indian peninsula, primarily encompassing the districts of Thanjavur, Tiruvarur, Nagapattinam, and parts of Trichy and Ariyalur in the state of Tamil Nadu. It is formed by the branching of the Cauvery River, which originates in Karnataka and drains into the Bay of Bengal. This fertile region, often referred

[1]Associate Professor, Department of Urology, KAP Viswanatham Government Medical College, Tiruchirappalli; [2]Associate Professor, Department of Urology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai; [3]Senior Resident, Department of Urology, Government Mohan Kumaramangalam Medical College Hospital, Salem, Tamil Nadu, India; *Corresponding Author

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to as the "Rice Bowl of Tamil Nadu," is predominantly agrarian and sustains dense rural populations. Genetically, the population is relatively homogeneous compared to cosmopolitan urban centers, making it a suitable cohort for phenotype–disease association studies.

Androgenic hair changes—namely male-pattern baldness—are driven by dihydrotestosterone (DHT) via the 5α-reductase pathway. In balding scalp follicles, increased local 5α-reductase activity converts testosterone to DHT, which binds androgen receptors and leads to follicular miniaturization and hair thinning.[7] DHT exerts androgenic effects on nonscalp areas, stimulating the development and persistence of terminal hair in androgen-sensitive regions such as the beard, axillae, chest, and limbs.[8]

Androgenetic alopecia (AGA) is extremely common–affecting ~50% of Caucasian men by age 50 and ~80% by age 70. Prevalence is somewhat lower in Asian populations, but still substantial. Given that both prostate cancer and AGA are androgen-dependent, several Western studies have investigated their link. There is limited evidence from India linking androgenic hair patterns to prostate cancer. Given ethnic differences in AR genetics and hair biology, results from Caucasian/Western cohorts may not generalize. It is plausible that Dravidian men manifest androgen effects differently (for example, a genetic propensity to body hair growth) that could uncouple hair phenotypes from cancer risk. To our knowledge, no previous study has assessed both scalp and body hair patterns in relation to prostate cancer in the Tamil population. We therefore undertook this age-stratified, population-based case-control study to evaluate whether androgenic alopecia or hypertrichosis is associated with histologically confirmed prostate cancer among men from the Cauvery Delta region.

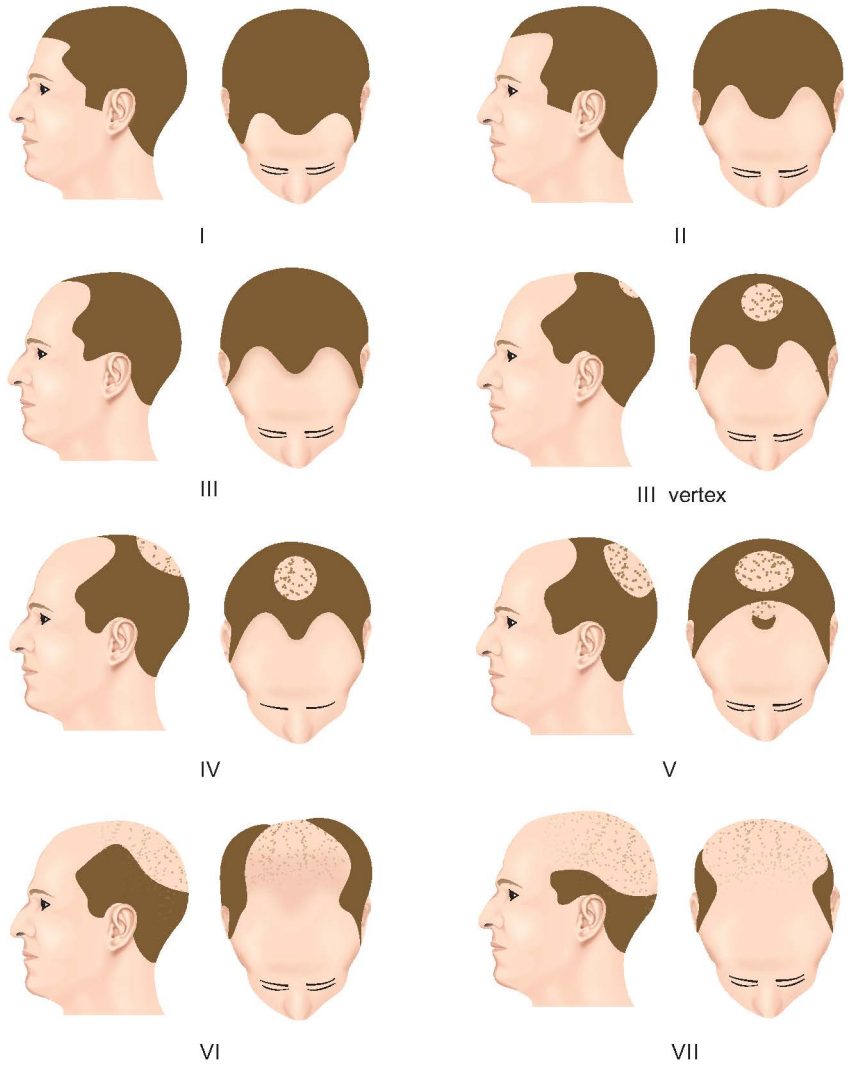

Fig. 1: Hamilton–Norwood classification

Materials and Methods

Study Design and Participants

This was an age-stratified, population-based case-control study conducted in the Cauvery Delta region of Tamil Nadu, India. The source population consisted of adult men of Dravidian (Tamil) ethnicity who sought urological care between 2010 and 2015.

The study included 117 men with histopathologically confirmed adenocarcinoma of the prostate, diagnosed in the Cauvery Delta region between 2010 and 2015. The control group comprised 123 men presenting with lower urinary tract symptoms and diagnosed with benign prostatic hyperplasia (BPH) during the same period at Thanjavur Medical College Hospital and FrontLine Hospital, Trichy. Controls were frequency-matched to cases based on age distribution to minimize confounding.

Men of non-Dravidian (North Indian or other) ethnicity and those with missing clinical or hair data were excluded. All participants provided informed consent for study procedures.

Data Collection

Trained investigators conducted structured, face-to-face interviews with each participant. Demographic information (age, education, occupation), lifestyle factors, medical history, and family history of prostate disease were recorded. The key exposures—androgenic hypertrichosis and androgenic alopecia—were assessed by direct physical examination during the interview:

- Androgenic hypertrichosis: Excessive hair growth in postpubertal males over and above the normal for the age, race of an individual, with normal prepubertal hair distribution. Examiners noted presence or absence of conspicuous body hair in typical androgen-dependent areas (e.g., central chest, shoulders, back). Any man with notably dense male-pattern body hair was classified as hypertrichotic.
- Androgenic alopecia: Assessed using the standardized Hamilton–Norwood classification (Fig. 1). Trained observers determined the Norwood grade of scalp hair loss in each participant. We categorized baldness into vertex (crown) baldness, frontal baldness, or no significant alopecia.

By direct observation (not self-report), the interviewers documented each subject's current hair pattern. All examiners were blinded to the subject's case/control status when assessing hair, to reduce observer bias.

Table 1: Comparison of hypertrichosis and androgenic alopecia scores between prostate cancer and BPH groups

Variables	*Diagnosis*	*No of patients*	*Mean*	*SD*	*SEM*
Hypertrichosis	CA prostate	117	0.06	0.238	0.022
	BPH	123	0.07	0.248	0.022
Androgenic Alopecia	CA prostate	117	0.23	0.423	0.039
	BPH	123	0.24	0.431	0.039

Table 3: Distribution of hypertrichosis among patients with prostate cancer and BPH

Diagnosis	*Hypertrichosis*		*Total*
	No	*Yes*	
CA prostate	110	7	117
BPH	115	8	123
Total	225	15	240

Table 4: Distribution of androgenic alopecia among patients with prostate cancer and BPH

Diagnosis	*Alopecia*		*Total*
	No	*Yes*	
CA prostate	90	27	117
BPH	93	30	123
Total	183	57	240

Statistical Analysis

We performed descriptive analyses comparing cases and controls. Continuous variables (e.g., age) were summarized as means ± standard deviations and compared by one-way ANOVA. Categorical variables (presence/absence of hypertrichosis or alopecia) were analyzed using chi-square tests. The strength of association between hair phenotype and prostate cancer was quantified using Cramer's V coefficient from the cross-tabulation: this gives the percentage of variance in cancer status explained by hair status. A multivariate linear discriminant analysis (Wilks' lambda) was also computed to examine the joint discriminative power of age, hypertrichosis, and alopecia for classifying case vs control. All tests were two-sided with $\alpha = 0.05$. Statistical analyses were conducted using SPSS v22 (IBM, Armonk, NY) and MedCalc v15 (Ostend, Belgium).

Results

Participant Characteristics

The final analysis included 117 prostate cancer cases and 123 BPH controls. Most subjects were aged between 60 and 75 years, reflecting the age distribution of PCa. The mean age (± SD) was almost similar in both groups (cases: 67.2 ± 7.8 years; controls: 66.5 ± 8.2 years). Educational levels and occupational backgrounds did not differ notably. There were no significant differences between cases and controls in known risk factors such as body mass index (BMI) or smoking history.

The mean BMI was 23.8 ± 3.1 kg/m^2 in the prostate cancer group and 23.6 ± 2.9 kg/m^2 in the BPH control group, consistent with regional averages. Approximately 26% of cases and 24% of controls reported current or past smoking, with no significant differences between groups ($p > 0.05$).

Hair Phenotypes

Among cases, hypertrichosis was observed in 6% (7/117), and in controls, 6% (8/123) had hypertrichosis. Baldness was observed in 23% (27/117) of prostate cancer cases and 24% (30/123) of BPH controls. Among these, vertex baldness was more prevalent—seen in 18% (21/117) of cases and 20% (25/123) of controls, while frontal-only baldness was observed in 5% (6/117) and 4% (5/123). This pattern indicates that vertex baldness was the predominant type in both groups.

The one-way ANOVA results (Table 1) indicate that there is no statistically significant difference in the mean scores of hypertrichosis and androgenic alopecia between prostate cancer and BPH groups ($p > 0.05$). This suggests that neither trait effectively differentiates the two diagnostic groups.

Therefore, hypertrichosis and androgenic alopecia are not associated with prostate cancer status in this study population. Prevalence of both traits was similar in cases and controls.

From multidiscriminant analysis (MDA) (Table 2), Wilks' lambda of 1.00 and a nonsignificant chi-square 0.07 ($p > 0.05$) indicate that hypertrichosis and androgenic alopecia do not significantly discriminate between prostate cancer and BPH groups.

Table 2: Results of multidiscriminant analysis

Variables	*Coefficient*
Hypertrichosis (X_1)	2.132
Androgenic alopecia (X_2)	1.886
Constant	−4.599

The results of cross-tabulation between hypertrichosis and diagnosis (carcinoma prostate vs BPH) showed a Cramer's V value of 0.011, indicating a very weak association (Table 3), accounting for only 1.1% of the variance. This confirms that the presence or absence of hypertrichosis is statistically independent of diagnosis at the 5% significance level.

Similarly, cross-tabulation between androgenic alopecia and diagnosis yielded a Cramer's V value of 0.015, indicating a very weak association (Table 4), explaining only 1.5% of the variance. This also confirms that the presence or absence of androgenic alopecia is statistically independent of diagnosis at the 5% significance level.

Discussion

In this first study of its kind in the Cauvery Delta, we found no evidence that androgenic hair phenotypes predict prostate cancer in Tamil men. Both scalp baldness and male-pattern body hair growth were distributed equally among prostate cancer cases and BPH controls. Negligible correlation was observed between hair phenotypes and prostate cancer status.

Consequently, neither androgenic alopecia nor hypertrichosis emerged as risk indicators in this population. These findings contrast with some previous reports in Western cohorts. For example, an Australian age-stratified case-control study[9] found that vertex baldness was significantly associated with higher prostate cancer risk (age-adjusted OR ~1.5 for any vertex balding vs none).

A systematic review[10] of seven case-control studies likewise found that vertex AGA conferred a modestly increased risk (pooled OR ~1.25, $p = 0.002$). No statistically significant association between AGA (any pattern) and prostate cancer was identified (OR 1.03; $p = 0.58$).

A French cohort study[11] reported that men with early-onset baldness (by age 20) had doubled odds of prostate cancer.

These studies suggest shared androgenic pathways between scalp hair follicle miniaturization and prostate tumorigenesis. Proposed mechanisms include chronically elevated DHT levels or heightened 5α-reductase activity, which can both shrink

scalp follicles and promote prostatic epithelial proliferation.

Some studies have reported no positive association between androgenic alopecia and prostate cancer. A Dutch population-based analysis[12] found that early-onset baldness at age 20 or 40 was linked to a slightly reduced risk, and a combined frontal and vertex pattern at age 40 showed a significantly lower risk (*OR* = 0.62). No associations were found with aggressive cancer. These findings align with our study, reinforcing that baldness is not a reliable marker for prostate cancer risk.

A Turkish prospective study[13] reported no significant difference between androgenic alopecia and serum androgen levels in BPH and prostate cancer patients.

Our results are consistent with these null findings, suggesting that hair pattern may be an unreliable surrogate for lifetime androgen exposure or that its association with prostate cancer risk may be influenced by ethnic and genetic factors.

Previous studies suggest that South Indian men tend to carry shorter CAG repeat lengths in the androgen receptor gene, a polymorphism strongly linked to higher prostate cancer risk.

Shorter CAG repeats lead to more sensitive ARs, meaning that prostatic tissue may be hyper-responsive to normal androgen levels.

Paradoxically, this might manifest as increased cancer risk without necessarily causing markedly increased scalp hair loss–especially if other genetic factors (or hair follicle receptor levels) differ. In other words, a man may have high prostatic AR activity but comparatively preserved scalp hair, depending on AR coregulators or local 5α-reductase activity in the skin. Additionally, body hair growth (hypertrichosis) itself reflects androgenic end-organ sensitivity. Increased androgen conversion to DHT by 5α-reductase results in thicker beard and chest hair, often accompanied by scalp hair loss.[14]

A genetic predisposition toward dense body hair in Dravidian men may not necessarily translate into increased prostate cancer risk with prostate cancer risk. Indeed, we found that hypertrichosis itself showed no predictive value. Moreover, androgenic alopecia is highly age-dependent and genetically complex. In Caucasian men, by age 50, about half have noticeable baldness; in Asian men, the prevalence is somewhat lower but still substantial.[15]

Such baseline differences mean that a given degree of alopecia may represent different relative androgen histories across populations.

It is also possible that differences in diet, lifestyle, or environmental exposures in rural Tamil Nadu modulate prostate cancer risk in ways unrelated to hair phenotype. An alternative explanation is methodological: our controls were men with symptomatic BPH, not general population controls. BPH and prostate cancer share some pathophysiology, but their relationship to androgens is complex. If the controls had elevated intraprostatic DHT similar to cases (as might occur in BPH), this could diminish any apparent difference in hair patterns between groups. Indeed, Faydaci et al.[13] found no significant differences in baldness frequency or serum testosterone between BPH and prostate cancer groups, as we did.

Limitations of our study include the moderate sample size and potential for selection bias. Although we used direct clinical observation of hair (a strength over self-report), we did not quantify hormone levels or scalp DHT directly. Also, we did not stratify by tumor grade; some reports suggest AGA may link more strongly to aggressive or early-onset prostate cancer,[9] which we could not evaluate.

Finally, this study was conducted in a geographically confined, ethnolinguistically uniform Dravidian Tamil population from the Cauvery Delta region of South India, allowing for reduced genetic and environmental variability. Results may not apply to other Indian ethnic groups. Despite these limitations, our findings have implications for risk assessment. In the Cauvery Delta, neither male pattern baldness nor excessive body hair should be regarded as independent risk indicators for prostate cancer. This contrasts with some Western data, emphasizing that ethnic context matters. It further suggests that underlying androgen-related cancer mechanisms might differ by population. Future studies should explore genetic markers (e.g., AR polymorphisms, 5α-reductase expression) and include diverse cohorts to understand these differences.

Conclusion

In this case-control study of Dravidian men from Tamil Nadu, we observed no significant association between androgenic alopecia or hypertrichosis and prostate cancer. These findings contrast with data from Western cohorts, suggesting that interethnic variation in androgen receptor polymorphisms, follicular sensitivity, and environmental exposures may modulate prostate cancer risk differently. Further research is needed to elucidate how androgenic traits influence prostate carcinogenesis across different ethnic groups.

Orcid

Sri Akshay Mathi https://orcid.org/0000-0003-2737-3192

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