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The Association of Male Pattern Baldness in Men with Benign Prostatic Hyperplasia

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Abstract

Aim: To evaluate the association between male pattern baldness (MPB) and benign prostatic hyperplasia (BPH).

Methodology: Cohort study, data collected by co-authors. All extracted data were input to Excel sheets and further imported into STATA version 14. The study's results were reported in the form of descriptive statistics.

Results: This study found no significant associations between MPB and BPH. Nonetheless, the study demonstrated that patients with increased baldness severity have higher rates of diabetes mellitus (DM) and obesity.

Conclusion: There is no association between MPB and BPH.

Keywords: Male pattern baldness, benign prostate hyperplasia, modified Norwood Hamilton scale

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Introduction

Benign prostatic hyperplasia (BPH) is a pressing public health pathology as it affects about 50% of all men in the later decades of their lives [1]. Precise prevalence estimates of BPH vary depending on the definition used and the characteristics of the studied population; nonetheless, a landmark study by Berry et al. demonstrated that men have an 8% prevalence rate of BPH in their fourth decade, which increases up to 50% by the fifth and sixth decades [2]. Additionally, while the growth of BPH varies on an individual basis, it is estimated that BPH growth time is 4.5 years in the third to fifth decade and 10 years for later decades [2].

Androgenic alopecia is a physiologic process affecting 30% of men from the age of 30 and 50% of men after the age of 50 [3]. Both BPH and androgenic alopecia are androgen-dependent conditions, in which testosterone is converted to androgen dihydrotestosterone (DHT) by the 5-alpha reductase enzymes [4]. Hair follicle type II 5-alpha reductase enzyme and prostate-specific types I and II 5-alpha reductase enzymes are all involved in the pathogenesis of both androgenic alopecia and BPH [5]. DHT binds to the androgen receptors on hair follicles, transforming them into vellus hairs and shortening their active growth

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phase [3]. DHT, in association with other hormones, is assumed to regulate the balance between cell synthesis and apoptosis in the prostate of adults.

In this study, we evaluate the association between male pattern baldness, BPH, prostate-specific antigen (PSA) levels, prostate size, and duration of treatment.

Methodology

We performed a cross-sectional evaluation of male patients over the age of 40 visiting the urology clinics at Jordan University Hospital from January 2020 to January 2021. We collected data and patients were conveniently sampled if they satisfied the aforementioned inclusion criteria and consented to participate.

Included participants were assessed for the following: age (in years), body mass index (BMI), smoking history, medical comorbid diseases (i.e., diabetes mellitus, hypertension), family history of BPH, and BPH-related variables, including time to diagnosis, severity of erectile dysfunction, and medications used. Participants' laboratory variables—PSA and HbA1c—were also extracted. In addition, participants were examined for their prostate size and their baldness using a modified Norwood Hamilton scale (Figure A). They were also required to recall their baldness status in different decades of their lives.

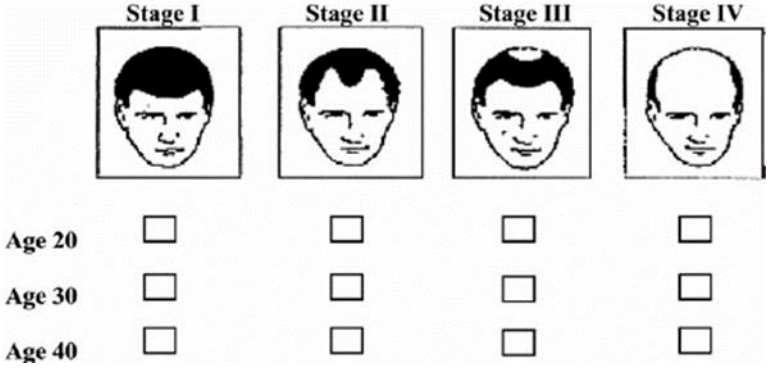

Figure A

Data Analysis Plan

All extracted data were input into Excel sheets and further imported into STATA version 14. The study's results are reported as descriptive statistics. Categorical variables are summarized as frequencies (n, %), while continuous data were reported as mean, median (when applicable) and standard deviations. Categorical associations were evaluated using a Chi-square test, while associations involving continuous data are assessed using Student's *t*-test and ANOVA. However, where these data were not normally distributed, non-parametric tests, such as Kruskal-Wallis, were utilized to assess the study's hypotheses and detect significant differences. Furthermore, logistic regression was utilized to assess clinical predictors with regards to presence of more baldness in patients with BPH. An alpha value of ≤ 0.05 (CI=95%) is considered statistically significant.

Ethical Considerations

Observational studies display minor ethical concerns due to the non-invasive nature inherently embedded within their design. Irrespective of its type, participant data were treated with the utmost confidentiality. No

institute member outside the affiliated research team was allowed to view, manipulate or transfer data. Any and all data contributing to the identification of participants' identities, such as names, phone numbers or addresses, were either turned into codes or omitted from the later stages of data analysis. Finally, the University of Jordan's ethics committee granted IRB approval after evaluating the study proposal.

Results

The study recruited 141 adults above the age of 40 to study the association between male pattern baldness and BPH. Of the included cohort, 34.8% fell into the 60–69 age group, while 29.1% were in the 70–79 age group. In terms of BMI, 44.0% of the cohort were overweight, while 25.5% were obese. The majority of participants (41.1%) were non-smokers, while 34.1% were ex-smokers. Comorbidities including hypertension and diabetes mellitus were found in 51.1% and 39.7% of the studied population, respectively. In addition, 36.2% of participants had a family history of BPH. Table 1 presents patient demographics and medical history.

Table 1: Differences in baldness severity in 141 BPH patients based on sociodemographic variables and medical history

Variable	Category	Total	Score 1 (%)	Score 2 (%)	Score 3 (%)	Score 4 (%)	*p* value
Age (years)		141 (100)	22 (15.6)	32 (22.7)	25 (24.8)	52 (36.9)	0.08
	40–49	14 (9.9)	2 (14.3)	2 (14.3)	8 (57.1)	2 (14.3)	
	50–59	21(14.9)	3 (14.3)	6 (28.6)	1 (4.7)	11 (52.4)	
	60–69	49 (34.8)	12 (24.5)	10 (20.4)	11 (22.4)	16 (32.7)	
	70–79	41 (29.1)	3 (7.3)	9 (22)	11 (26.8)	18 (43.9)	
	80–90	16 (11.3)	2 (12.5)	5 (31.25)	4 (25)	5 (31.25)	
BMI							0.66
	Underweight	0 (0)	-	-	-	-	
	Normal	43 (30.5)	9 (20.9)	11 (25.6)	10 (23.3)	13 (30.2)	
	Overweight	62 (44)	6 (9.7)	15 (24.2)	16 (25.8)	25 (40.3)	
	Obese	36 (25.5)	7 (19.4)	6 (16.7)	9 (25)	14 (38.9)	
Smoking history							0.4
	None	58 (41.1)	6 (10.4)	13 (22.4)	14 (24.1)	25 (43.1)	
	Current smoker	35 (24.8)	7 (20)	7 (20)	12 (34.3)	9 (25.7)	
	Ex-smoker	48 (34.1)	9 (18.75)	12 (25)	9 (18.75)	18 (37.5)	
Hypertension		72 (51.1)	11 (15.3)	17 (23.6)	18 (25)	26 (36.1)	1
Diabetes		56 (39.7)	8 (14.3)	12 (21.4)	13 (23.2)	23 (41.1)	0.9
Family history of BPH		51 (36.2)	8 (15.7)	10 (19.6)	10 (19.6)	23 (45.1)	0.4
Time since BPH diagnosis (years)							0.4
	<1	26 (18.5)	5 (19.2)	4 (15.4)	4 (15.4)	13 (50)	
	1–5	58 (41.1)	6 (10.3)	13 (22.4)	17 (29.4)	22 (37.9)	
	6–10	24 (17)	7 (29.2)	5 (20.8)	6 (25)	6 (25)	
	>10	33 (23.4)	4 (12.1)	10 (30.3)	8 (24.3)	11 (33.3)	
Erectile dysfunction severity							0.8
	None	33 (23.4)	6 (18.2)	8 (24.2)	5 (15.2)	14 (42.4)	
	Rare	10 (7.1)	2 (20)	4 (40)	2 (20)	2 (20)	

Variable	Category	Total	Score 1 (%)	Score 2 (%)	Score 3 (%)	Score 4 (%)	*p* value
	Sometimes	29 (20.6)	3 (10.3)	7 (24.1)	9 (31.1)	10 (34.5)	
	Most of the time	69 (48.9)	11 (15.9)	13 (18.9)	19 (27.5)	26 (37.7)	
Used medications							
	Fenstride	50 (37)	11 (22)	11 (22)	12 (24)	16 (32)	0.6
	Tamsulosine	62 (44)	13 (21)	14 (22.6)	15 (24.2)	20 (32.2)	0.5
	Alfuzocin	63 (44.7)	7 (11.1)	16 (25.4)	16 (25.4)	24 (38.1)	0.6

Most of the participants (41.1%) had 1–5 years' time to BPH diagnosis. Moreover, almost half of the sample complained of erectile dysfunction. In terms of BPH treatment, participants commonly used alfuzocin 10 mg once daily (44.7%), tamsulsoine 0.4 mg once daily (44.0%), and finasteride 5 mg once daily (37.0%). Mean duration of finasteride usage in the sample was 2.6 ± 1.8 years. Table 2 illustrates participant laboratory variables, including PSA and HbA1c.

Table 2: Differences in baldness severity in 141 BPH patients based on laboratory findings, duration of treatment and prostate size

Variable	Total (mean±SD)	Score 1 (mean±SD)	Score 2 (mean±SD)	Score 3 (mean±SD)	Score 4 (mean±SD)	*p* value
PSA-free	0.8±1	1±1.6	0.9±1.1	0.6±0.5	0.7±0.8	0.15
PSA-total	2.9±4	4.3±5.7	3.4±5	1.8±1.7	2.8±3.5	0.1
HbA1c	6.2±0.9	6.2±0.9	6.2±1	6.1±0.8	6.2±1	0.7
Prostate size	62.1±31.2	65±32	73±39.9	55.6±25.6	58.4±27.1	0.9
Duration of fenstride use (years)	2.6±1.8	2.6±2	2.8±1.9	2.8±1.6	2.4±1.8	0.07

Univariate analysis showed that no single variable was significantly different across baldness scores. On the other hand, multivariate analysis demonstrated that age, BMI, diabetes, and HbA1c levels are significantly associated with more baldness in patients with BPH (refer to Table 3).

Table 3: Multivariable ordinal logistic regression for factors associated with more baldness in BPH

Variable	Category	B coefficient	*p* value	95% confidence interval
Age		0.08	**0.05**	0.03–0.15
BMI				
	Normal (reference)			
	Overweight	2.4	**<0.01**	0.7–4
	Obese	1.9	**0.02**	0.4–3.4
DM		2.9	**<0.01**	1.0–4.7
HBA1C		-1.7	**<0.01**	-2.8– -0.6
Finastride use duration (years)		0.001	**0.95**	-0.3–034

Discussion

In males with genetic susceptibility, DHT, as converted from testosterone through the 5-alpha reductase enzyme, causes hair thinning along the frontal and temporal areas of the scalp [3]. Once these hair cells atrophy, significant hair loss ensues. Similarly, BPH is the result of cellular hyperplasia influenced by high degrees of DHT, the ultimate result of which is urinary symptoms. Therefore, both disease entities share the same molecular pathophysiologic pathway through the 5-alpha reductase activity. This study aimed to investigate the relationship between androgen-influenced baldness and BPH in men referred to Jordan University Hospital.

The literature on this particular association contains significant discrepancy due to its scarcity. Oh et al. [6] investigated a Korean cohort of 225 patients with BPH and 160 controls, and found that patients with BPH had a significantly higher frequency of severe male pattern baldness (grade IV or higher based on the Norwood's classification). Similarly, Arias-Santiago et al. [5] showed that androgenic alopecia is an independent predictor of prostate volume larger than 30mL, urinary flow less than 15 mL/s, and BPH. Such results indicate that middle aged men with androgenic alopecia should be screened for urinary symptoms and examined for PSA levels and IPSS score.

In consensus with our findings, Faydaci et al. [7] found no significant differences in the frequency of androgenic alopecia between those with BPH and patients with prostate cancer. Androgenic alopecia was not associated with any urinary symptoms. Moreover, the study demonstrated that androgenic alopecia is not significantly correlated to androgenic hormones such as free testosterone, total testosterone, luteinizing hormone, or follicle stimulating hormone. Chen et al. [8] demonstrated that androgenic alopecia was not significantly different in those with BPH and their controls. However, the study found a significant association between larger prostate size and higher prevalence of androgenic alopecia.

Our results demonstrate no significant correlation between MPB and BPH. Despite both diseases affecting a significant proportion of men in a chronobiological fashion, other molecules exist which are key to regulating the proliferation and turnover of prostate and hair cells, such as insulin-like growth factors [8]. Moreover, age could be a confounding factor preventing a proper interpretation of androgenic alopecia within the context of BPH.

Multivariate analysis showed that higher BMI and diabetes are significant predictors of more baldness in patients with BPH. The literature demonstrates that androgenic alopecia and BPH are associated with higher prevalence of cardiovascular risk factors such as abdominal obesity, insulin levels, diabetes, hypertension and systemic inflammation [5]. The exact pathway facilitating the development of BPH through any of the aforementioned risk factors is yet to be determined; however, it is believed that obesity causes BPH through inducing inflammation and oxidative stress [9]. On the other hand, high insulin levels due to diabetes may induce BPH since insulin shares similar structure to insulin-like growth factors, which promotes prostate enlargement [10].

Our study is subject to several limitations, including its cross-sectional nature and associated biases which could hinder the generalizability of the results. Moreover, the study's sample size is small, and this could have influenced the statistical power of the study and therefore impacted its associations. In addition, there was no control group.

Conclusion

In summary, this small cross-sectional study found no significant associations between MPB and BPH. Nonetheless, the study demonstrated that patients with increased baldness severity have higher rates of DM and obesity. Large-scale studies are needed to conform such associations in which the controls and prostate cancer patients must be included as references.

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الارتباط بين الصلع الذكوري النمطي والرجال المصابين بتضخم البروستاتا الحميد

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الملخص

الأهداف: تقييم العلاقة بين الصلع الذكوري النمطي وتضخم البروستاتا الحميد.

منهجية البحث: الدراسة جماعية، وقد تم جمع البيانات من قبل المؤلفين المشاركين. تم إدخال جميع البيانات المستخرجة في أوراق Excel ثم تم معالجتها باستخدام برنامج STATA 14. تم تصنيف نتائج الدراسة في شكل إحصائيات وصفية.

النتائج: لم تجد هذه الدراسة ارتباطات ذات دلالة إحصائية بين الصلع الذكوري النمطي وتضخم البروستاتا الحميد ومع ذلك، أظهرت الدراسة أن المرضى الذين يعانون من زيادة حدة الصلع لديهم معدلات أعلى من حيث الاصابة بمرض السكري والسمنة.

الاستنتاج: لا يوجد ارتباط بين الصلع الذكوري النمطي وتضخم البروستاتا الحميد.

الكلمات الدالة: الصلع الذكوري النمطي، تضخم البروستاتا الحميد، مقياس نوروود هاملتون المعدل.