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Benign prostatic hyperplasia, metabolic syndrome and androgenic alopecia: Is there a possible relationship?

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ABBREVIATIONS

AGA, androgenetic alopecia;
BMI, body mass index;
CRP, C-reactive protein;
CVD, cardiovascular disease;

Abstract ***Objective:*** To evaluate the incidence of benign prostatic hyperplasia (BPH) and metabolic syndrome in patients with androgenetic alopecia (AGA) in comparison with those with no AGA, as several previous studies have reported inconsistent results of an association between metabolic syndrome and BPH with AGA.

Patients, subjects and methods: This cross-sectional study included 400 participants, divided into 300 patients diagnosed with AGA, with different grades according to Norwood–Hamilton classification, and 100 control subjects with no AGA. Criteria for diagnosis of metabolic syndrome according to Adult Treatment Panel-III criteria (waist circumference, blood pressure, fasting blood sugar, high-density lipoprotein and triglycerides), as well as criteria for diagnosis of BPH (prostatic volume, urine flow, and prostate-specific antigen) were assessed in all patients and compared with the control subjects.

Results: There were significant differences between the AGA and no-AGA groups for the following variables: waist circumference, body mass index, fibrinogen level, fasting blood sugar, cholesterol, C-reactive protein, erythrocyte sedimentation rate, and glycosylated haemoglobin. There was a significant difference in number of

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DHT, dihydrotestosterone;
ESR, erythrocyte sedimentation rate;
FBS, fasting blood sugar;
HbA1c, glycosylated haemoglobin;
HDL, high-density lipoprotein

patients with AGA manifesting criteria of metabolic syndrome (51% vs 28%), as well as BPH diagnostic criteria (36% vs 6.8%) compared with the control subjects. Both BPH and metabolic syndrome were shown to be significant independent variables associated with AGA.

Conclusions: Dermatologists, urologists, and primary care physicians should monitor patients with early onset AGA for the development of urinary symptoms, to permit an earlier diagnosis of BPH; and for metabolic syndrome symptoms, to permit early diagnosis of cardiovascular risk factors.

Introduction

Androgenetic alopecia (AGA) is the most common form of hair loss in humans, affecting almost 50% of men throughout their lifetime. A link between male pattern baldness and androgens has previously been documented. Clinical observations and histopathological findings have shown that AGA is a process dependent on dihydrotestosterone (DHT) with continuous miniaturisation of sensitive hair follicles. As most male patients with AGA have normal circulating androgen levels, a local imbalance of DHT and androgen hypersensitivity in balding vs non-balding scalps are considered to be the main pathogenic factors [1].

BPH is the most common benign neoplasm in men, affecting $>70\%$ of men aged $\geqslant 70$ years with or without obstructive symptoms [2]. Epidemiological study has shown that elevated free PSA levels, heart disease, and the use of β-blocker medications increase the risk of BPH [3]. The prostate gland depends on stimulation of androgens, in particular DHT, for its development and growth, and BPH predominantly involves the stromal compartment of the gland. DHT is converted from testosterone by 5α-reductase in prostatic stromal and basal cells, with type 2 5α-reductase being the predominant isozyme. The role of androgens in the pathogenesis of human BPH is debated, but they undoubtedly play at least a permissive role. Although not elevated in BPH, DHT levels in the prostate remain at a normal level with ageing, despite a decrease in plasma testosterone [4].

AGA and BPH are both androgen-dependent disorders, displaying *in situ* high levels of DHT with a good therapeutic response to finasteride. The growth and development of hair follicles and the prostate gland are both dependent on the interaction between mesoderm (hair dermal papilla vs prostatic stroma) and ectoderm (outer root sheath keratinocytes vs prostatic epithelium) [5]. On the basis of similar androgen-responding tissue growth and disease pathogenesis, as well as therapeutic experience, speculation has arisen as to the association between AGA and BPH. Several studies have shown a relationship between both conditions, such as those of Arias-Santiago et al. [6] and Kaplan [7], while Dastgheib et al. [8] found there was no relationship between AGA, PSA level, IPSS, and prostate volume.

Previous studies have shown a positive association between male pattern baldness and insulin resistance, metabolic syndrome, and hypertension [9,10]. It has also been postulated that baldness is linked to cardiovascular disease (CVD) by mechanisms, such as hyperinsulinaemia, chronic inflammation, and increased peripheral sensitivity to androgens.

The main objective of the present study was to evaluate the incidence of BPH and metabolic syndrome in patients with AGA in comparison with control subjects with no AGA.

Patients, subjects and methods

This study was a cross-sectional study including 400 participants, who were divided into 300 patients diagnosed with AGA (AGA group) recruited from the dermatology and urology outpatient clinic of the main University Hospital and Faculty of Medicine, University of Alexandria (between May 2012 and May 2015) and 100 control subjects (no AGA group), who were selected from visitors of different outpatient clinics of the hospital, security personnel, and workers in the hospital who did not have AGA and were willing to do a free evaluation for their prostatic status and other blood investigations. Patterns of baldness and severity were recorded by a dermatologist as grade I–VII according to the Norwood–Hamilton classification. The AGA grades were categorised as mild (grade I–III), moderate (IV, V) or severe (VI, VII).

Exclusion criteria were: history of prostate disease; prostatitis; neurogenic bladder; previous consultation with urologist or family physician for prostate problems; and treatment with minoxidil (in previous 6 months), α-blockers, testosterone, 5α-reductase inhibitors, or any other hormone therapy. The study was approved by the Ethics Committee of Alexandria University Hospital, and a fully informed consent was signed by all participants.

The weight, height, and abdominal circumference of participants were measured, and their body mass index (BMI) was calculated. Systolic and diastolic blood pressure was measured after a 5-min rest and again 10 min later, recording the mean value.

Triglycerides, total cholesterol, high-density lipoprotein (HDL), fasting blood sugar (FBS), fibrinogen, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels were studied in samples drawn between 0800 and 0900 h after a rest period of $\geqslant$30 min.

Criteria for diagnosis of metabolic syndrome

This diagnosis was made according to the most recent consensus report of the National Cholesterol Education Programme, i.e. the Adult Treatment Panel-III criteria, by the presence of three of the following: abdominal circumference of $>$102 cm. Hypertriglyceridaemia of $>$150 mg/dL, HDL of $<$40 mg/dL, blood pressure of $>$130/85 mmHg, or glycaemia $>$110 mg/dL [9,10].

All participants underwent TRUS examination at the urology department to determine the prostate volume and uroflowmetry to determine the maximum urinary flow rate, urination time, and volume.

Definition of BPH was defined and diagnosed according to the following criteria

(i) Prostate volume $>$30 mL; (ii) peak urinary flow rate $<$15 mL/s, mean urinary flow rate $<$10 mL/s at a voided volume between 200 and 400 mL; and (iii) PSA level of $<$10 ng/mL. Patients with higher serum levels of PSA were excluded due to the possible presence of prostate cancer [2].

Statistics

Data were collated and then analysed using IBM SPSS software package version 20.0. Comparisons between groups for categorical variables were assessed using the chi-square test. Student's *t*-test was used to compare two groups for normally distributed quantitative variables, while an ANOVA was used for comparison between more than two groups. The Kruskal–Wallis test was used to compare different groups for abnormally distributed quantitative variables and the Mann–Whitney *U*-test was used for comparing each two groups. Multivariate logistic regression was assessed to find the factors independently affecting AGA. Statistical significance of the obtained results was judged at the 5% level.

Results

The two groups did not significantly differ in age, at a mean (SD) of 44.16 (8.48) years for the AGA group and 44.08 (8.54) years for the no-AGA group (Table 1). In the AGA group, 40% were classified as having mild severity AGA (grade I, II, III), 28% had moderate severity AGA (IV, V), and 32% had severe AGA (IV, VII). The AGA group more frequently reported a family history of AGA and family history of BPH but this was not significant compared with the no-AGA group (Table 2). There was a significant difference between the two groups for the following variables: waist circum-

Table 1 Comparison between the two studied groups according to quantitative data.

Variable	AGA group (n = 300)	No-AGA group (n = 100)	P
Mean (SD):			
Age, years	44.16 (8.48)	44.08 (8.54)	0.957
Weight, kg	83.74 (16.79)	83.88 (16.90)	0.962
Waist circumference, cm	103.22 (23.54)	96.60 (115.20)	0.040*
Height, cm	169.63 (9.26)	169.78 (9.33)	0.926
BMI, kg/m^2	29.92 (4.30)	26.16 (4.78)	$<$0.001*
Median (range):			
Fibrinogen, mg/dL	384.0 (142.0–750.0)	214.0 (142.0–600.0)	$<$0.001*
FBS, mg/dL	145.0 (121.0–297.0)	95.50 (70.0–291.0)	$<$0.001*
Cholesterol, mg/dL	196.0 (121.0–297.0)	121.0 (78.0–220.0)	$<$0.001*
TG, mg/dL	133.0 (36.0–370.0)	138.0 (36.0–370.0)	0.768
HDL, mg/dL	42.0 (28.0–90.0)	41.50 (28.0–90.0)	0.943
CRP, mg/mL	13.17 (2.90–127.0)	2.98 (1.23–127.0)	$<$0.001*
ESR1, mm/1 h	10.50 (5.0–135.0)	9.0 (5.0–86.0)	0.137
ESR2, mm/2 h	21.0 (14.0–167.0)	17.50 (12.0–61.0)	$<$0.001*
SHBG, ng/dL	29.40 (11.80–67.90)	18.0 (9.70–45.0)	$<$0.001*
HbA1c, ng/dL	7.90 (6.40–21.0)	4.70 (2.34–10.0)	$<$0.001*

TG, triglyceride. Abnormally distributed quantitative data are expressed as the median (range) and compared using Mann–Whitney *U*-test; and normally distributed data are expressed as the mean (SD) and compared using the Student's *t*-test.

* Statistically significant at $P \leqslant 0.05$.

Table 2 Comparison between the two studied groups according to different qualitative data.

	AGA group, *n* (%) (*n* = 300)	No-AGA group, *n* (%) *n* = 100)	*P*[*]
Blood pressure (> 130/85 mmHg)	51 (17.0)	18 (18.0)	0.879
Smoking	99 (33.0)	32 (32.0)	0.902
Drug Intake	99 (33.0)	34 (34.0)	0.903
FH of AGA	204 (68.0)	66 (66.0)	0.806
FH of BPH	159 (53.0)	54 (54.0)	0.908

FH, family history.
[*] Chi-square test.

ference, BMI, fibrinogen level, FBS, cholesterol, CRP, ESR2, and glycosylated haemoglobin (HbA1c; Table 1).

In all, 153 patients (51%) in the AGA group manifest metabolic syndrome according to the Adult Treatment Panel-III criteria vs 28 in the no-AGA group (28%), which was significant (Table 3).

Patients in the AGA group had a significantly higher mean prostate volume and a significantly lower mean maximum urinary flow rate (Table 4). BPH diagnostic criteria were met by 108 (36%) patients in the AGA group vs 6.8 (6.8%) in the no-AGA group (Table 4). There was a significant association between metabolic syndrome, maximum urinary flow, and SHBG, with AGA severity while BPH and prostatic volume did not show this significant association (Table 5).

Using multivariate logistic regression analysis, both BPH and metabolic syndrome were significant independent variables associated with AGA (Table 6), with BPH being more significant than metabolic syndrome.

Discussion

AGA in genetically susceptible males is induced by androgens and their metabolites. The enzyme 5α-reductase converts testosterone to DHT, which causes progressive thinning of the hair shaft and villus of terminal hairs in the vertex, frontal and temporal areas of the scalp. By the time the hair follicles become miniaturised and atrophic in these patients a recognisable variable degree of hair loss is seen [1]. BPH occurs in middle-aged men and is associated with high levels of DHT formed by 5α-reductase enzymatic conversion of testosterone to DHT. The effect of DHT on the prostate is hyperplasia followed by the urinary symptoms [2]. Accordingly, both AGA and BPH are influenced by 5α-reductase enzymatic pathways. However, it is unclear whether these diseases are related epidemiologically. The present study was a cross-sectional trial to evaluate the prevalence of BPH among patients with AGA.

Table 3 Comparison between the two studied groups according to metabolic syndrome Adult Treatment Panel-III criteria.

Variable	AGA group (*n* = 300)	No-AGA group (*n* = 100)	*P*
Blood pressure (> 130/85 mmHg), *n* (%)	51 (17.0)	18 (18.0)	0.879
Mean (SD) waist circumference, cm	103.22 (23.54)	96.60 (115.20)	0.040[*]
Median (range)			
FBS, mg/dL	145.0 (121.0–297.0)	95.50 (70.0–291.0)	< 0.001[*]
TG, mg/dL	133.0 (36.0–370.0)	138.0 (36.0–370.0)	0.768
HDL, mg/dL	42.0 (28.0–90.0)	41.50 (28.0–90.0)	0.943
Metabolic syndrome, *n* (%)	153 (51.0)	28 (28.0)	0.007[*]

TG, triglyceride. Qualitative data are described using *n* (%) and are compared using the chi-square test. Abnormally distributed quantitative data are expressed as the median (range) and compared using Mann–Whitney *U*-test; and normally distributed data are expressed as the mean (SD) and compared using the Student's *t*-test.
[*] Statistically significant at $P \leqslant 0.05$.

Table 4 Comparison between the two studied groups according to BPH criteria.

Variable	AGA group (*n* = 300)	No-AGA group (*n* = 100)	*P*
Median (range)			
Prostate volume, mL	27.0 (12.40–97.90)	21.50 (12.40–97.90)	< 0.001[*]
PSA level, ng/mL	6.07 (0 .84–23.0)	1.51 (0.20–8.03)	< 0.001[*]
Urinary flow rate, mL/s	13.0 (4.50–34.0)	25.0 (12.0–45.0)	< 0.001[*]
BPH, *n* (%)	108 (36)	6.8 (6.8)	< 0.001[*]

Qualitative data are described using *n* (%) and are compared using the chi-square test. Abnormally distributed quantitative data are expressed as the median (range) and compared using Mann–Whitney *U*-test
[*] Statistically significant at $P \leqslant 0.05$.

Table 5 Relation between severity of AGA grade and different variables in the AGA group.

Variable	AGA grade			*p*
	Mild (grade I, II, III) (n = 120)	Moderate (grade IV, V) (n = 84)	Severe (grade VI, VII) (n = 96)	
Metabolic syndrome, *n* (%)	42 (35.0)	45 (53.6)	66 (68.8)	0.017*
Median (range)				
Urinary flow rate, mL/s	15.0 (10.80–32.0)	12.0 (4.50–31.0)	12.30 (5.40–34.0)	0.001*
Prostate volume, mL	26.20 (12.40–46.20)	27.00 (13.50–97.90)	27.65 (16.00–72.60)	0.225
SHBG, ng/dL	37.20 (18.0–67.90)	27.25 (12.20–43.0)	28.30 (11.80–45.40)	0.002*
BPH, *n* (%)	27 (22.5)	36 (42.9)	33 (34.4)	0.196

Qualitative data are described using *n* (%) and are compared using the chi-square test. Abnormally distributed quantitative data are expressed as the median (range) and compared using the Kruskal–Wallis test.
* Statistically significant at $P \leqslant 0.05$.

Table 6 Multivariate logistic regression analysis for AGA.

	B	SE	Sig.	OR	95% CI	
					LL	UL
BPH	1.517	0.572	0.008*	4.559*	1.485	13.997
Metabolic syndrome	0.784	0.386	0.042*	2.191*	1.027	4.672

B, unstandardised regression coefficient; SE, stand error; UL, upper limit; LL, lower limit.
* Statistically significant at $P \leqslant 0.05$.

There are some discrepancies in studies performed for determining the relationship between AGA and BPH. For example, Oh et al. [11] studied 225 patients with BPH, with a mean age of ≈70 years, and found more severe AGA (Norwood–Hamilton scale) and a greater frequency of family history of AGA compared with control subjects. Arias-Santiago et al. [6] and Kaplan [7] showed that AGA might be an early marker of BPH, and their results are in agreement with the present study. In another similar study of elderly patients (mean age ≈70 years), Chen et al. [12] observed a significantly higher prevalence of AGA in those with a prostate volume of >30 mL compared with those with a smaller prostate (83.3% vs 61.3%), which did not vary in relation to the severity of AGA (Norwood–Hamilton scale). Those findings are in agreement with our present results in which the prostate size was significantly larger in the AGA-group patients vs those in the no-AGA group (26.85 vs 21.50 mL), also in our study there was no significant relation between the prostate volume and the severity of AGA. The presence of BPH in the control group in the Chen et al. [12] study cannot be excluded, as no TRUS or uroflowmetry studies were undertaken.

In the development of BPH, three main components have been suggested, a static component related to the overgrowth of the prostate gland influenced by androgens (obstructive symptoms), a dynamic component associated with detrusor muscle hypercontractility (irritative symptoms), and a third component related to chronic inflammation leading to prostate overgrowth [13].

The present study showed a significantly higher incidence of metabolic syndrome in the AGA-group patients compared with those in the no-AGA group (51% vs 28%), also metabolic syndrome was significantly associated with AGA severity. This is in agreement with the Arias-Santiago et al. [10] study, which reported the prevalence of metabolic syndrome according to the Adult Treatment Panel-III criteria in male patients with AGA.

Abdominal obesity or waist circumference was recently defined as an essential criterion for the diagnosis of metabolic syndrome. In the present study, there was a higher mean waist circumference in men with AGA compared with control subjects, although there was no significant difference in weight or height. This is in agreement with Arias-Santiago et al. [10] study and indicates that patients with AGA undergo an abdominal redistribution of fat, which is considered an important cardiovascular risk factor.

In our present study, patients with AGA had significantly higher BMI, cholesterol, FBS, and HbA1c compared with the controls. Similar results were found by Sadighha and Zahid [14], who confirmed that male patients with AGA have elevated triglyceride levels.

Higher FBS and HbA1c values were also reported by Hirsso et al. [15], who found that 21% of patients with AGA had diabetes vs 12% of control subjects, which is related to peripheral insulin resistance.

Interestingly, some studies, such as that of Lee et al. [16], suggest that BPH is associated with abdominal obesity, which is in agreement with our present study. The physiological mechanisms by which obesity promotes BPH remain to be described. A potential explanation is inflammation; obesity may influence prostate growth through mechanisms other than sex steroid growth pathways. Obesity is one of the main components of the metabolic syndrome and is associated with systemic inflammation and oxidative stress [17].

In the present study, the mean fibrinogen, CRP and ESR2 values were significantly higher in the patients with AGA vs the control subjects, these variables are referred to as ‘acute phase reactants’, as they play a role in acute inflammation. Chronic inflammation was found

to play an important role in the development of insulin resistance, endothelial dysfunction, and CVD [18]. A chronic inflammatory state underlying AGA, reflected by higher mean values of acute phase reactants, may favour an increase in proinflammatory cytokines found in the arterial wall and hair follicle. Hence, the micro-inflammation found in the hair follicle, which may be implicated in the pathogenesis of AGA, could be the manifestation of a systemic inflammation, which is related to the higher frequency of metabolic syndrome and CVD in patients with AGA [18].

There are multiple lines of evidence connecting BPH with inflammation. BPH in surgical specimens is associated with inflammation and the extent and severity of inflammation corresponds to the degree of prostate enlargement, in addition, the level of CRP is associated with higher risk of LUTS [19]. Insulin secretion, secondary to diabetes, may induce an enlarged prostate because of its structural similarity with insulin-like growth factor [20] and higher insulin is associated with lower SHBG, which may raise the androgen/oestrogen ratio, increasing the risk of BPH.

Future studies may clarify if treatment of patients with AGA may benefit any concomitant benign prostatic hypertrophy, which would be present at an earlier stage in its natural evolution. Further research is also warranted into the long-term role of 5α-reductase inhibitors. The precise role of dutasteride, widely used for BPH in patients with AGA also deserves investigation. Some authors have suggested that the protective effects of finasteride and dutasteride against prostate cancer have yet to be confirmed.

In conclusion, dermatologists, urologists and primary care physicians should monitor patients with early-onset AGA for the development of urinary symptoms, to permit an earlier diagnosis of BPH; and for metabolic syndrome symptoms, to permit early diagnosis of cardiovascular risk factors.

Conflicts of interest

None.

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None.

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