Arab Journal of Urology (2016) **14**, 157–162

Arab Journal of Urology
(Official Journal of the Arab Association of Urology)

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PROSTATIC DISORDERS
ORIGINAL ARTICLE

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

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Received 10 November 2015, Received in revised form 14 January 2016, Accepted 20 January 2016
Available online 23 February 2016

KEYWORDS

Androgenetic alopecia;
Benign prostatic hyperplasia;
Metabolic syndrome;
Cardiovascular risk

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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Peer review under responsibility of Arab Association of Urology.

Production and hosting by Elsevier

http://dx.doi.org/10.1016/j.aju.2016.01.003

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.