

Clinical and Trichoscopic Evaluation of Male Androgenetic Alopecia Patients

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Abstract

Background: Androgenic alopecia (AGA) is a prevalent hair condition characterised by progressive pattern hair loss in the frontotemporal and vertex areas of the scalp in both men and women. Androgenic alopecia worsens with age and lowers patients' body image satisfaction. Androgenic alopecia affects at least fifty percent of men by the age of fifty, and up to seventy percent of all men in their later years. Aim of the Work: To assess the effectiveness and safety of therapies for androgenetic alopecia in males. Patients and Methods: his randomized interventional study included 40 male patients complaining of AGA. They were selected from those attending the Outpatient Clinic of the Dermatology & Andrology Department, Benha University Hospital science 2019 to 2023. This research comprised 40 male patients with AGA complaints. They were picked from Benha University Hospital's Dermatology and Andrology Department Outpatient Clinic patients. Results: It was discovered that medications for the management of male androgenetic alopecia are both safe and effective. Conclusion: For the treatment of male androgenetic alopecia, it was discovered that therapies are both safe and effective.

Keywords: Treatments, Androgenetic, Alopecia.

Male Androgenetic Alopecia

Hair loss represents the most frequent and distressing clinical complaint encountered by dermatologists in clinical practice. Androgenetic alopecia, TE, and AA are the three most common types of hair loss, with AGA being the most prevalent type in dermatology practice. Androgenetic alopecia or pattern baldness, characterized by a progressive miniaturization of the hair follicle, is a nonscarring hair loss disorder that predominantly affects up to 80% of men and 50% of women during adolescence and postadolescence [1].

AGA etiology is purported to be polygenic and androgenic, but the underlying molecular mechanisms governing its onset and progression are not fully understood [2]. Androgenic alopecia's pathobiological suspects include androgens (i.e., 5 α -dihydrotestosterone), hair-cycle-regulating signaling proteins (i.e., interleukin 6, transforming growth factor β 1&2), inflammatory fatty acid derivatives (i.e.,

prostaglandin D2), signaling pathways and pathway inhibitors (i.e., Wnt/ β -catenin,

dickkopf-1), and concomitant morphology (i.e., vascularity, perifollicular fibrosis) [3] Activation of the androgen receptor shortens the anagen or growth phase in the normal hair growth cycle. In AGA,

excessive activation leads to follicular miniaturization through a progressively shorter anagen phase,

resulting in thinner and shorter hair follicles which in the end may not even penetrate through the epidermis. Pathological specimens will show a decreased 5: 0 ratio of anagen to telogen hair where the norm is 12: 1 [4].

Male patients with AGA have higher production of dihydrotestosterone, higher levels of 5 alpha-reductase and androgen receptors in balding scalp [5].

The androgenetic alopecia patients were classified according to the severity of baldness based on Hamilton Norwood's classification in males [6, 7].

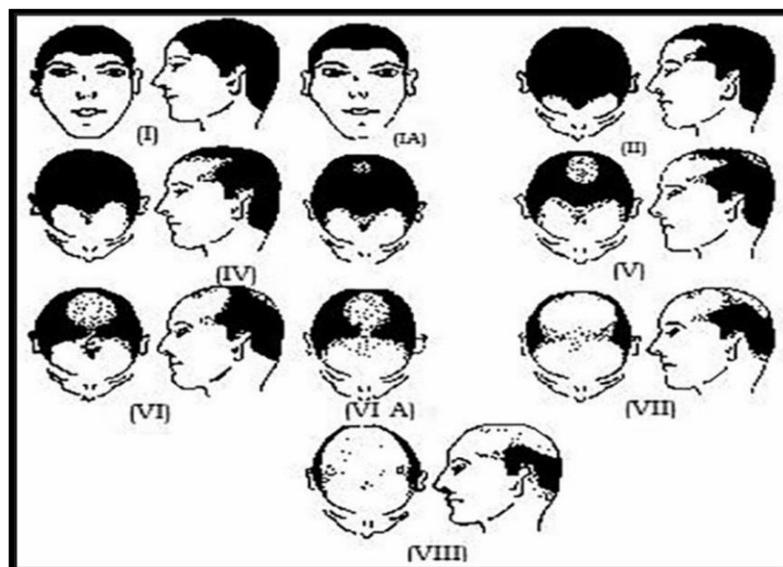


Fig. (1) Hamilton classification of male pattern hair loss. Type III has not been included in this figure as a large variety of conditions were included in this type.

Type I: There is an absence of bilateral recessions along the anterior border of the hairline in the frontoparietal regions. In this, there is a variant form in which the entire anterior border of the hairline lies high on the forehead, which is referred to as Type IA.

Type II: The anterior border of the hairline in the frontoparietal regions has triangular areas of recession, which tend to be symmetrical and extend no farther posteriorly than a point 3 cm anterior to a line drawn in a coronal plane between the external auditory meatuses. Hair is also lost, or sparse, along the midfrontal border of the scalp but the depth of the affected area is much less than in the frontoparietal regions.

Type III: Borderline cases were listed separately as Type III, which also included scalps in which classification is rendered inaccurate due to scars, lateral asymmetry in denudation, unusual types of sparseness and thinning of the hair, and other factors.

Type IV: It represents the minimal hair loss considered sufficient to represent baldness. There are deep frontotemporal recessions, usually symmetrical, and are either bare or very sparsely covered by hair. These recessions extend farther posteriorly than a point, which lies 3 cm anterior to a coronal line drawn between the external auditory meatuses. If hair is sparse or lacking as a broad band along the entire anterior border of the hairline, it is classified as Type IVA.

Type V: It includes extensive frontoparietal and frontal recessions with a sparseness or absence of hair on the crown.

Type VI: In this type, the tonsural region of alopecia remains separated from more anteriorly located areas of denudation by a laterally-directed bar of scalp in which the hair is only slightly sparse. An island of hair lies in the midline anterior to this laterally-directed hairy bridge. In the variant pattern, Type VIA, the peninsula or island of mid-frontal hair is sparse or lost.

Type VII and VIII: In these types, the

horseshoe-shaped area of sparse hair or of denudation is unbroken by any well-haired, laterally-directed bridge of scalp. These are a result of the spread and confluence of the tonsural and the anteriorly located regions of alopecia.

Hamilton classification set a benchmark for future classifications of male patterned hair loss as it had elaborately described the various evolutionary stages of hair loss and had based the classification on them but it did not describe a few rare patterns of hair loss, which were later on added by Norwood to give the commonly used Hamilton-Norwood classification.

Trichoscopic evaluation

Trichoscopy (Scalp dermoscopy) is a fast and noninvasive method for evaluating hair density. This method is simple, quick, easy to perform, well-accepted by patients, and useful for monitoring treatment, determining severity of the disease and follow-up [8].

In trichoscopic examination, the hair and scalp were examined in the three areas: frontal, temporal and occipital.

Major trichoscopic criteria of androgenetic alopecia

More than four yellow in four areas of the frontal region (70× magnification), decreased average thickness of hairs in the frontal region when compared with the occipital region (evaluation of at least 50 hairs from each area), and more than 10% of fine hairs (<0.03 mm) in the frontal region.

Minor trichoscopic criteria of androgenetic alopecia

Ratio between the number of isolated hairs per follicular unit of the frontal area and the number of isolated hairs per follicular unit of the occipital area is >2: 1, ratio between the number of fine hairs in the frontal area and the number of fine hairs in the occipital area is >1.5: 1 and ratio between the number of hyperpigmented follicles in the frontal area and the number of hyperpigmented follicles in the occipital area is >3: 1.



Fig. (5) Trichoscopy of male androgenetic alopecia.

Hair examined to assess hair density/ cm square area: examined at a fixed site in the scalp by trichoscope and hair loss: counted after constant combing of patient's hair for 1 minute with the same comb.

There are several traditional treatment options available for the treatment of AGA; however, their effectiveness remains limited. Therefore, a safe and effective treatment modality with fewer side effects that can significantly benefit patients with AGA in a dermatology practice setting is highly desirable [9].

Oral finasteride and topical minoxidil are the only FDA-approved treatment for AGA management. Finasteride is a 5 α -reductase inhibitor, which prevents the conversion of testosterone to

dihydrotestosterone (DHT) [10].

Oral minoxidil is an antihypertensive and a vasodilator. Topical minoxidil has a complex mechanism of action. It results in the synthesis of prostaglandins and vascular endothelial growth factors in dermal papilla and the opening of potassium channels, resulting in an increase in anagen to telogen ratio [11].

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