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ANDROGENETIC ALOPECIA

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ABSTRACT

The most prevalent type of nonscarring alopecia is androgenetic alopecia. Our knowledge of the pathogenesis of androgenetic alopecia has advanced over the past few years, opening the door to more effective treatment approaches. In addition to identifying a more recent genetic basis for the disorder, recent research has focused on the involvement of stem cells in the pathophysiology of androgenetic Alopecia. AGA can now be diagnosed by Trichoscopy. While the major treatment options continuous to be topical minoxidil, systemic Finasteride and hair transplantations, newer modalities are under investigation. Evidence-based principles have also been used to produce specific recommendations for diagnosis and therapy. The latest ideas regarding AGA are reviewed in this Article .We have attempted to highlight current understanding of the genetic basis of AGA in relation to the pathophysiology. hormones, genetics, deficit in micronutrients, and microinflammation. Whereas psychosocial anguish and cutaneous correlate of cardiovascular illnesses have become causes of unrelenting. Men are more likely to get AGA, a systematic hair loss.

KEYWORDS: Androgenetic Alopecia, History, Hair loss type, Diagnosis, Treatment.

INTRODUCTION

Androgenetic alopecia also known as pattern alopecia, is an non functional or dead hair follicles. It is most common hair loss in men and women.Androgenetic alopecia occurs in males that is called as male androgenetic alopecia. Regarded females that is called as female androgenetic alopecia is considered below 30 years and later oneset 50 years. Androgenetic alopecia mainly genetic and androgen hormon dependent. Other facters, such as chronic micro informatio, oxidative stress, etc.[1]

During the times of Shakespeare, when the pathogenesis of androgenetic alopecia (AGA) was not clearly understood, the only option for those affected was to live with it, other than using wigs for cosmetics purposes. Now, we have a deeper understanding of the genetics, molecular basis and pathophysiology of AGA, which has paved the way for effective treatment modalities. Alopecia induced by androgens in genetically predisposed individuals is termed androgenetic alopecia.[2]

AGA mainly genetic and hormone androgen dependent. However, many other factors, such as chronic micro-inflammation, and oxidative stress, have also been implicated. Based on the induction of chronic inflammation, AGA has been linked to coronary heart disease, metabolic syndrome and prostate cancer.

Subjects with AGA have been reported to experience cosmetic implications, with low self-esteem, anxiety and depression. AGA has a more significant emotional impact than alopecia areata and causes more distress from anxiety, depression and a worse quality of life. Similarly, Enitan et al. reported that AGA significantly affected domains, including symptoms, function, emotions, stigmatization and self – confidence.[3]

History

Androgenetic alopecia manifest as gradual and predictable hair loss pattern after puberty**.** A proper history often helps to rule out other causes of the hair loss like telogen effluvium. The typical history is of chronic hair loss with thinning mainly over the frontal, parietal or vertex areas.[3] The patient might also complain of itching and trichodynia. History of systemic diseases, new medications especially within the previous year should be taken. Family history is usually[4] positive for AGA. Diet is another important aspect of history, to rule out nutrition related effluvium. Lifestyle related enquiries should cover effect of traction, smoking and ultraviolet exposure on AGA, all of which have been implicated as aggravating factors. In

female patients, careful attention mus be given to assess any associated hormonal dysfunction.[5]

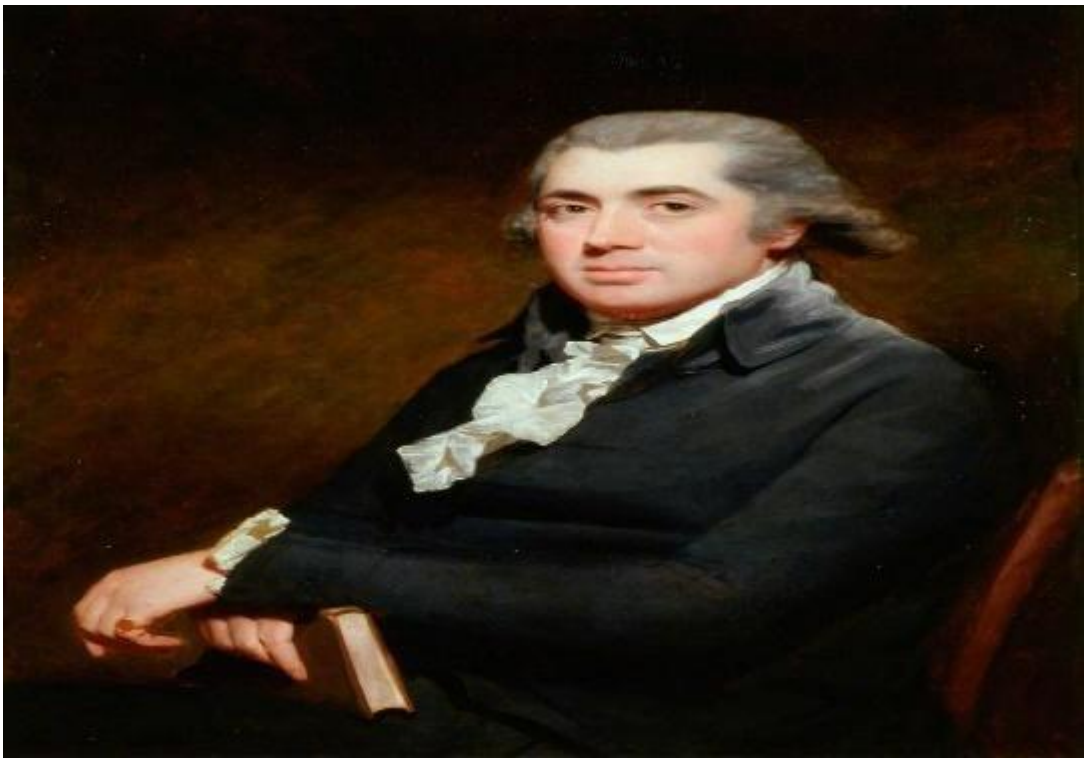


Fig. 1: Dr. James. B. Hamilton.[60]

1) Dr. James B. Hamilton

Hamintons works**;** in the 1940s**,** hamiltons studies on enuchus and castrated men demonstrated that without the precence of male [androgen], comman bladness did not devlop. He established that hair loss was influenced by main three facters; agr, the persons of androgens, genetic predisposion.[7]

His research the ground work for undanstanding the mechanism behind androgenetic alopecia.

Epidemiolog

Men

Androgenetic alopecia (AGA) is considered to be the most common type of baldness characterized by progressive hair loss. AGA can affect all races, but the prevalence rates vary. Prevalence is considered to be highest in Caucasians. It is estimated that prevalence rates in Caucasian populations is around 30% for men in their 30s, 40% for men in their 40s and 50% for men in their 50s. In the Indian context, a population based study of 1005 subjects showed a 58% prevalence of AGA in males aged 30-50 years. In oriental races, a lower prevalence

has been shown. In a Chinese study by Wang et al., the overall prevalence was 21.3%, while in a Korean study, the overall prevalence was 14.1%. All studies demonstrate a gradual increase in incidence with age.[8]

Women

Epidemiological studies of AGA in women are fewer in number. A study by Norwood, showed a total prevalence of around 19% in a population of 1006 Caucasian patient). In a Chinese population study, the prevalence was only 6.0% and a Korean study had a relatively similar lower prevalence of 5.6%, suggesting that like in men, the prevalence is considered to be lower in oriental races compared to Caucasians. The incidence of AGA in women also tends to increase with age. It should be noted that experts have suggested that female androgenetic alopecia is not exactly the female counterpart of male AGA. A better term for female AGA would be ′female pattern alopecia′ or ′female pattern hair loss.[9]

Causes of androgenetic alopecia

1) Dihydrotestosterone [hormone]
2) Androgens
3) Genetic sensetivity

Clinical Features

While there are different grading systems available for AGA, the most accepted is the modified Norwood- Hamilton classification [Table- 1], modified from the earlier Hamilton classification, consisting of seven broad groups and four specific variant types.[11]

Table 1: Modified Norwood-Hamilton classification.[12]

Type	Clinical defination
1st	Minimal recession of the hairline along the anterior border in the frontotemporal (FT).
2nd	The anterior border of the hair in the FT region has triangular areas of recession that tends to be symmetrical.
3rd	Charecterized by deep FT hair recession ,usually symmetric and either bald or sparsely covered with hair.these areas of hair recession extend further posterior than a point that lies approximately 2cm anterior to a line drawn in a coronal plane between the external auditory meatus on either side.
4th	The frontal and FT recession is more severe then type 3rd .there is also sparsenss or absence of hair in the vertex area. These bald areas extensive, but seprated from each other by a band or moderate dense dense hair that joins the fully haired fringe on each side of the head.

5th	The hair loss over the vertex and FT areas is larger than in type 4th and the band of hair between them is narrower and sparser.

In women, typically three patterns have been described

- Diffuse thinning of the crown area with preservation of the frontal hair line
- Thinning and widening of the central part of the scalp with breach of the frontal hair line
- Thinning associated with bi-temporal recession.

The commonest grading scales used for female androgenetic alopecia are the three-point Ludwig scaleand the five-point Sinclair scale [Table- 2] and [Table- 3].

Table 2: Ludwig's scale for female AGA.[13]

Stage 1st	Thinning of hair is seen mainly over the anterior part of the crown with minimal widening of the parting width .
Stage 2nd	Thinning of the crown becomes more evident because of an increase in the number of thin and short hairs.
Stage 3rd	The crown becomes almost total bald .there is significant widening of the parting width.but the frontal hairline is still maintained .

Table 3: Sinclair scale for female pattern AGA.[14]

Grade 1st	Is normal this pattern is found in all girls prior to puberty but in only forty five percent of women aged eighty or over .
Grade 2nd	Shows a widening of the central part.
Grade 3rd	Shows a widening of the central part and thinning of the hair on either side of the central part.
Grade 4th	Reveals the emergence of a diffuse hair loss over the top of the scalp.
Grade 5th	Indicates advanced hair loss.

A newer, systematic and universal classification has been suggested by Lee, et al., known as the basic and specific classification (BASP). The basic (BA) types represent the shape of the anterior hairline, and the specific types (SP) represent the density of hair on distinct areas (frontal and vertex). There are four basic types (L, M, C, and U) and two specific types (F and V). The final type is decided by the combination of the assigned basic and specific types. The basic types are classified by the English alphabetical letter shape of the anterior hairline, except L type, which means linear.[15]

Type L - No recession is observed along the anterior border in the frontotemporal region. It appearslinear. Type M - Recession in the frontotemporal hairline is more

prominent than the mid-anterior hairlineThehairlineresemblestheletterM.

Type C - Recession in the mid-anterior hairline is more prominent than the frontotemporal hairline. The entire anterior hairline regresses posteriorly in the shape of half-circle, resembling the letterC.

Type U - The anterior hairline recedes posteriorly beyond the vertex forming a horseshoe shaperesemblingtheletter.

Type F - This represents a general decrease in the density of hair over the entire scalp, regardless of the anterior hairline. It is usually more marked over the frontal area of the scalp.

Type V - Hair loss is seen more distinctly in the vertex than in the frontal area.[16]

Diagnosis

1) General scalp and hair examination

The scalp is usually normal in AGA, but look for factors which can aggravate AGA like seborrheicdermatitis and photo-damage. The main aim of clinical examination is to identify whether or not the hair loss is patterned.[17]

2) Pull test

The "pull test" is a simple method to assess the severity of hair loss. About 60 hairs are grasped between the thumb and the index and middle finger. The hairs are then gently but firmly pulled. A negative test (six or less hairs/less than 10% obtained) indicates normal shedding, whereas a positive test (more than six hairs or 10% obtained) indicates definite active shedding of hair. Shampooing should be withheld for 24 hrs prior to a pull test.[18]

3) Trichoscopy

Trichoscopy has emerged as an useful tool in the diagnosis of androgenetic alopecia. Important features of AGA on trichoscopy are hair diameter diversity (HDD) greater than 20% (which corresponds to vellus transformation), perifollicular pigmentation/peripilar sign (the commonest change seen in Asians) and yellow dot [Figure- 2] The term ′anisotrichosis′ has been proposed to describe the HDD seen in AGA.

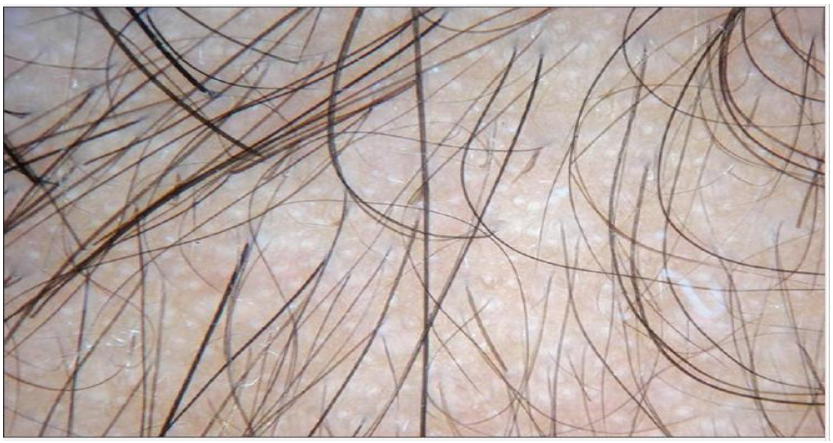


Fig 2: Trichoscopy showing increase HDD.[19]

4) Hair wash test

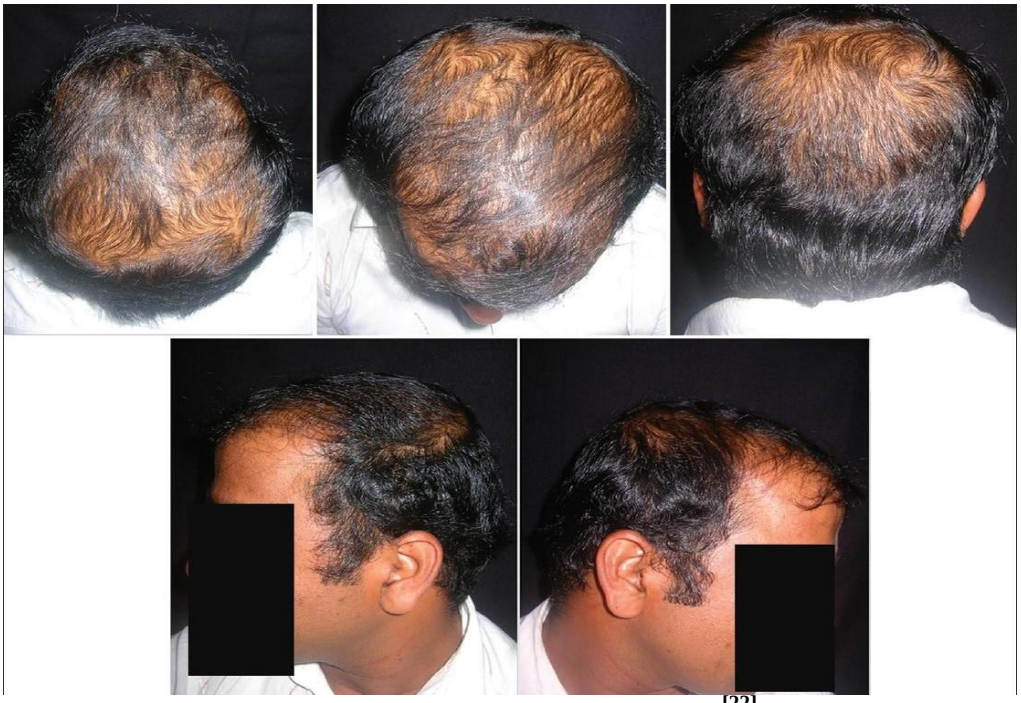
Rebora et al., have devised a test known as the AGA/TE wash test to distinguish between AGA and TE based on the count of vellus and terminal telogen hairs that are rinsed out on washing the scalp after a five day abstinence from washing and shampooing. The results are given in terms of total telogen hairs and the percentage of telogen vellus hairs.[20]

5) Laboratory Investigations

The general consensus is that extensive laboratory investigations are not required for AGA, especially in males. Some authors recommend testing for Prostate Specific Antigen prior to starting finasteride in men above the age of The main aim of laboratory investigations in women is to rule out any underlying hormonal dysfunction especially polycystic ovarian disease. The tests recommended are - free androgen index test, Dehydroepiandrosterone (DHEAS) and prolactin.[21]

6) Global Photography

A global photograph of a patient with hair loss is a useful tool for follow-up and assessment of treatment response. This requires among other things, a cooperative patient with clean, dry hair and ideally a technician who is able to take the time to comb and prepare the hair precisely the same way at each office visit The patient should be advised to maintain the same hair style and color. Multiple images should be shot covering all areas of the scalp.



Fig; Global photography in AGA.[22]

Treatment

1) Minoxidil

Topical minoxidil is the main treatment for AGA and is also used off-label for other forms of hair loss It was repurposed as a hair loss treatment when hypertensive patients on oral treatment experienced increased hair growth as a side effect. Topical minoxidil is thought to dilate scalp blood vessels, promoting hair growth by improving nutrient delivery to hair follicles It is available in different forms, such as solutions, foam, and shampoo, with the 5% solution being more effective than the 2% solution.[24]

2) Laser therapy

In 1967, a study showed that low-level light/laser therapy (LLLT) using a ruby laser promoted hair growth in mice LLLT devices were later FDA-cleared in 2007 for men and 2011 for women as a potential treatment for hair loss. LLLT stimulates hair growth by influencing the hair cycle using specific wavelengths between 650 nm and 1200 nm, with LLLT red or near-infrared light ranging between 600 and 950 nm and fluences between 2 and 10 Joules per square centimetres (J/cm^2), over 15-20 minutes, 3 times a week for 6 months.[25]

3) Hair transplantation

Hair follicle transplantation is a surgical procedure that involves removing and transplanting

hair follicles from non-androgen sensitive areas to areas affected by AGA. The transplanted follicles do not miniaturize, grow in groups of 1 to 4 hairs and are harvested as units. In 2009, the FDA-approved a robotic hair restoration device to assist surgeons. Later in 2011, the ARTAS system was also approved to harvest curly hair in black men. ARTAS robotic hair transplant is a minimally invasive hair restoration system that uses artificial intelligence technology to restore hair faster and more precisely than traditional hair restoration methods.[26]

4) Phytomedicine

Phytomedicine, the use of plant-based products for medicinal purposes, is popular for treating AGA and can be used as a complementary or alternative treatment.Plants like Serenoa repens, Panax ginseng, Curcuma aeruginosa, Cucurbita pepo and Trifolium pratense palm extract (tocotrienol/tocopherol complex), horsetail and ashwagandha have been reported to treat AGA. Although high quality evidence from controled studies are needed these plants are reported to inhibit 5-alpha-reductase, lower cortisol levels, reduce inflammation, promote homeostasis, and maintain collagen stores.[27]

5) Injectables

Platelet-rich plasma (PRP) refers to a naturally derived mixture of platelets in a concentrated plasma solution, typically containing over 1,000,000 platelets per microliter or 2-7 times the concentration found in regular blood. The application of PRP has been shown to stimulate hair growth, enhance cell survival, and extend the active growth phase (anagen) of the hair cycle. It enhances grafting and improves follicular unit survival, resulting in hair density and thickness, according to recent meta-analyses of 30 articles with 687 patients using various injection methods. However, the efficacy of PRP compared to other treatments for androgenetic alopecia (AGA) remains uncertain due to a lack of standardized protocols, long-term follow-up outcomes, and limited clinical evidence.[28]

Future prospects

The discovery of SRD5A2's structure (Fig. 5) in 2020 has opened new possibilities for developing more effective AGA treatments. Understanding its function and interactions allows researchers to identify drug targets and design drugs specifically targeting the protein. This breakthrough has the potential to enhance drug efficacy and safety. It also has implications for treating other diseases. The discovery represents a milestone in AGA treatment and

promises a brighter future for patients.[29]

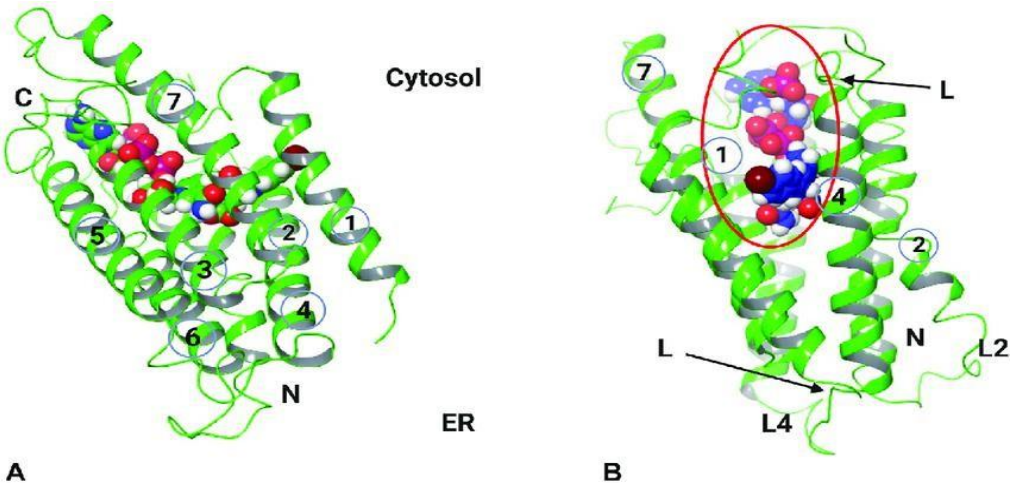


Fig 5: The structure of human SRD5A2. (A), Spheres represent NADP-DHF adduct. L1-6 are the 6 loops connecting the 7 transmembranes (TM), and the TM portion has 254 amino acid residues. (B), The active site inside the 7 TM channels surrounded by L1, L3, and L5, with 2 separate pockets for NADP and DHF (shown in red circle).[30]

New treatments

New insights into AGA's pathogenesis have led to new research prospects . Promising treatments are being developed, including clascoterone, an FDA-approved topical androgen receptor inhibitor initially used for acne. Recent studies indicate clascoterone's potential effectiveness in treating AGA. Patients treated with clascoterone 7.5% twice daily for 6 months experienced reduced hair loss and improvement compared to the placebo group and their baseline.[31]

% of curable

It is not curable condition in india or else in the world .

Progression can be slowed or managed and hair can be restored but it can not be permenently reversed.[32]

Cost of treatment of androgenetic alopecia

1. injection therapy =2000-10000 Rs per section
2. prp therapy =4500-15000 Rs
3. hair trasplants =50000-10 laks
4. oils and lotions =500 Rs
5. oral medications =100-500Rs[33]

Safety

It is not safe for pregnet women.[33]

SUMMARY

Androgenetic alopecia is a comman, progressive hair loss condition influnced by genetic and androgens, cousing charecteristic hair thinning pattern. AGA a common pattrened hair loss disorder caused by a combination of genetic predisposition and androgen sensitivity ,the primary FDA -approved treatments are topical minoxidil and oral finasteride, but they require continuous use to maintain results and can have side effect. Recent research focuses on improving existing treatments through combination therapies (like adding microneedling to minoxidil) and exploring newer options like low-dose oral minoxidil, platlets -rich plasma, and stem cell -based therapies. Androgenetic alopecia (AGA) is a comman, genetically determined type of pattern hair loss in men and women. It is primarily caused by the effect of the hormone (DHT)dihydrotestosterone on hair follicles that are genetically susceptible.

In men, AGA typically presents as bitemporal recession and vertex balding following the Hamilton Norwood pattern, while women usually exhibit diffuse central thinning with preservation of the frontal hairline, described by the ludwig pattern.

The pathophysiology of AGA involves a complex interplay of genetic predisposion ,androgen metabolism, follicular sensitivity to DHT binding to androgen receptors in susceptible folicles shortenes the anagen (growth) phase, prolong telogen (resting) phase, and lead to progressive follicle, producing thinner, shorter hair over time.

Diagnosis is primarily clinical, supported by trichoscopy, pattern recognition, and sometimes laboratroy evalivation to exclude secondary causes, particularly in women. Histopathology, which needed, demonstrates miniaturized follicles with increase telogen to anagen ratios.

Although AGA chronic and progressive, early identification and timely treatment can significantly improved long term outcomes and patient quality of life.

CONCULSION

Androgenetic alopecia is one of the commonest dermatologicall complaints for which patients seek treatment. Androgenetic alopecia can be soured of sigmificant psychological distress to the affected patient. It ts important for the dermatologist to undrstand the process

of daignosis and treatment of androgenetic alopecia. Androgenetic alopecia continues to remain an area where expanding research is adding more information regarding pathogenesis and newer therapeutic option are being developed accordingly. AGA is troubling as a it is common across ages, races and genders, olders she groups and with a higher prevalence in men. It is largely a genetically determined diseases, but the roles of micronutrients deficiency, stress and inflammation have been established AGA is considered a cotaneous marker of cardiovascular and mental health disorders. Treatment over the years has been aimed at satisfying the patients clinical as well as psychological newer medical and surgical methods all dermatologist are hereby advised to have a holistic review of all patients with AGA. As we continue to learn more about AGA and the structure of SRD5A2, we can work toward developing better targeted approches to managing and trating this common and distressing conditions.

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