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Androgenetic alopecia - just a cosmetic defect or a biomarker and risk factor for systemic diseases? - literature review

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Abstract

Introduction: Androgenetic alopecia (AGA) is the most common cause of hair loss. It has traditionally been considered a cosmetic defect, but a growing body of evidence suggests that it may serve as a biomarker and risk factor for systemic diseases, suggesting its potential role in early disease detection and prevention.

Purpose: The aim of this study is to review the available literature, analyze and summarize the current state of knowledge on the relationship between AGA and systemic diseases.

Materials and research methods: The article is based on an analysis of PubMed and Google Scholar research. A literature review was conducted using the following keywords and medical subject headings: androgenetic alopecia; quality of life; cardiovascular disease; hair loss; metabolic syndrome; comorbidity. The analysis included clinical trials, systematic reviews, meta-analyses.

Results: The analysis showed that patients with AGA were more likely to have cardiovascular disease, metabolic or endocrine disorders. Cases of increased risk of prostate and scalp cancers have also been described, as well as negative effects on mental health in people with AGA. These links underscore the need for an interdisciplinary approach to the patient and collaboration between the dermatologist and other specialists.

Conclusion: AGA is not only an aesthetic problem, but can act as a biomarker for systemic diseases and be a signal for further diagnosis. Therefore, screening should be considered, as well as collaboration between dermatologists and other specialists. Future research should focus on the mechanisms linking AGA to other conditions, developing screening protocols, and analyzing the age of onset of AGA as a potential risk for systemic diseases.

Keywords: androgenetic alopecia; quality of life; cardiovascular disease; hair loss; metabolic syndrome; comorbidity

Introduction

Androgenetic alopecia (AGA) is the most common form of hair loss faced by a significant proportion of the population, both men and women. [1] AGA is a genetic basis in most cases, a progressive, non-scarring alopecia caused by a decrease in the activity of androgen-sensitive hair follicles, leading to hair death and hair loss. In men, frontotemporal region and vertex baldness is most common, and in women diffuse apical hair thinning is most common. [2] The frequency of AGA correlates with race, gender and age. It usually occurs in Caucasians and increases with age. [1,3] In about half of men, it already appears before age 50, and in about 80% after age 70. [2,4] In women, it is present less frequently than in men, and the incidence of AGA increases after menopause, due to hormonal changes, including a decrease in estrogen levels and relatively higher androgen effects. [1,5] Hair is usually seen as an important part of appearance and a measure of attractiveness. Their loss is considered unattractive and translates into dissatisfaction with one's appearance, poorer interpersonal relationships, difficulty finding a job and lack of self-confidence. [6-8] AGA is mainly considered an aesthetic problem that affects quality of life and self-esteem. [6]

However, more and more studies indicate that it is not only a local problem, but also a signal and risk factor for systemic diseases, such as psychiatric [9], metabolic [10], cardiovascular [11] or endocrine disorders. [12] Thus, we can use androgenetic alopecia to monitor the development of new diseases or to detect them early and treat them effectively. This opens up many new possibilities in prevention and therapy and the need for a comprehensive look at the problem of AGA. The purpose of this paper is to review the current scientific evidence that addresses the correlation of AGA with systemic diseases and to discuss the pathophysiological mechanisms that link these phenomena.

Pathophysiology of AGA

Genetic, hormonal and environmental factors are primarily involved in the formation of AGA. AGA is a condition with a complex genetic background, involving both genes related to androgen metabolism (e.g. AR, SRD5A2) and genes affecting the hair growth cycle (e.g. WNT10A, FGF5). [13] However, it is the androgen receptor (AR) gene located on the X chromosome and the ectodysplasin A2 receptor (EDA2R) locus that are most strongly associated with AGA formation. [14] Therefore, family history is important in patients with AGA. Salman et al. reported that the incidence of AGA in men increased when AGA was present in the father, brother and second-degree relatives, but there was no correlation with a history of AGA on the mother's side. In contrast, in women, the incidence of AGA increased when there was a positive family history on the mother's and sister's side, but no correlation was found with a history of AGA on the father's and second-degree relatives' side. [15]

Hormones are also a key factor in the development of AGA. The most important hormone in the pathogenesis of AGA is dihydrotestoterone (DHT), which is converted from testosterone by 5α-reductase. DHT has a much higher affinity for AR than testosterone and causes a shortening of anagen (growth) and an increase in telogen (resting) duration and accelerates miniaturization of hair follicles. [16] AGA patients have also been found to have increased AR expression, increased 5α-reductase activity and higher DHT concentrations. [16-18] This mechanism is exploited in the treatment of AGA, which involves the use of oral and topical 5α-reductase inhibitors. [19]

In addition, a significant effect of oxidative stress in the development of AGA has been demonstrated. [20, 21] Bahta AW et al. proved that the premature entry of the hair into the catagen phase, its miniaturization and apoptosis of keratinocytes, as well as the premature aging of the dermal papilla cells of the hair follicle, is led by the excessive production of reactive oxygen species (ROS). [21] Upton JH et al. proved that ROS also leads to the secretion of negative hair growth regulators - TGF-β1/2. [20] The result of all these changes is cellular aging and degeneration and disruption of hair growth pathways. In addition, patients with AGA have been confirmed to have reduced activity of antioxidant enzymes such as superoxide dismutase (SOD) and lower levels of thiol compounds. This exacerbates impaired metabolism of antioxidant enzyme cofactors such as zinc, copper and magnesium. [22] Following a Mediterranean diet, which has antioxidant and anti-inflammatory properties, could prevent AGA, but this requires further research. [23,24]

Some studies point to the influence of environmental factors that may exacerbate the development of AGA. These include a stressful environment, fear of losing hair, [25] sleep disturbances [26], excessive consumption of alcohol [27] and red meat, and insufficient consumption of fruits and vegetables. [28] Studies have also shown that early-onset AGA i.e. below or even 30 years of age, may be predisposed by overweight, metabolic syndrome, insulin resistance, cardiovascular disease, and a positive family history. [2,29]

A detailed understanding of the pathophysiology of AGA may lead to more personalized and effective therapies in the future, as well as link AGA to the occurrence of other systemic diseases, so more research is needed.

AGA vs. comorbidities and systemic complications

In recent years, a growing number of scientific studies have been emerging that describe the co-occurrence of AGA with other diseases, as well as its potential impact on the development of certain conditions. A growing body of evidence points to a link between AGA and the development of metabolic syndrome (MetS). [30,31] MetS is defined as the coexistence of obesity with elevated blood pressure, abnormal carbohydrate and lipid metabolism, which increases the risk of cardiovascular events and type 2 diabetes. [30,32] Studies show that AGA may not only be a symptom of MetS, but also a risk factor. Oxidative stress and inflammation have a significant impact in the pathogenesis of both diseases. Increased ROS production is caused, among other things, by mitochondrial dysfunction, leading to a decrease in ATP production. [24,33,34] Hamed, Ahmed Mohammed, et al. in a recent study showed that the alarin peptide could be a potential biomarker linking AGA and MetS. Its elevated concentration suggests an important role in the pathogenesis of both conditions. [35] A key component of MetS is insulin resistance (IR), which has also been linked to AGA in studies. Men with early onset AGA have higher IR, implying that AGA may be an early marker of metabolic disorders. [36,37] This leads to dyslipidemia, hypertension, type 2 diabetes, atherosclerosis, and cardiovascular disease. [38,39]

There are studies that link heart disease risk to AGA. [40,41] Yamada et al. showed that male pattern baldness at the top of the head is associated with a higher risk of coronary heart disease (CHD), which can lead to myocardial infarction. [42] Arias-Santiago, Salvador, et al. found high levels of triglycerides and low levels of HDL cholesterol, as well as a higher prevalence of atherosclerotic plaque in people with AGA than in controls. [10] Ahouansou et al. also showed a strong association between AGA and hypertension. [43] The potential mechanisms linking AGA to cardiovascular disease are likely related to the already mentioned oxidative stress, inflammatory response, and metabolic disorders, but they still require more research to understand their pathomechanism in detail.

AGA, especially in women, can also be a significant sign of endocrine disruption. It is cited as one of the signs of hyperandrogenism (HA) in women. [44] HA does not occur on its own. It is most often a symptom that occurs in patients with polycystic ovary syndrome (PCOS). A study found that 22% of women who meet diagnostic criteria for PCOS have AGA. [45] There have also been publications that have linked hypothyroidism to AGA. [46] One of the functions of the thyroid gland is to regulate sex hormone binding globulin (SHBG) levels. In hypothyroidism, SHBG levels decrease, resulting in an increase in free testosterone that promotes AGA. [47]

An interesting topic for researchers is the relationship between AGA and prostate cancer incidence. [48] Amaretti et al. demonstrated that vertex baldness significantly increases the risk of prostate cancer. [49] However, this is not equivalent to the possibility of using AGA as a phenotypic marker of prostate cancer risk. [48] There is insufficient evidence for this. Higher PSA levels and higher AGA levels have also been shown in individuals with benign prostatic hyperplasia (BPH). [50] There have also been reported cases of ovarian Leydig cell tumor [51] and thyroid cancer [52] in people with AGA. Studies on a larger group of people are needed to detect correlations between AGA and genitourinary diseases and cancers.

Individuals who go bald lose the scalp's natural protection against harmful UV rays, which can result in the development of scalp cancers. [53] Studies have shown that a lack of hair increases the risk of squamous cell carcinoma (SCC), basal cell carcinoma (BCC), and melanoma. [54, 55]

AGA co-occurs with many other diseases. It may not only be a symptom, but also a risk factor for new diseases, but it is difficult to delineate. It shares a common pathomechanism with metabolic diseases such as oxidative stress and inflammation, among others, but more research is needed to understand the formation of these diseases in detail.

Psychological and social consequences of AGA

In today's world, hair is considered an important part of one's image and influences whether a person is perceived as physically attractive. [56] Many studies indicate that AGA causes negative mental health consequences, especially in young people. [8] The quality of life of patients with AGA is significantly reduced. Lack of hair in these patients can lead to anxiety, distorted body image perception, a sense of aging, and reduced self-esteem. These are factors that may predispose to depression, but in AGA patients, studies have not shown a statistically significant association. [7,57] AGA has been shown to affect loss of self-confidence and avoidance of social contact due to appearance-related shame and stigma, resulting in social withdrawal. [57-59] It can also negatively affect the establishment of romantic relationships, problems in the sexual sphere, not only as a result of the disease, but often as a side effect of the treatment used. [57,60] AGA is not only an aesthetic problem, but realistically affects mental health. Such patients should receive adequate psychological support and their problem should not be underestimated, and early intervention can significantly improve the quality of life of people with AGA. [57]

AGA as a potential biomarker and risk factor for systemic diseases

AGA is often viewed by patients, but also doctors, as an aesthetic problem, rather than looking at the problem from a broader perspective. A number of diseases that coexist with AGA and those that it can lead to make us wonder whether we can consider AGA as a potential biomarker and risk factor for systemic diseases. Noteworthy is the fact that AGA, especially with early onset, predisposes to an increased risk of conditions such as metabolic syndrome, type 2 diabetes and cardiovascular disease, especially premature atherosclerosis, hypertension and myocardial infarction. These diseases, whose association with AGA has been proven by numerous scientific studies, allow us to conclude that AGA can be treated as a visible warning sign, prompting us to consider controlling patients for these diseases and early detection of already existing diseases.

The diagnosis of AGA may be a signal for additional testing for other conditions. Unfortunately, clinical practice lacks official recommendations for screening patients with AGA for comorbidities. Therefore, each patient should be approached individually. However, it is worth considering in particular the assessment of the lipid profile: total cholesterol, LDL cholesterol and triglycerides, measurement of waist circumference, assessment of body mass index (BMI), measurement of blood pressure and assessment of fasting glucose and insulin levels. The introduction of such screening tests for AGA patients can contribute to the early detection and prevention of the induction of new systemic diseases and improve patients' quality of life. A definite advantage of AGA is its easy diagnosis, which is often based on clinical examination alone, without the need for additional expensive tests. This makes it possible to use AGA in primary prevention. Genetic tests that determine the risk of future AGA in healthy individuals and those that predict response to finasteride treatment are also now available. These can help assess the benefit of starting treatment early and evaluate the likelihood of successful therapy and prevention of AGA-related diseases. However, these tests are not routinely used in clinical practice. [61] Currently, a number of scientific studies linking the genetic links between AGA and other diseases are ongoing. In the future, they will allow us to better understand the nature of the disease, opening up new possibilities for early diagnosis and effective treatment. Although AGA is a condition that affects the scalp, it should not be within the competence of a dermatologist alone. The problem of AGA should not be ignored, but the patient should be looked at from many sides. A holistic approach to the patient is advisable, and if other diseases are suspected due to the presence of AGA, doctors should consider consulting other specialists, primarily an endocrinologist, diabetologist, cardiologist, gynecologist, psychiatrist. Thus, an interdisciplinary approach allows for comprehensive care of the patient, effective treatment, and increased comfort in patients' lives.

Conclusion

Androgenetic alopecia is the most common cause of hair loss in men and women, significantly affecting patients' quality of life. Commonly, AGA is seen as a cosmetic problem, but more and more scientific studies are showing a link to systemic diseases. Individuals with early onset AGA are at higher risk of developing metabolic syndrome, type 2 diabetes and other endocrine disorders. In women, on the other hand, AGA often co-occurs with PCOS, further confirming the link to endocrine disruption. An increased risk of cardiovascular disease has also been observed in people with AGA. These associations indicate that AGA may serve as a potential marker of systemic diseases. It is also necessary to change the clinical approach to AGA. It should be treated not just as an aesthetic defect, but should take into account the potential risk of comorbidities present or diseases that may develop. To this end, physicians should undertake a comprehensive evaluation of the patient, consider screening for other conditions, and include support from other specialists. Unfortunately, the topic of AGA as a biomarker and risk factor for systemic diseases is still poorly understood. Further research is needed to understand in detail the pathomechanisms linking these correlations, early detection and treatment to improve the quality of life of patients with AGA, as well as the development of screening tests and analysis of the age of onset of AGA as a potential risk for systemic diseases.

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