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Metabolic syndrome and associated comorbidities in alopecia

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ABSTRACT

Alopecia, a prevalent condition characterized by hair loss, bears a significant psychological impact on affected individuals. The condition can be classified into two primary categories: scarring (cicatricial) and non-scarring forms. Alopecia is frequently associated with various comorbidities, particularly autoimmune disorders. This review critically examines the existing literature to elucidate the potential relationship between different types of alopecia and metabolic syndrome (MetS), along with MetS-associated comorbidities. MetS is defined by a cluster of conditions, including hypertension, hyperglycaemia, abdominal obesity, and dyslipidaemia, which collectively heighten the risk for cardiovascular diseases (CVD) and diabetes. Furthermore, the shared risk factors, encompassing lifestyle habits and genetic predispositions, suggest a possible bidirectional relationship between these conditions. This underscores the importance of adopting integrated treatment approaches to address the complex interactions between alopecia and MetS. The review highlights the necessity for more comprehensive and diverse cohort studies to enhance our understanding of the interplay between alopecia and MetS.

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Keywords: alopecia, metabolic syndrome, comorbidities, autoimmune disorders, hypertension, dyslipidaemia, hyperglycaemia

INTRODUCTION

Alopecia, commonly known as hair loss, is a medical condition characterized by partial or complete loss of hair on the scalp or other parts of the body. It is a prevalent condition that can affect individuals of various ages and genders. The prevalence of androgenetic alopecia, one of the most common forms, affects approximately 50% of men and women by the age of 50 [1]. Alopecia is associated with a significant psychological burden, often leading to decreased self-esteem, anxiety, depression, and social phobia, which substantially reduce the quality of life for affected individuals [2]. There are several types of alopecia, which can be categorized into scarring (cicatricial) and non-scarring forms. Scarring alopecia leads to permanent hair loss due to the destruction of hair follicles, while non-scarring alopecia usually does not result in permanent follicular damage [3]. Examples of scarring alopecia include conditions such as lichen planopilaris (LPP) and frontal fibrosing alopecia (FFA). In contrast, androgenetic alopecia and alopecia areata are common types of non-scarring alopecia [4]. The pathogenesis of alopecia is complex and multifactorial, involving genetic, hormonal, cytokine, adipokine, oxidative stress, and microbiota-related factors [1, 4]. Genetic predisposition plays a crucial role, particularly in androgenetic alopecia, where the inheritance pattern is polygenic. Hormonal influences, especially androgens like dihydrotestosterone (DHT), are also critical in the development of androgenetic alopecia [5]. Cytokines and adipokines, such as tumour necrosis factor-alpha (TNF-alpha) and leptin, are implicated in inflammatory pathways that contribute to various forms of alopecia [6]. Oxidative stress and alterations in the scalp microbiota are emerging areas of research, suggesting their involvement in the pathogenesis of alopecia [4]. Autoimmune conditions can trigger systemic inflammation, which may contribute to the development of comorbidities [7]. The interrelationship between dermatological conditions and systemic diseases underscores the importance of a holistic approach to patient management.

Metabolic syndrome (MetS) is a cluster of conditions, including increased blood pressure, high blood sugar levels, excess body fat around the waist, and abnormal cholesterol levels. Criteria for the diagnosis of MetS are shown

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Table 1. Criteria for the diagnosis of metabolic syndrome according to the position of the Polish Society of Hypertension, the Polish Society of Obesity, the Polish Diabetes Society, the Polish Society of Endocrinology, the Polish Society of Family Medicine, the Polish Society of Clinical Nutrition, the Prevention and Epidemiology Section of the Polish Cardiac Society, the "Club 30" of the Polish Cardiac Society, and the Metabolic and Bariatric Surgery Section of the Polish Association of Surgeons [8]

Basic criteria for the diagnosis of metabolic syndrome		
Waist circumference: Male ≥ 102 cm Female ≥ 88 cm	BMI ≥ 30 kg/m^2	
Additional criteria (at least 2 of 3)		
Prediabetes or diabetes	**Elevated non-HDL cholesterol**	**High normal or hypertensive blood pressure**
Fasting glucose level ≥ 100 mg/dL or ≥ 140 mg/dL 120 minutes after an oral glucose tolerance test, or haemoglobin A1c ≥ 5.7%, or use of hypoglycaemic medication	Non-HDL cholesterol level ≥ 130 mg/dL, or use of lipid-lowering medication	Blood pressure ≥ 130 and/or 85 mmHg (office measurement), or blood pressure ≥ 130 and/or 80 mmHg (home measurement), or use of antihypertensive medication

BMI — body mass index; non-HDL — non-high-density lipoprotein cholesterol

in Table 1 [8]. According to the latest definition, MetS is widely recognized as a significant risk factor for cardiovascular disease (CVD) and CVD-related mortality [9]. The syndrome is thought to be driven by insulin resistance and central obesity, leading to a pro-inflammatory and pro-thrombotic state that predisposes individuals to CVD [10].

Understanding the intricate relationship between alopecia and MetS, along with their associated comorbidities, is essential for comprehensive patient management. Given the significant overlap between the pathophysiological mechanisms underlying these conditions, investigating their connections can provide insights into more effective treatment strategies and improve patient outcomes. This study aims to explore these connections and their implications for patient care, providing a detailed analysis of the current evidence on the interplay between MetS and alopecia.

ANDROGENETIC ALOPECIA (AGA)

AGA — definition, pathogenesis and risk factors

Androgenetic alopecia (AGA) is the most common form of hair loss and affects up to 70% of men and 30% of women in the course of their lives [11, 12]. In men, it typically involves the frontal, temple, and vertex areas, whereas women experience diffuse thinning over the crown. AGA results from both androgen hyperactivity and genetic susceptibility [4, 13]. There are two main key points in AGA pathogenesis — the shortened anagen phase (growth phase) of scalp hair, which causes the hair to be thinner and shorter in the process of follicular miniaturization; the other one is increase of interval between the exogen phase and anagen phase that results in increased number of "empty" hair follicles. Androgens, particularly DHT, play a key role in the pathogenesis of AGA. DHT, a potent metabolite of testosterone, binds strongly to androgen receptors in hair follicle dermal papilla cells, which subsequently inhibits adenyl cyclase and activates genes that transform large terminal hair follicles into smaller ones with a shorter growth phase (anagen), resulting in follicular miniaturization [4, 14].

Multiple conditions may result in an increased production of DHT and other androgens. The most common conditions influencing these hormonal imbalances are polycystic ovary syndrome (PCOS) in which hyperandrogenism can contribute to female pattern hair loss; and menopause which causes a decline in oestrogen and increases the relative effect of androgens, exacerbating hair thinning [15]. Another factor that plays a significant role in hair follicle miniaturization is adipokines and pro-inflammatory cytokines produced by adipose tissue. They influence hair follicle biology through regulatory roles in metabolism and inflammation. Leptin promotes hair growth by stimulating the anagen phase and inhibiting apoptosis whereas adiponectin exhibits anti-inflammatory properties and supports hair follicle health [16, 17]. Patients with MetS have been shown to have lower levels of adiponectin and higher levels of pro-inflammatory cytokines, such as tumour necrosis factor-alpha (TNF-alfa) and interleukin-6 (IL-6), that can contribute to chronic inflammation affecting hair follicles [18]. The interaction between androgens and adipokines creates a feedback loop influencing hair follicle health, exacerbated by conditions like insulin resistance and obesity. Additionally, oxidative stress, resulting from an imbalance between reactive oxygen species (ROS) production

and antioxidant defences, damages hair follicle cells, particularly in the dermal papilla [19]. Elevated ROS levels, exacerbated by androgens like DHT, lead to hair follicle miniaturization and anagen phase shortening. Oxidative stress also stimulates pro-inflammatory cytokine production, perpetuating inflammation and hair loss. Moreover, the scalp microbiome, comprising diverse bacterial and fungal species, is vital for scalp health and hair growth regulation [20]. Dysbiosis, or microbial imbalance, can lead to inflammation and oxidative stress, worsening AGA. Certain microbes, like *Malassezia*, promote a pro-inflammatory environment that harms hair follicles. Androgen-driven sebum production further supports these microbes, exacerbating dysbiosis and inflammation. Dysregulation of both skin and gut microbiota is often present in patients with metabolic syndrome and associated conditions, such as diabetes or obesity. This may influence the gut-skin axis and result in exacerbation of skin disease, as it is seen in atopic dermatitis or psoriasis [21, 22].

ASSOCIATION OF AGA WITH METS

MetS is found more frequently in patients with AGA according to several studies [23–28]. Female sex, early-onset AGA, and African ethnicity were found to have the highest correlation with an increased risk of MetS [14]. One study found patients with early-onset (20–36 years) AGA to have an approximately four times increased frequency of MetS [27]. In a large, population-based case-control study comparing patients with early-onset AGA to patients with late-onset AGA, they found that patients with earl-onset AGA are significantly more likely to have MetS.

However, no statistically relevant differences between the groups in body mass index (BMI), waist circumference and blood parameters were noticed. Moreover, patients with early-onset AGA had lower high-density lipoprotein (HDL) levels [28]. A meta-analysis by Qiu et al. [29] analysed 19 large studies that found a link between AGA and MetS in Europe, Asia, Africa, and South America. They found that patients with AGA have overall poorer metabolic profiles, such as body mass index, waist circumference, fasting glucose, blood lipids, and blood pressure, leading to MetS [29]. Similar results were obtained from case-control studies on 100 patients with AGA — patients were found to have statistically significant differences compared to controls in terms of higher body weight, height, waist circumference, body mass index, systolic and diastolic blood pressure, fasting glucose, triglycerides, lower HDL cholesterol, elevated fasting insulin ($p = 0.02$), and an increased homeostasis model assessment of insulin resistance (HOMA-IR) ($p < 0.001$) [24]. Males seem to be more affected — a case-control study on 77 participants with AGA and 77 healthy controls found that 60% of male patients (compared to 12.5% in the control group) and 48.6% of female patients (compared to 8.1% in the control group) met the criteria for MetS diagnosis [30].

There is a strong link between dyslipidaemia and AGA, with higher serum levels of total cholesterol, low-density lipoprotein (LDL) and triglycerides but lower HDL levels among patients with AGA. According to the studies, there is a need for screening metabolism-related indicators in patients diagnosed with AGA. A study from Nigeria found that AGA subjects had higher systolic blood pressure (SBP) compared to the controls (125.96 ± 17.91 mmHg *vs.* 121.36 ± 21.49 mmHg); moreover, elevated SBP also positively correlated with AGA severity [27]. The study found MetS significantly more prevalent in patients with AGA and that AGA was significantly associated with traditional CVD risk factors such as dyslipidaemia, sedentary lifestyle, and alcohol intake. AGA has also been linked with coronary artery disease, polycystic ovary syndrome, and Cushing syndrome, as well as several nutritional deficiencies [14].

Mechanisms underlying the link between MetS and AGA are yet to be elucidated, however some studies suggest the role of various molecules. For example, alarin, a 25-amino acid peptide, is significantly elevated in patients with both AGA and MetS. Serum alarin levels were positively correlated with the severity and duration of AGA, as well as with metabolic markers such as triglycerides, cholesterol, LDL, very low-density lipoprotein (VLDL), fasting glucose, and BMI. These findings suggest that alarin may play a role in both glucose homeostasis and the progression of AGA. [31]. Another study showed significantly higher serum regulated on activation, normal T-cell expressed and secreted (RANTES) in patients with AGA when compared to the control group. This adipocyte-derived pro-inflammatory chemokine is found to be positively correlated with BMI, FBG, TC, and LDL-c levels in AGA patients with MetS [32]. One study suggests a possible role of a pro-inflammatory glycoprotein YKL-40 in the link between AGA and MetS. YKL-40 is expressed by various inflammatory cells and is involved in the pathogenesis of many diseases, such as obesity, atherosclerosis, diabetes or inflammatory bowel disease. The study found YKL-40 to be a marker of an early-onset AGA and also a predictor of early MetS development and severity in patients with early-onset AGA [33].

ALOPECIA AREATA (AA)

AA — definition, pathogenesis and risk factors

Alopecia areata (AA) is an autoimmune condition that attacks the hair follicles, causing nonscarring hair loss. The AA incidence is estimated between 13.6–26 cases per 100,000 person-years [34, 35]. A 20-year retrospective analysis found that the lifetime risk of alopecia areata

is around 2.1% [36]. In its early phase, AA typically presents as patchy hair loss, but it can progress to the loss of all scalp hair (alopecia totalis), hair loss in a band along the sides and back of the head (alopecia ophiasis), or the loss of all body hair (alopecia universalis) [37]. Approximately 14–25% of patients will progress to alopecia totalis or alopecia universalis [35, 37, 38].

Researchers suggest that AA is caused by an autoimmune reaction to the hair follicles due to genetic and environmental factors. Central to the autoimmune process in AA are T cells, especially CD8+ cytotoxic T lymphocytes [39]. These cells infiltrate the hair follicle's peribulbar region, leading to follicular damage. In AA, there is evidence of dysfunction or reduction in regulatory T cells (Tregs), which generally help to suppress excessive immune responses [7]. This issue may contribute to the unchecked immune response against hair follicles. The presence of CD8+ T cells leads to the production of inflammatory cytokines such as interferon-gamma (IFN-γ), interleukin-2 (IL-2), and interleukin-15 (IL-15). IFN-γ and IL-2 can lead to the infiltration of CD8+, CD4+, and other inflammatory cells into the immune privilege zone. Additionally, tumour necrosis factor-alpha (TNF-α) and other inflammatory mediators further contribute to the destruction of hair follicles, exacerbating the condition [40]. Genetic factors play a major role in predisposing individuals to AA. In a recent meta-analysis, the human leukocyte antigen-DR (HLA-DR) was identified as the most significant risk factor for AA. Another genetic factor identified in the study was BCL2-like protein 11 (BIM), which may influence the pathogenesis by regulating the apoptosis of immune cells, potentially leading to the uncontrolled destruction of hair follicles [41]. The most common environmental risk factors for the development of AA identified in the literature are smoking, emotional stress, and sleep disorders. Moreover, these factors also contribute to the development of MetS. Smoking promotes inflammation and oxidative stress, disrupting metabolic processes and leading to insulin resistance and dyslipidaemia. Emotional stress is closely linked to MetS through its impact on inflammatory processes. Additionally, sleep disorders can disrupt hormonal regulation, resulting in increased appetite, weight gain, and insulin resistance [42–44]. Current smokers compared to non-smokers have a 1.88 (1.22–2.88) times higher risk of developing alopecia areata [42]. Moreover, people who have smoked for more than 10 years or who smoke more than five cigarettes a day have an even higher risk of developing alopecia areata [42, 45]. Furthermore, people with sleep disorders, especially those under 45 years old, also exhibit a higher risk of developing alopecia areata [43, 46].

Association of AA with MetS

The meta-analysis confirms that overall prevalence rates of MetS in AA patients are not significantly higher, though there are notable associations with individual components of the syndrome, such as hypertension, diabetes, and obesity, particularly in pediatric and late-onset cases [47]. The prevalence of MetS among patients with alopecia areata was not statistically significantly higher in any of the studies. However, studies exhibit a lot of limitations such as small sample sizes ranging from 35 patients in the study by Incel-Uysal et al. to 106 patients in the study by Singdia et al. [48–51]. However, in the largest study, it was reported that MetS cases occurred at a younger age in patients with AA compared to the control group [49]. There was only one small-sample study (51 patients) that found an association between AA and insulin resistance. In this study, lipid profiles were found to be similar between the patient and control groups [52]. Interestingly, in Singdia et al. [49] study it was noted that only the level of HDL was significantly lower in the AA group compared to the control group [49].

A large 11-year study from the USA on 3,568 participants, however, reported a high prevalence of hyperlipidaemia (24.5%) and hypertension (21.9%) among patients with AA [53]. A recent study by Conic et al. [54], which included 33,130 patients with AA, showed a significantly increased prevalence of metabolic diseases including hypertension (28% *vs.* 17.5% in controls), hyperlipidaemia (19.8% *vs.* 6.6%) obesity (18.1% *vs.* 3.0%) diabetes mellitus (11.4% *vs.* 7.4%) and MetS (1.4% *vs.* 0.3%) [54]. In a smaller cohort study by Andersen et al. [55] involving 1,494 adult patients, there was no evidence found linking higher body mass with the presence of AA [55]. In a study by Lee et al. [56] comparing patients with late-onset and early-onset disease, it was observed that the late-onset group had a significantly higher prevalence of hypertension and diabetes. The study included a total of 871 patients, with 98 patients (11.3%) in the early-onset group and 773 patients (88.7%) in the late-onset group of AA [56]. The study results for the paediatric population look somewhat different. Recent research by Conic et al. [57] indicated a significant association between AA and MetS (0.3% *vs.* 0.04% controls), highlighting an increased prevalence of hyperlipidaemia (1.4% *vs.* 0.2% controls), diabetes (1.1% *vs.* 0.6% controls), and obesity (5.7% *vs.* 1.1% controls) [57]. One of the conditions that is part of MetS, obesity, was identified as the third most common comorbidity in studies examining paediatric populations with AA.

Family history of AA and autoimmune diseases was more frequently observed in the AA group compared to healthy controls [51]. Among autoimmune conditions associated with AA, thyroid disorders showed the highest

frequency across multiple studies, ranging from 8.9% to 14.6% [58–61]. This highlights the importance of screening for thyroid dysfunction in AA patients, even in the absence of specific clinical symptoms related to thyroid abnormalities noted in the cohort study [61]. The most common comorbidity in children is atopic dermatitis with a prevalence of 17.4–34.4% [57, 62].

CICATRITIAL ALOPECIA (CA)

Definition, pathogenesis and risk factors of CA

Cicatricial alopecias (CAs) are conditions that lead to the irreversible destruction of hair follicles and their replacement by fibrous structures resulting in scarring [63]. Based on the mechanisms and target structures of the inflammation CAs are classified as either primary cicatricial alopecia (PCA) or secondary cicatricial alopecia (SCA) [64]. In PCA the inflammatory process directly destructs hair follicles which are the primary target of the inflammation while in the course of SCA, the destruction of hair follicles is caused by the inflammatory process or mechanical damage of surrounding tissue which consequently leads to the destruction of hair follicles [64]. According to the North American Hair Research Society (NAHRS), PCA was divided into 3 groups based on the most prominent inflammatory cells infiltrating hair follicles: neutrophilic, lymphocytic and mixed infiltrate [63]. Alopecias occurring in the course of FFA, discoid lupus erythematosus (DLE) and LPP are classified as PCA neutrophilic [63]. FFA is currently the most common form of CA worldwide. It most often affects postmenopausal women and presents with receding hairlines and bilateral eyebrow loss. FFA has been described in families and genes associated with FFA have been discovered, but the aetiology of the disease remains controversial [65]. DLE is the most common form of cutaneous lupus erythematosus. The disease more often affects women and ultimately leads to scarring and skin atrophy. The etiopathogenesis of the disease is complex and not fully understood. Genetic and immunological factors, disruption of the cytokine cascade, apoptosis, necrosis, dendritic cells, T and B lymphocytes, and vascular changes play a role in the development of the disease [66]. LPP is an idiopathic T-cell-mediated inflammatory disease affecting the scalp. It is estimated to affect 13.4 per 100,000 in the general population [67, 68]. In the context of the association of CA with MetS, an important form of alopecia is central centrifugal cicatricial alopecia (CCCA), which is classified as lymphocytic PCA. This form of alopecia is characteristic of African American women and most often affects women in the 2nd and 3rd decades of life [63]. According to estimates, it may affect as many as 15% of black women [69]. Hair loss begins at the crown and vertex of the head and progresses over time. The etiology of CCCA remains unknown, although infectious, genetic, and autoimmune factors are believed to underlie the disease [69].

Association of CA with MetS

Although the available literature on scarring alopecia and its correlation with MetS and comorbidities is limited, some studies show an association between these diseases. A case-control study of 62 patients with DLE showed a higher incidence of MetS than in the control group, with simultaneous higher HDL reduction and hyperglycaemia [66]. It should be noted that patients who had taken medications that could affect metabolic parameters (steroids, retinoid acid, cyclosporine, and methotrexate) in the past were excluded from the analysis. A retrospective cohort study by Ayandibu and Bergfeld [70] of black women with CCCA showed an increased prevalence of risk factors for MetS and related cardiovascular disease. In the study, 38.8% of participants met the criteria for MetS, which is significantly higher than for the general population [70]. Patients suffering from LPP did not show a higher risk of developing MetS compared to the control group [67].

Furthermore, there is information in the available medical literature about the correlation between poorly controlled diabetes and the severity of CCCA symptoms. The mean severity of CCCA in the study group (with diabetes/prediabetes) was 3.16 *vs.* 2.57 in the control group [71]. The authors of the publication also conducted another study showing a 4 times higher risk of developing diabetes in patients with CCCA than in the control group, which may be the result of similar pathomechanisms underlying the development of both diseases [72]. In addition, Colleen's [73] cohort study of African American and Caucasian women with two distinct types of CA (CCCA and FFA) showed that these women were at increased risk for hypertension compared with controls. This may be due to the overlapping mechanisms of these two disease entities. Dysfunction of the renin-angiotensin-aldosterone system (RAAS), which plays a key role in the pathogenesis of hypertension, also contributes to the occurrence of fibrosis and thus scarring hair loss. The cohort study conducted in 2011 shows that women and men with LP have elevated lipid levels compared to the control group [74]. This allows patients with LP to introduce cardiovascular disease prophylaxis early enough. Seven years later, another study of lipid levels in patients with LP was conducted, which did not show any significant differences in the lipid profiles of patients from both groups. This could have been due to study limitations, such as the cross-section of patients and the specificity of the facility where the study was conducted [75].

FOLLICULITIS DECALVANS (FD)

Definition, pathogenesis and risk factors of FD

Folliculitis decalvans (FD) is a rare form of neutrophilic primary cicatricial alopecia. The percentage of FD accounts for approximately 11% of all cases of primary cicatricial alopecia [76, 77]. FD is significantly more common in men than in women [78, 79]. Clinically, FD manifests as scarring alopecia with patches of hair loss, often accompanied by follicular pustules, crusting, and the presence of tufted hairs. The most commonly affected area is the vertex [80]. The pathogenesis of FD remains unclear, but new studies suggest a role of immunology. The early FD lesions are marked by the infiltration of activated T-helper cells. IL-8 and ICAM-1 contribute to the recruitment of neutrophils, while b-FGF and TGF-β are crucial mediators in the development of fibrosis during the late stages of the disease [81]. *Staphylococcus aureus* (*S. aureus*) appears to play a significant role in the pathogenesis of FD, as it is commonly isolated in nearly all patients with this disease. Clinical improvement in most cases was associated with the disappearance of *S. aureus* [82].

Association of FD with MetS

There are few studies on FD and its comorbidities. The largest study, conducted by Lyakhovitsky et al. [83] between 2010 and 2020, included 192 patients and found that 23.4% of them were obese. However, obesity and smoking showed no correlation with disease severity. Metabolic syndrome was present in 21% of patients with mild to moderate FD and in 14% of those with severe FD, with a p-value of 0.98.

In a smaller study involving 60 patients, the results were similar. The associated comorbidities included hypercholesterolemia in 8 patients (13.3%), arterial hypertension in 7 patients (11%), and obesity in 2 patients (3.3%). No comorbidities were found in 39 patients (65%) [84].

This suggests that the prevalence of metabolic syndrome and its components may be comparable to that of the general population of a similar age.

CONCLUSIONS

The biggest challenge in this review was the lack of large studies focusing on MetS and alopecia. Existing studies were of poor quality, often relying on small and inadequately selected samples, and frequently focusing on patients within a single racial group. There are numerous studies examining the most common comorbidities, but autoimmune diseases are predominant in these. In some studies, researchers focus only on certain components of MetS, not on MetS as a whole. Table 2 summarises the clinical data regarding the prevalence of metabolic syndrome and associated comorbidities with different types of alopecia.

Table 2. Table summarising the clinical data regarding the prevalence of metabolic syndrome and associated comorbidities with different types of alopecia, extracted from the cited literature

Key features	AGA	AA	CA
Main pathogenesis key points	Hair shedding and follicular miniaturization due to androgen activity	Autoimmune reaction due to genetic and environmental factors	Either primary autoimmune reaction (neutrophilic, lymphocytic or both); or secondary to trauma or injury
MetS	11.9–53% *vs.* 3–17% in controls	1.4% *vs.* 0.3% in controls	DLE: 48.3% *vs.* 24.4% in controls LPP: no significant differences FD: 14–21%
Obesity	32–37.8% *vs.* 12.5–18.8% in controls	18.1% *vs.* 3.0% in controls	FD: 23.4%
Hypertension	Significantly higher, correlates with severity	28% *vs.* 17.5% in controls	FD: 11%
CVD	Nd	Coronary artery disease: 5.5% *vs.* 1.8% in controls; Stroke: 0.45% *vs.* 0.31% in controls	Nd
Dyslipidaemia	Hypertriglyceridemia: no significant differences Reduced HDL: 25.9–44.23% *vs.* 9.4–24.23% in controls	19.8% *vs.* 6.6% in controls	DLE: hypertriglyceridemia: 48.3% *vs.* 24.4% in controls Reduced HDL: 61.7% *vs.* 23.2% in controls LPP: no significant differences FD: 13,3%
Diabetes/IR	Diabetes: no significant differences IR: 35% *vs.* 19% in controls Elevated plasma glucose: 47.1% *vs.* 43.5%	Diabetes: 11.4% *vs.* 7.4% in controls	Nd

AA — alopecia areata; AGA — androgenetic alopecia; CA — cicatricial alopecia; CVD — cardiovascular diseases; DLE — discoid lupus erythematosus; FD — folliculitis decalvans; HDL — high-density lipoprotein; IR — insulin resistance; LPP — lichen planopilaris; MetS — metabolic syndrome; Nd — no data

The situation is much different in the case of AGA, which has the highest association with MetS. Another challenge was the diversity of study groups and the different results within each group. This prevents drawing general conclusions. Standardizing all studies without understanding the specific groups that were examined would give us a false picture of the situation. AA shows a connection with MetS in several studies, particularly in pediatric patients and those with late-onset disease. Likewise, AGA is associated with MetS, but differences in studies arise due to variations in the populations examined and other factors such as genetic and hormonal changes. CCCA shows an increased risk of MetS, especially among black women. This result may be linked to a higher prevalence of obesity (waist circumference), insulin resistance, and blood pressure in Black individuals [85]. The National Health and Nutrition Examination Survey indicates that Black women are more likely than White women to have MetS [86]. This highlights the need for new cohort research and the development of new tactics and strategies with screening tests, especially including diverse populations among patients with various types of alopecia, particularly those groups found to have a higher risk of developing MetS.

Early diagnosis of MetS in alopecia patients is crucial because research indicates that treating components of MetS can lead to improvements in various types of alopecia. Comprehensive management of MetS should be emphasized not only for overall health but also for its potential benefits on hair growth in patients suffering from various types of alopecia. Among these types of alopecia, AA and AGA have distinct etiologies: AA is primarily an autoimmune disease, while AGA results from hormonal and genetic processes. However, lifestyle factors also play a role in both conditions. Similarly, in MetS, risk factors include smoking, a sedentary lifestyle, sleep disorders, chronic stress, and diets high in refined sugars, unhealthy fats, and processed foods, along with non-modifiable risk factors such as age, genetic factors, and hormonal changes [87, 88]. It can be observed that the risk factors for MetS and various types of alopecia overlap. This might explain the frequent co-occurrence of these conditions, especially in the case of AGA. Not only are the risk factors in alopecia and MetS similar, but some dysregulations occur in both disorders. Intestinal dysbiosis, which is present in AGA, is also found in obesity and type 2 diabetes [89, 90]. Therefore, it can be concluded that in this case, one disease may drive the other. One can observe it in other skin diseases for example psoriasis and atopic dermatitis. The differences in comorbidities between patients and the control group may be linked to the side effects of alopecia treatments. The most common treatment for both CCA and AA is corticosteroids (administered via injection or topically, and less commonly orally). Among the side effects of corticosteroid therapy, all components of the MetS can be found. Corticosteroids are a leading cause of drug-induced diabetes mellitus and also increase insulin resistance in patients with diabetes mellitus. Additionally, corticosteroid use is associated with hypertension, obesity, and hyperlipidaemia, although evidence on these associations is conflicting [91].

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