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AUTOIMMUNE CICATRICIAL ALOPECIA AND THE ROLE OF TRICHOSCOPY IN ITS DIAGNOSIS: A LITERATURE REVIEW

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ABSTRACT

Cicatricial alopecia represents a challenge in modern medicine. It not only results in permanent hair loss but can also be a symptom of other, more serious health conditions. It is important to note that cicatricial alopecia often co-occurs with other pathologies such as lichen planopilaris, sarcoidosis, systemic lupus erythematosus, and scleroderma. In the course of the above-mentioned diseases, alopecia has specific trichoscopic features, that is why it is so important to use this method during diagnostic procedures.

Aims: The aim of this paper is to present the latest knowledge on alopecia in autoimmune diseases, particularly in the course of sarcoidosis, lupus erythematosus, lichen planopilaris and scleroderma.

Methods: A literature review of recent scientific articles from PubMed, Google Scholar, and National Library databases was performed.

Results: Sarcoidosis of the scalp is very rare, primarily in African-American women. It requires differentiation from lupus erythematosus or lichen planus made possible by trichoscopy and histopathological examination. Hair loss in lupus can manifest in various forms, complicating diagnosis, and requires a comprehensive evaluation to differentiate it from other hair loss conditions. Lichen planopilaris is a complex condition with various presentations requiring early diagnosis, accurate classification, and trichoscopy for optimal management. Alopecia can also be one of the first symptoms of scleroderma. Scientist suggest considering scalp morphea as a differential diagnosis of mono-focal scarring alopecia involving the scalp.

Conclusions: Prompt diagnosis and treatment are key to a better outcome and preventing further health issues.

Keywords: cicatricial alopecia, scleroderma, lichen planopilaris, lupus erythematosus, sarcoidosis, trichoscopy

INTRODUCTION

Cicatricial alopecia (CA) refers to a group of diseases characterized by the irreversible loss of hair due to the replacement of hair follicles by fibrous connective tissue and hyalinized collagen. Primary cicatricial alopecia involves direct involvement of the hair follicle, while secondary cicatricial alopecia occurs when the hair follicle is damaged by a disease process in the dermis. According to the North American Hair Research Society (2001) classification, primary cicatricial alopecia is classified based on the predominant inflammatory infiltrate seen on histopathology. Subtypes include: lymphocytic predominant, neutrophilic predominant, mixed pattern, and unclassified cases [1]. CA represents 5% of all cases of hair loss [2].

An autoimmune disease is a condition characterized by the immune system incorrectly targeting the body's

own cells and tissues. These diseases can be classified as primary, where the immune system initiates an attack against self-antigens, or secondary, where the autoimmune response is a consequence of another underlying medical condition [1].

Trichoscopy is a non-invasive method of visualizing hair shafts and the scalp using a video dermatoscope, allowing for differentiation of cicatricial alopecia [3]. Prior to commencing any treatment, an accurate and precise differential diagnosis must be established [4].

AIM OF THE WORK

The purpose of this paper is to present the latest knowledge on alopecia in autoimmune diseases, particularly in the course of sarcoidosis, lupus erythematosus, lichen sclerosus, and atrophic lichen and scleroderma. And also to highlight the role of trichoscopy in the diagnosis and monitoring of alopecia in these diseases.

METHODOLOGY

A review of the recent literature on alopecia in autoimmune diseases was performed. Articles were selected on alopecia in sarcoidosis, lupus erythematosus, lichen sclerosus, atrophic lichen, and scleroderma. Articles were searched for in PubMed, Google Scholar, and the National Library database using the phrases: "alopecia", "alopecia in scleroderma", "alopecia in lupus", "alopecia in sarcoidosis", "alopecia in lichen", "scleroderma", "sarcoidosis", "lichen sclerosus", "atrophic lichen". Inclusion criteria were publication of the article from 2008 to 2024, English or Polish language of the article and description of alopecia in a specific disease. Most of the articles are not older than 6 years.

RESULTS AND DISCUSSION

CHARACTERISTICS OF SCARRING ALOPECIA

Sarcoidosis

Sarcoidosis is a multi-system disease characterized by non-caseating granulomas, often called naked granulomas, which can affect any organ [5,6,7]. It most commonly involves lymph nodes, liver, lungs, spleen, skin, bones, and eyes [5] . Skin involvement occurs in 20-30% of cases and is usually the first symptom of the disease [7]. Skin changes can be divided into specific and nonspecific types. Specific changes have typical histopathological findings associated with the disease but can vary significantly regarding clinical morphology and location. The most common cutaneous manifestation is papular sarcoidosis, which includes numerous non-scaled papules often located on the nasolabial folds and eyelids, but it can also involve other areas, such as the upper back [6]. The etiology of sarcoidosis is unknown [5]. Histopathologically, classic sarcoid granulomas are characterized by non-necrotizing centers composed of macrophages, epithelioid cells, multinucleated giant cells, and lymphocytes. These central areas are surrounded by CD8 and CD4 positive T lymphocytes and fibrosis. The extent of the lymphocytic infiltrate and surrounding fibrosis can vary depending on the patient and the duration of the disease [7].

Scalp sarcoidosis is extremely rare and difficult to diagnose. It primarily affects African American women aged 23 to 78 years. It is usually associated with other systemic and cutaneous symptoms, such as involvement of the neck, trunk, limbs, pulmonary tissue, and lymph nodes [5,8,9]. It is marked by erythematous, scaly, and indurated plaques, which can lead to follicular fibrosis and destruction, ultimately causing alopecia [9]. In most cases, it is associated with scarring alopecia, although cases of non-scarring alopecia have also been described [6].

Diagnosis

During the diagnosis of scalp sarcoidosis, it is important to differentiate it from other causes of scarring and non-scarring alopecia. Clinical symptoms may resemble those seen in lupus erythematosus or follicular lichen planus [6]. Therefore, the basis for diagnosis is histopathological and trichoscopic examination. Histopathological analysis reveals sarcoidal granulomas in the dermis, composed of epithelioid cells, a few multinucleated giant cells, and a dense infiltrate of lymphocytes. In trichoscopy, there is a loss of follicular ostia, reddish-orange discoloration of the skin, and capillary telangiectasias due to vasodilation in the papillary dermis. Sarcoidal hairless patches are typically described as red, well-defined, and itchy. In scalp sarcoidosis, along with the dermoscopic features observed on the skin, the presence of dilated ostia, particularly at the edges of the alopecic area and larger than the characteristic yellow dots seen in alopecia areata, and dystrophic hairs which are non-specific and may appear in both scarring and non-scarring alopecia can be identified. These features are crucial as they indicate an active disease state and provide insights into the disease's progression and prognosis. Conversely, the loss of follicular ostia signifies scarring, which shows permanent hair loss. Therefore, trichoscopy provides important diagnostic clues in the case of scalp sarcoidosis and facilitates prompt diagnosis and appropriate therapeutic intervention.

However, histopathological examination is essential to confirm the diagnosis [7].

Patients with alopecia due to scalp sarcoidosis should be evaluated for additional cutaneous manifestations and undergo a thorough assessment for systemic sarcoidosis, as most reported cases have shown systemic involvement, especially in the thoracic region [5].

Lupus erythematosus

Lupus erythematosus (LE) is a chronic and multi-systemic disease and has clinical and serological characteristics. The joints, skin, kidneys, lungs and nervous system are most commonly affected in lupus. A common characteristic is the ANA antibody. More than 80% of patients experience dermatological changes at any stage of the disease. In lupus, hair loss is observed in more than half of patients [10]. Alopecia may be the first symptom that causes a patient visit a doctor. In lupus, alopecia may affect the scalp, beard, eyelashes, eyebrows or body hair [11]. Several patterns of hair loss may occur in lupus and the exact cause is often unclear. It's essential to determine if hair loss in lupus is related to the underlying lupus condition or due to a separate hair loss disorder. Alopecia with histological features consistent with lupus is considered lupus-specific and can manifest as diffuse, patchy, or discoid lupus erythematosus (DLE), which often leads to scarring [10]. It may be limited to skin lesions (cutaneous lupus erythematosus - CLE) or involve multiple organs (systemic lupus erythematosus - SLE). A variant of chronic cutaneous lupus erythematosus is discoid lupus erythematosus (DLE) [2]. The cause of alopecia may be both the activity of the disease itself and the treatment used [11]. Characteristic of SLE is a fluctuating course with periods of exacerbations and remissions. Individuals with a genetic predisposition can develop SLE when exposed to environmental factors such as ultraviolet radiation, sex hormones, infections, or chemicals, triggering autoimmune reactions with autoantibody production and chronic inflammation. Women are more likely to develop SLE than men, with a female-to-male ratio of approximately 9:1, and most commonly affect individuals aged 14-64 [14]. In SLE, hair loss has been reported in 85% of patients, but the occurrence of scarring is characteristic of CLE [2].

Discoid lupus erythematosus

Discoid lupus erythematosus (DLE) is a chronic, scarring skin condition sensitive to sunlight, often leading to tissue thinning. While DLE can occur in patients with systemic lupus erythematosus (SLE), most individuals with DLE do not develop systemic symptoms. DLE typically affects the skin below and above the neck, including the scalp. High levels of antinuclear antibodies and lymphocytopenia are common laboratory findings in SLE but less frequent in DLE [15].

In the early stages, the lesion is well-defined, red, and scaly with follicular hyperkeratosis within the surrounding skin. Over time, the lesion expands outward, forming coin-shaped, ivory-colored patches that are flat, thin, smooth, and have plugged hair follicles with adherent scale and a raised, active border. Telangiectasias are often present [11]. The acute phase of DLE is accompanied by a burning sensation, skin tension and itching. The pull test for anagen hair may be positive. In trichoscopy, the characteristic feature of DLE is "red dots" or the presence of blue-gray dots [3].

Diagnosis

When evaluating scalp involvement in DLE, the entire scalp should be examined to assess the location and type of hair loss, as well as to identify any extracranial or systemic symptoms. Diagnosis is confirmed through scalp biopsy and direct immunofluorescence, which helps determine inflammation severity and differentiate scalp DLE from other primary cicatricial alopecias. The main histopathological features include follicular hyperkeratosis, epidermal atrophy, superficial and deep lymphocytic infiltrates around the appendages, thickening basement membrane and vacuolar degeneration of basal layer cells at the dermo-epidermal junction. It is also often found mucin deposits in the papillary layer of the skin and fibrosis. Luminance pattern of granular C3 and IgG deposits (less often IgM) within the dermal-epidermal junction or at the junction of the hair follicle epithelium and dermis is typical in active lesions [11].

Lesions are characteristically round or disc-shaped with adherent scales and follicular plugging. Patients may experience scalp tenderness and itching. Distinguishing DLE from other cicatricial alopecias is aided by the presence of erythema, scaling, and pigmentation changes in DLE. Additionally, in DLE, changes usually occur at the center of the hair loss patch rather than at its edges. With chronic disease progression, ivory-colored, smooth, and sclerotic plaques develop [3].

Lichen planopilaris

Lichen planopilaris (LPP) is a follicular variant of lichen planus and is the most common cause of primary cicatricial alopecia. Various subtypes are recognized clinically: classic lichen planopilaris (also known as follicular lichen planus), frontal fibrosing alopecia (FFA), Graham-Little-Piccardi-Lassueur syndrome (GLPLS), and recently described – fibrosing alopecia in a pattern distribution, which is marked by progressive scarring hair loss that follows the pattern of female pattern hair loss [12]. Classic LPP typically manifests as

cicatricial alopecia, primarily affecting the parietal region and vertex. FFA is mainly described in postmenopausal women, however, premenopausal women and men. This condition is characterized by a progressive, symmetrical recession of the frontotemporal or frontal hairline often linked with eyebrow loss. Moreno et al. proposed a clinically relevant classification system for FFA, stratifying patients into three distinct patterns with varying prognoses. The pattern I ("linear") manifests as a uniform band of frontal hairline recession without a decrease in hair density behind the hairline. This most common pattern carries an intermediate prognosis. Pattern II ("diffuse") presents with diffuse or zigzag band-like alopecia affecting the frontal hairline and significant hair loss density behind it. This second most common pattern has the worst prognosis. Finally, Pattern III ("pseudo-fringe sign") is characterized by the preservation of hairs along the frontotemporal hairline and boasts the best prognosis, despite being the least frequent presentation [13,16]. GLPLS, also known as Graham-Little syndrome, is distinguished by a triad of cicatricial alopecia presenting in patches on the scalp, non-cicatricial hair loss in the axillary and pubic regions, and a lichenoid follicular eruption [12].

The exact etiology of lichen planopilaris remains unknown, though it is hypothesized to be a cytotoxic autoimmune response targeting an unidentified hair follicle antigen. The condition has minimal genetic or drug-induced associations, with pembrolizumab being a rare exception. Several factors may predispose individuals to lichen planopilaris, including sunlight exposure, hepatitis C infection, and topical agents such as amla oil, mustard oil, hair dyes, and fragrances [17].

Lichen planopilaris manifests as smooth, white patches of alopecia with absent follicular openings. Erythema and scaling surround affected follicles at the patch periphery, sometimes exhibiting a spiny texture upon palpation. Loose hairs are readily extracted. The disease typically presents as a multifocal process, with small patches potentially coalescing into larger, irregular areas. The sides, frontal scalp, and lower occiput are the most commonly involved scalp regions. While often asymptomatic, lichen planopilaris may present with pruritus, pain, tenderness, discomfort, or burning. The disease course is typically one of slow progression, with diffuse alopecia being an uncommon presentation [4,17].

- presence of one or more frontal veins at the cutaneous-subcutaneous level
- increased skin vascular flow
- hypoechoic perifollicular thickening.

Hair Pull Test in FFA can be particularly informative during active disease phases. Even in the absence of visible inflammation, positive results (increased hair shedding) may be observed at the margins of affected areas [13].

TRICHOSCOPY FINDINGS

Lichen planopilaris (LPP)

Trichoscopy reveals characteristic findings in lichen planopilaris, including white dots, a target pattern of perifollicular blue-black dots, perifollicular erythema, and hyperkeratosis. Notably, the absence of follicular openings is also a key trichoscopic feature [18,19]. Dermoscopically identified erythema and hyperkeratosis suggest active lichen planopilaris (LPP) and can guide targeted biopsy selection in cases of diagnostic ambiguity [19,20,21]. Some authors report the presence of follicular tufts, a feature occasionally observed in LPP that can differentiate it from folliculitis decalvans, where single follicles erupt with multiple hairs [19,20,22]. In contrast to discoid lupus erythematosus (DLE), lichen planopilaris (LPP) may exhibit regrowth and the presence of tapered hairs [20]. The early fibrotic phase of classic lichen planopilaris may present with characteristic milky-red (strawberry ice cream-colored) areas devoid of follicular openings. Trichoscopy revealed the presence of blue-gray dots exhibiting a characteristic concentric arrangement around follicular ostia, resembling a target pattern. This finding contrasts with discoid lupus erythematosus (DLE), where blue-gray dots typically present in a diffuse, speckled pattern [12].

FRONTAL FIBROSIS ALOPECIA (FFA)

Graham-Little-Piccardi-Lassueur syndrome (GLPLS)

While research on trichoscopic findings in Graham-Little syndrome is still emerging, existing studies offer valuable insights for diagnosis. However, we came across two study cases. Patokar et al. (2022) reported a case of coexisting oral lichen planus and lichen pigmentosa in a pregnant woman. Trichoscopy revealed fibrotic white dots, perifollicular erythema, and tubular casts. Hair growth was observed along the hair shafts [23].

In turn, Li and colleagues (2020) described the case of a woman who presented with a two-year history of patchy hair loss, itchy scalp, and dandruff-like lesions. The woman was diagnosed with Graham-Little syndrome. Trichoscopic examination revealed perifollicular erythema, tubular casts, and fibrotic white dots

on the scalp. Additionally, hair growth was observed along the hair shafts. Lesions on the trunk displayed a characteristic "target" pattern of blue-gray dots with reduced follicular openings [24].

Fibrosing alopecia in a pattern distribution (FAPD)

FAPD necessitates differentiation from alopecia androgenetica (AGA) due to distinct trichoscopic features. While both may show individual hair follicles and variable hair diameter, FAPD is characterized by:

- excessive perifollicular keratinization
- perifollicular erythema
- loss of follicular ostia.

The presence of perifollicular casts surrounding 2-3 hair tufts is a characteristic finding and are considered ideal for biopsy specimens. The authors acknowledge that trichoscopic evaluation alone might not be sufficient for definitive diagnosis, particularly in individuals with darker skin pigmentation. This limitation arises due to the potential for misinterpretation of scattered white spots, which could resemble scarring alopecia, and the persistence of the honeycomb pigment network [15].

Scleroderma

Scleroderma is a connective tissue disease and is divided into limited and systemic. Unlike systemic scleroderma, which affects internal organs, limited scleroderma (morphea) mainly affects the skin, less often involving deeper tissues and less often leading to complications. They are separate disease entities but can coexist [25].

Systemic scleroderma (scleroderma)

Scleroderma is a chronic disease that is characterized by fibrosis and has an autoimmune basis [26]. The disease affects the skin and various internal organs. Its complex pathophysiology involves early endothelial cell damage, inflammatory infiltration, and a subsequent reaction leading to fibrosis [26,27]. Most commonly, systemic scleroderma affects the skin, gastrointestinal tract, and lungs [28].

This disease manifests itself, among other things, with ulceration of the phalanges, the appearance of Raynaud's sign, increased pruritus of the skin, the presence of hypopigmentation or hyperpigmentation, a feeling of fatigue or weakness of muscle strength, arthromuscular changes, kidney damage, and in the case of lung involvement: pulmonary hypertension or interstitial lung disease [29].

Diagnosis of systemic scleroderma

Currently, doctors use classification criteria prepared by experts from the European League Against Rheumatism (EU- LAR) and the American College of Rheumatology (ACR). These include thickening of the skin proximal to the metacarpophalangeal joints (bilateral) (9points), thickening of the skin of the fingers (2-4points depending on the subtype), changes on the fingertips (2-3points), telangiectasias (2points), capillaroscopy changes (2points), pulmonary hypertension and/or interstitial lung disease (2points each), Raynaud's puff (3points), and systemic scleroderma-specific autoantibodies (ACA, Scl-70, RNAP III) (3points). To diagnose systemic scleroderma, ≥ 9 points are required [29].

In addition to a detailed history and physical examination, every patient with newly diagnosed systemic scleroderma should undergo a comprehensive serological evaluation to detect disease-specific autoantibodies [28].

Alopecia can also be one of the first symptoms of scleroderma and its comorbidities, as described, among others, in a case report of a 48-year-old woman with a 3-year history of progressively worsening hemiatrophy (Parry-Romberg syndrome) of the right side of her face on the background of systemic scleroderma. Her initial symptom was just baldness. Parry-Romberg syndrome is a rare neurocutaneous disease seen during scleroderma, affecting the skin, subcutaneous tissue, bones and muscles of one side of the face [30].

Morphea

Morphea can be distinguished from systemic scleroderma by the absence of Raynaud's phenomenon, sclerodactyly and nail capillary lesions, which are present in the latter [31].

Speaking of alopecia, mention should be made of the clinical variant of morphea - linear scleroderma, manifested by a linear patch of alopecia, localized on the frontal surface of the scalp (LSCS - Linear scleroderma en coup de sabre) [32]. This subtype can be accompanied by complications involving the eyes, nervous system, mouth, and teeth, or progressive hemifacial atrophy (Parry-Romberg syndrome) [31].

Diagnosis of morphea

The diagnosis of morphea is usually based on typical clinical symptoms. In most cases, there is no need for histopathologic evaluation of skin biopsies or laboratory tests [31]. A useful diagnostic noninvasive method for diagnosing this disease is trichoscopy. In the trichoscopic image in the course of this disease, fibrosis can be seen, interspersed with thick, short, or curvilinear vessels with branching, white patches, loss of hair follicle outlets, broken hairs, or black dots [32]. Campione and colleagues described fibrotic bands and small white patches cut by linear, branching vessels that may show a lilac ring in the trichoscopic image of this condition. Histopathologically, these findings are consistent with dermal sclerosis found in advanced fibrotic lesions. The black dots and broken hairs seen on trichoscopic images are correlated with the presence of perifollicular fibrosis. Sclerosis that is progressive in LSCS is the cause of scarring alopecia, which can be confirmed by the absence of follicular openings on trichoscopy [33]. Romagnulo M, Moltrasio Ch, and colleagues suggest considering scalp morphea as a differential diagnosis of monofocal scarring alopecia involving the scalp [34].

Diagnostic methods for cicatricial alopecia

The diagnosis of scarring alopecia should begin with a thorough medical history and physical examination. Symptoms such as itching, pain, burning, and tenderness suggest active disease. However, sometimes an active inflammatory state can only be confirmed through a scalp biopsy, even without clinical symptoms.

The cornerstone of diagnosis is a scalp biopsy, involving the collection of multiple samples from active areas under trichoscopic guidance. This procedure allows for precise histopathological results and determines the type of scarring alopecia.

Additional dermatological examinations, such as trichoscopy, are useful in diagnosing scarring alopecia. A characteristic trichoscopic feature of the final stage of scarring alopecia is the loss of follicular openings due to their irreversible destruction [35].

TRICHOSCOPY IN MONITORING THE TREATMENT OF SCARRING ALOPECIA

Trichoscopy is a valuable tool for monitoring therapeutic progress in scalp conditions characterized by alopecia scarring [12,36]. Inactive lesions of DLE exhibited a complete absence of follicular openings, porcelain-white areas, and white dots. In contrast, active lesions were characterized by prominent, bulging "large yellow dots" and scattered dark brown skin discoloration [3]. In the active form of DLE, red dots can be observed, regularly distributed around follicular openings [36].

Nevertheless, additional research is imperative to elucidate the evolution of trichoscopic findings throughout therapeutic interventions.

CONCLUSIONS

Cicatricial alopecia poses a significant challenge to modern medicine. It not only results in permanent hair loss but can also be a symptom of other, more serious health conditions. It is important to note that cicatricial alopecia often co-occurs with other pathologies such as lichen planopilaris, sarcoidosis, systemic lupus erythematosus, and scleroderma. In the course of the above-mentioned diseases, alopecia has specific trichoscopic features. It is known that hair loss in systemic lupus erythematosus is a common symptom with diverse manifestations. It can present as patchy, diffuse, or cicatricial alopecia, making differential diagnosis challenging. A thorough scalp examination, including trichoscopy, and biopsy are crucial for confirming the diagnosis and ruling out other causes of hair loss. Lichen planopilaris is a heterogeneous disease syndrome with diverse clinical manifestations, leading to cicatricial alopecia. Trichoscopy is an invaluable tool in differential diagnosis, enabling rapid and accurate assessment of skin and hair changes. Scalp sarcoidosis is a rare disease that is difficult to diagnose. For this reason, it is crucial to perform microscopic and histopathological examinations to analyze it. Alopecia can also be one of the first symptoms of scleroderma. Scientist suggest also to consider scalp morphea as a differential diagnosis of mono-focal scarring alopecia involving the scalp. Early diagnosis and aggressive therapy are crucial for improving prognosis and preventing complications.

AUTHOR CONTRIBUTIONS

HD: conceptualization, literature review, writing - original draft preparation; ŁS, KM, SS: literature review, writing - review and editing.

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CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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