Skin Appendage Disorders

Review Article

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Hair Disorders in Autoimmune Diseases

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Abstract
Alopecia is a common feature in several autoimmune diseases. With a wide spectrum of clinical presentations, it may manifest with a scarring or non-scarring nature, in a diffuse, patchy, or localized pattern. We as dermatologists have the opportunity of assessing patients with hair loss who may have an underlying undiagnosed autoimmune disorder. This review aimed to describe the main clinical, trichoscopic, and histopathological features of hair disorders associated with autoimmune diseases.

Introduction

Skin manifestations are almost always present in patients with autoimmune diseases. Within the wide spectrum of cutaneous presentations, hair disorders represent a significant feature that greatly affects quality of life [1].

Alopecia in autoimmune disorders may manifest as patchy or diffuse alopecia, scarring or non-scarring alopecia, etc. [2]. Identifying hair loss is of utmost importance, particularly when it represents disease activity (i.e., lupus erythematosus [LE], dermatomyositis [DM], and scleroderma) [1]. Dermatologists may be the first physicians to assess patients with hair loss, thus providing valuable opportunities to contribute to the timely diagnosis and prompt treatment of an underlying autoimmune disease. This review aims to describe the clinical and histopathological features of hair loss associated with autoimmune diseases (Table 1).

Lupus

Hair loss is one of the most frequent manifestations of systemic lupus erythematosus (SLE), affecting approximately 50% of patients at some point in their disease [3]. Alopecia may be the initial sign of SLE, affecting not only the hair on the scalp but also that of the eyelashes, eyebrows, and body [1]. Cutaneous findings, including hair loss, are the initial disease manifestations in 20–25% of all SLE cases [4].

Proper identification of hair loss relies on its importance as a marker of disease activity. The spectrum of alopecia varies from scarring alopecia, typical of discoid lupus erythematosus (DLE), to non-scarring alopecia, commonly seen in acute LE. Alopecia in LE has been subclassified as LE-specific and LE-nonspecific according to their histopathological findings (Table 2) [3, 5].

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Scarring Alopecia

Discoid LE

Epidemiology and Pathogenesis

Scarring alopecia in chronic DLE is a LE-specific cutaneous lesion in Gilliam's classification [6]. The scalp is involved in almost 60% of cases. One-third of cases of scalp DLE progress to irreversible scarring alopecia, which negatively affects the quality of life [4]. Its etiology remains unknown; however, it may involve genetic, hormonal, immunoregulatory, and environmental factors. Damage to the pilosebaceous unit in DLE is mediated by cytokines, T cells, immune complexes, and autoantibodies [2].

Its pathogenesis involves cytotoxic inflammation, loss of hair follicle immune privilege, and bulge stem cell injury [7]. Permanent damage to the bulge area, where multipotent stem cells reside, results in irreversible scarring alopecia [8].

Clinical Presentation

A well-demarcated erythematous scaly patch with follicular hyperkeratosis is observed in the early stages. It may be asymptomatic, pruriginous, or tender to the touch. As it progresses, an alopecic atrophic-whitish discoid plaque with follicular plugging and telangiectasias appears (shown in Fig. 1a) [9]. The mnemonic PASTE (plugging, atrophy, scale, telangiectasia, and erythema) is useful for describing the main findings [1].

These scalp lesions are susceptible to Koebnerization. Only 5–15% of all cases evolve into SLE. Patients at highest risk are those with cutaneous lesions below the head and neck, arthritis, or abnormal laboratory test results (high antinuclear antibody titer, anemia, leukopenia, and an elevated erythrocyte sedimentation rate) [5].

Trichoscopy

Follicular red dots, the most characteristic and specific sign of scalp DLE, are erythematous, polycyclic, concentric structures distributed around follicular openings that indicate active disease (shown in Fig. 1b). Additional dermoscopic features include white structureless areas, thick radially distributed arborizing vessels, white scales, large yellow dots, scattered dark-brown discoloration, loss of follicular ostium, and blue-gray dots in a speckled pattern (shown in Fig. 1c) [5, 10].

Histopathology

The epidermis has follicular keratotic plugs, a thickened basement membrane, and vacuolar interface dermatitis with apoptotic keratinocytes. Dermal mucin deposition

Table 1. Summary of hair manifestations in autoimmune diseases

Autoimmune disease	Scarring/Non-scarring alopecia	Clinical presentation	Trichoscopic findings
DLE	Scarring alopecia	Early stage: erythematous scaly plaque with follicular plugging Late stage: atrophic-whitish depressed plaque with follicular plugging and telangiectasias	Follicular red dots, plugging, atrophy, scales, telangiectasias, scattered dark-brown discoloration, and erythema
Lupus panniculitis	Scarring alopecia	Erythematous-indurated scarring plaques	Black dots, large yellow dots, thick arborizing vessels, short vellus hairs, and diffuse erythema
Acute LE	Non-scarring alopecia	Diffuse hair loss: resembles pattern hair loss Patchy alopecia: multiple erythematous alopecic patches "Lupus hair": dry and fragile short hairs in frontal hairline	Hypopigmented hair shafts, prominent arborizing blood vessels, black dots, brown-scattered pigmentation, and blue-gray speckled pigmentation
Linear and annular lupus panniculitis of the scalp	Non-scarring alopecia	Linear erythematous alopecic plaques along Blaschko's lines	Angulated hairs, broken hairs, thick arborizing vessels, patchy erythematous areas, black dots, large yellow dots, perifollicular scale
SSc	Non-scarring alopecia Scarring alopecia	Diffuse or patchy hair loss	Salt and pepper sign, avascular areas, polymorphic microvessels, spider vessels, capillary loops, and arborizing vessels
LSCS	Scarring alopecia	Dyschromic atrophic and shiny linear plaque following Blaschko's lines (paramedian forehead and frontal scalp)	Whitish patches, thick linear and branching vessels, loss of follicular openings, and isolated pili torti
DM	Non-scarring alopecia	Diffuse, erythematous-violaceous, atrophic, scaly plaques, accompanied by intense pruritus	Enlarged capillaries, peripilar casts, tufting, and interfollicular scales

and perivascular and periadnexal inflammatory lymphohistiocytic infiltrates are frequently observed. In the late stages, interstitial fibrosis, a decreased number of sebaceous glands, and a loss of follicular units occur (shown in Fig. 1d) [3, 11]. Direct immunofluorescence reveals linear or discontinuous deposition of immunoglobulin G (IgG) and complement component 3 (C3) on the dermo-epidermal junction and follicular epithelium [11].

Differential Diagnosis.

DLE scalp lesions must be differentiated from lichen planopilaris, which presents as multiple coalescing areas of hair loss associated with erythema, scaling, and scarring. Patients often present with moderate to intense pruritus. Trichoscopy reveals white peripilar scales, perifollicular erythema, violaceous-blue interfollicular areas, elongated blood vessels, and large irregular white dots. Other important differential diagnoses to consider are frontal fibrosing alopecia (FFA), central centrifugal cicatricial alopecia, and pseudopelade of Brocq [3, 11].

Treatment

Prompt treatment is key to avoiding irreversible DLE scarring alopecia. Photoprotection and smoking cessation are recommended for the prevention of new lesions. The first-line topical treatments include potent corticosteroids or intralesional triamcinolone. Additionally, topical calcineurin inhibitors are useful in thinned or atrophic skin areas [1]. Oral antimalarials are first-line systemic therapy. Hydroxychloroquine is usually the preferred initial medication. Phosphate chloroquine or quinacrine may also be used [1]. Approximately 75% of patients respond adequately to these therapies. Other treatment options include topical R-salbutamol, dapsone, methotrexate, thalidomide, mycophenolate mofetil, and topical and systemic retinoids [5, 12, 13]. In severe or recalcitrant cases, rituximab, intravenous immunoglobulin, or ustekinumab may be considered [13, 14].

Non-Scarring Alopecia

Acute LE

Epidemiology and Pathogenesis

Hair loss is frequently observed in SLE, especially during exacerbations. It predominantly affects the scalp; however, involvement of the eyelashes, eyebrows, and body hair has been observed as well [1]. Three main clinical presentations have been described: diffuse alopecia, patchy alopecia, and lupus hair [5]. The pathogenesis of non-scarring alopecia in SLE is not fully elucidated. High levels of proinflammatory cytokines may have a negative effect on the hair growth cycle. Type I interferons are well known to play a role in SLE and CLE, as they inhibit the anagen phase and induce premature hair shedding [15].

Table 2. Lupus alopecias

Lupus-specific alopecia
Scarring
DLE
Lupus panniculitis/profundus
Non-scarring
Diffuse alopecia (acute LE)
Patchy alopecia (acute LE)
Linear and annular panniculitis of the scalp
Lupus hair
Lupus nonspecific alopecia
Anagen effluvium
Telogen effluvium
AA

Clinical Presentation

Diffuse non-scarring alopecia is the most frequent presentation (22–31% of patients) [16]. It resembles female-pattern hair loss or telogen effluvium, with a positive hair pull test. Its severity ranges from mild to severe hair thinning involving more than 50% of the scalp [15, 17].

Another common clinical scenario is patchy, non-scarring alopecia. Patients have multiple well-defined erythematous patches of partial or total hair loss with a positive hair pull test [18]. Additionally, yellow scales and hypopigmented hairs may be observed. It can be easily mistaken for alopecia areata (AA) without proper trichoscopic examination [19, 20].

And finally, lupus hair is a well-recognized disease entity that is present in 5–30% of patients with chronically active SLE [21]. The anterior hairline features dry and fragile short hairs. Slow hair regrowth and hair shaft breakage are common manifestation [4]. The growth of depigmented fine hairs along the frontal hairline is observed during recovery. In these three clinical scenarios, hair regrowth occurs once SLE is properly controlled [16, 19].

Trichoscopy

Trichoscopic examinations help distinguish non-scarring LE alopecia from other types of hair loss. Hair

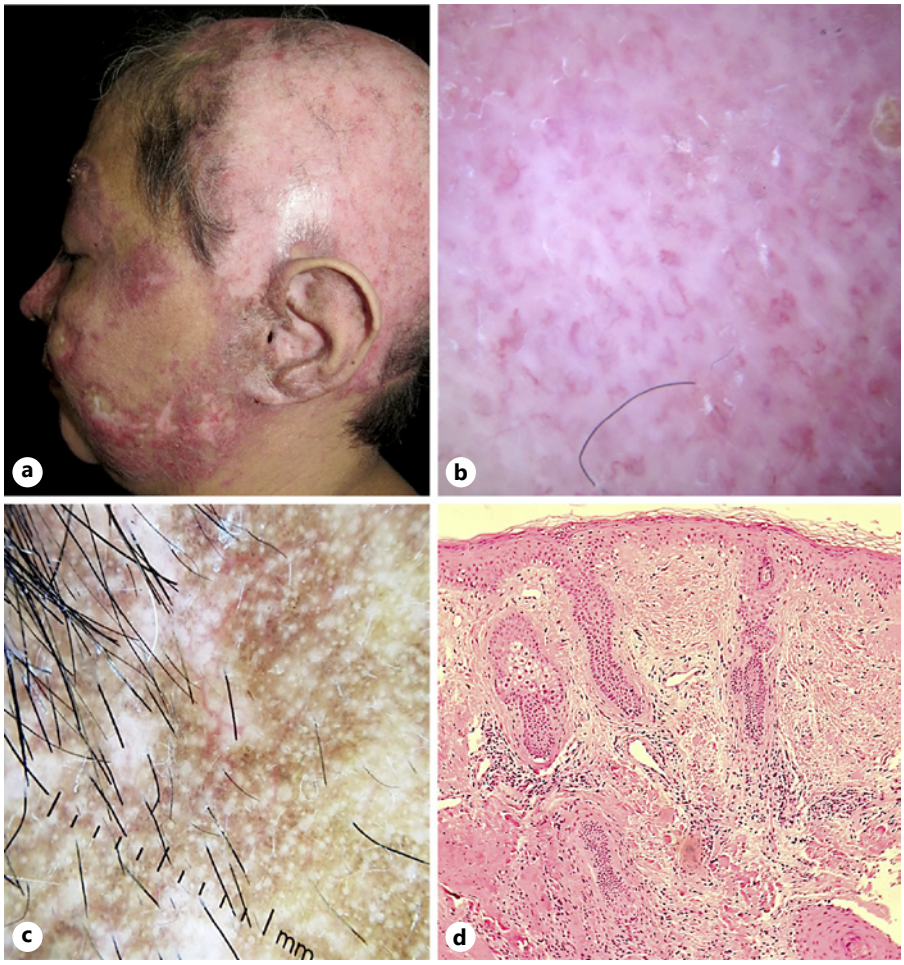

Fig. 1. Discoid LE. **a** Clinical image: erythematous patches, atrophic confluent alopecic discoid plaques, and telangiectasias. **b** Trichoscopy: follicular red dots. **c** Trichoscopy: thick arborizing vessels, scales, white structureless areas, scattered brown discoloration, and loss of follicular ostia. **d** Histopathologic findings (H&E stain, × 10): epidermal atrophy, vacuolar interface dermatitis, periadnexal and perivascular lymphohistiocytic infiltrate, and perifollicular mucin deposition.

shaft thinning and hypopigmentation, broken hairs, interfollicularly prominent and thick arborizing blood vessels, blue-gray pigmentation, black dots, and scattered brown pigmentation are common trichoscopic features in the three types of SLE non-scarring alopecias [15, 17].

Histopathology

Histopathological features that support the diagnosis of non-scarring alopecia in SLE are epidermal atrophy, vacuolar interface dermatitis, pigment incontinence, peri-eccrine, perifollicular, and perivascular lymphoplasmacytic inflammation, and increased dermal mucin deposition [22]. Direct immunofluorescence studies of patients with SLE and non-scarring alopecia show homogenous granular depositions of IgG, immunoglobulin M (IgM), immunoglobulin A, and *C3* along the hair follicles and at the dermo-epidermal junction [15].

Differential Diagnosis

In diffuse alopecia, the main differential diagnosis must be made with telogen effluvium, anagen effluvium, and pattern hair loss. In contrast, patchy alopecia must be distinguished from AA. Trichoscopy may help in distinguishing between these two entities. In LE patchy alopecia, trichoscopy reveals interfollicular polymorphous telangiectasias, which are not a reported feature in AA [3, 5]. Syphilitic alopecia also must be ruled out. Due to the receding hairline in lupus hair, an accurate diagnosis is needed to avoid classifying it as FFA. However, in lupus hair, no fibrosis is evident, and the presence of brittle hairs is characteristic. Physical examination, trichoscopic features, and histopathological findings are imperative for a timely and precise diagnosis [15, 19].

Treatment

Complete hair regrowth occurs with SLE treatment. As with DLE, antimalarials are considered first-line systemic

therapy. Oral corticosteroids and dapsone are other useful options. In recalcitrant cases, methotrexate, thalidomide, mycophenolate mofetil, azathioprine, and cyclosporine may be considered [1, 17]. Additionally, baricitinib, a JAK 1 and 2 inhibitor, has been reported effective in improving refractory diffuse non-scarring alopecia in a patient with SLE [13].

Linear and Annular Panniculitis of the Scalp

Epidemiology and Pathogenesis

Linear and annular panniculitis of the scalp (LALPS), first described by Nagai et al. in 2003, is a rare non-scarring subtype of classic lupus panniculitis that involves the scalp exclusively along Blaschko's lines [23]. A slightly higher prevalence in young Asian males has been observed, but Caucasians and blacks are also affected. Nevertheless, the overall female-to-male ratio is 1:1 [24].

The exact pathogenesis of LALPS remains unknown. It is hypothesized that the inflammatory process is limited to the subcutaneous tissue without affecting the hair bulge stem cells, thus allowing complete hair regrowth. Its reversible and non-scarring nature is explained by the fact that inflammation spares the epithelial hair follicle stem cells [25].

Clinical Presentation

Clinically, patients present with linear, annular patches of non-scarring alopecia following Blaschko's scalp lines. Linear patches are the most common presentations. In some cases, the coexistence of the two morphologies, such as linear and annular lesions, has been observed. Additionally, some infrequent clinical presentations (spiral and bizarre configurations) have been reported [26]. The parietal scalp appears to be the most frequently affected (in almost 70% of patients) [27]. Antinuclear antibodies are positive in 50–53.3% of all cases [23, 27]. Systemic involvement is extremely unusual; in fact, there has only been one case of LALPS associated with SLE to date [28].

Trichoscopy

Trichoscopic descriptions of LALPS are scarce. Udompanich et al. [23] published a case of LALPS featuring angulated hairs, broken hairs, black dots, exclamation mark hairs, prominent blood vessels, and large yellow dots as its main trichoscopic features. Additional findings included perifollicular white scales, patchy erythematous areas, thick arborizing vessels, hair miniaturization, and empty follicles [5, 25, 27].

Histopathology

In LALPS, alterations are usually limited to the subcutaneous tissue. Although the epidermis is often spared, atrophy and vacuolar interface dermatitis may occur. Dense perivascular and periadnexal lymphocytic infiltrates are common in the dermis. Lobular lymphocytic panniculitis, fat necrosis, hyaline fat degeneration, and abundant mucin deposition are also seen [5]. Additional findings include follicular plugging, apoptotic keratinocytes, and a plasma cell inflammatory infiltrate [24, 25]. Granular deposition of IgG or IgM in the basement membrane and peribulbar area may be seen on a direct immunofluorescence test, but such results may also be negative [5].

Differential Diagnosis

The differential diagnosis of LALPS must be made with other causes of local non-scarring alopecia, including AA, syphilitic alopecia, trichotillomania, and SLE patchy alopecia. Some of these conditions share trichoscopic findings, so a detailed physical examination usually leads to a proper diagnosis. A skin biopsy can aid in the diagnosis of LALPS. Morphea en coup de sabre clinically may resemble the linear lesions of LALPS; however, trichoscopy and histopathology will reveal features of scarring alopecia [5, 23].

Treatment

In most cases, combining hydroxychloroquine and corticosteroids (topical, intralesional, and/or systemic) helps achieve good results with complete hair regrowth. Additionally, dapsone, intravenous immunoglobulin, thalidomide, mycophenolate mofetil, methotrexate, and topical minoxidil may be effective therapeutic options [23, 24, 28].

Dermatomyositis

DM is an autoimmune inflammatory myopathy with cutaneous and systemic symptoms [29]. The scalp is frequently affected by moderate to severe burning pruritus; hair loss may also be present [30].

Epidemiology and Pathogenesis

DM, similar to other autoimmune diseases, has a female predominance. Hispanics and African Americans have a higher prevalence of DM [31, 32]. Scalp involvement occurs in 63–82% of patients [30, 33]. Associated diffuse alopecia is present in 33–87.5% of all cases [30, 31].

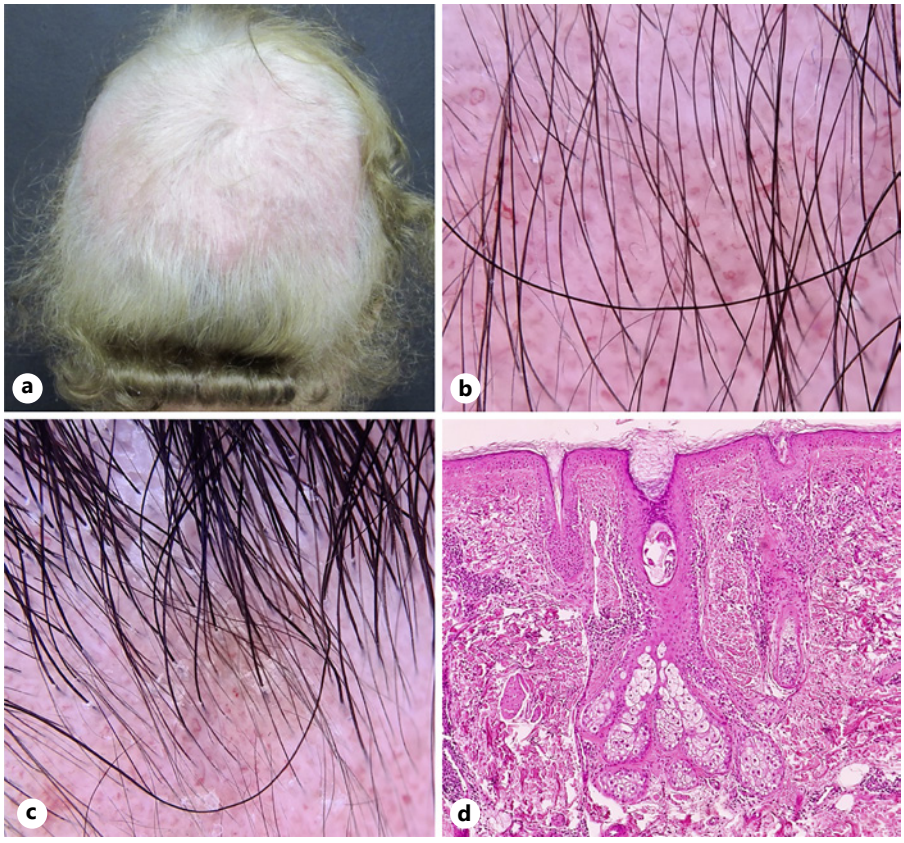

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Fig. 2. DM. **a** Clinical image: diffuse alopecia, erythema and scaling. **b** Trichoscopy: multiple enlarged tortuous capillaries, decreased hair density and vellus hairs. **c** Trichoscopy: peripilar casts, interfollicular scales, and tortuous capillaries. **d** Histopathological findings: follicular plugging, epidermal atrophy, vacuolar interface dermatitis, and perifollicular lymphocytic infiltrate.

The pathophysiology of DM is multifactorial and not fully understood. A combination of environmental, genetic, and immune factors, such as specific antibodies (anti-Mi2, MDA5, anti-NXP2, anti-TNF1, and SAE), are involved in DM development [32].

Clinical Presentation

Scalp DM is characterized by erythema, scaling, poikiloderma, and non-scarring diffuse alopecia (shown in Fig. 2a) [31]. Intense pruritus or burning sensations are key symptoms [34]. Scalp involvement may precede other cutaneous findings (Gottron papules, Gottron sign, heliotrope rash, shawl sign) or appear later during disease evolution. Diffuse hair loss is reversible once the disease is controlled [33].

Trichoscopy

The most common trichoscopic findings are enlarged tortuous capillaries and peripilar casts. Additionally, hair tufting, interfollicular scales, bushy capillaries, vascular lake-like structures, and interfollicular and perifollicular pigmentation can occur [31, 35, 36] (shown in Fig. 2b–c).

Histopathology

DM skin lesions classically show epidermal atrophy, vacuolar degeneration of the basement membrane, interstitial mucin deposition, and sparse lymphocytic infiltrates [2]. Scalp DM biopsies predominantly show dilated capillaries and mucin deposits. Interface dermatitis, atrophic epidermis, basement membrane thickening, hyperkeratosis, and acrosyringial hypergranulosis are other common findings (shown in Fig. 2d) [37].

Differential Diagnosis

When the scalp is the only involved site, DM is commonly mistaken for contact dermatitis, seborrheic dermatitis, or psoriasis. If an autoimmune disease is considered, DLE must be ruled out [1, 2]. A thorough medical history, physical examination, and proper laboratory work-up in addition to histological findings can aid the correct diagnosis.

Treatment

The treatment of cutaneous lesions in DM can be challenging. Since pruritus is often a prominent complaint, sedating antihistamines or tricyclic antidepressants such as doxepin or amitriptyline may be used. Other options

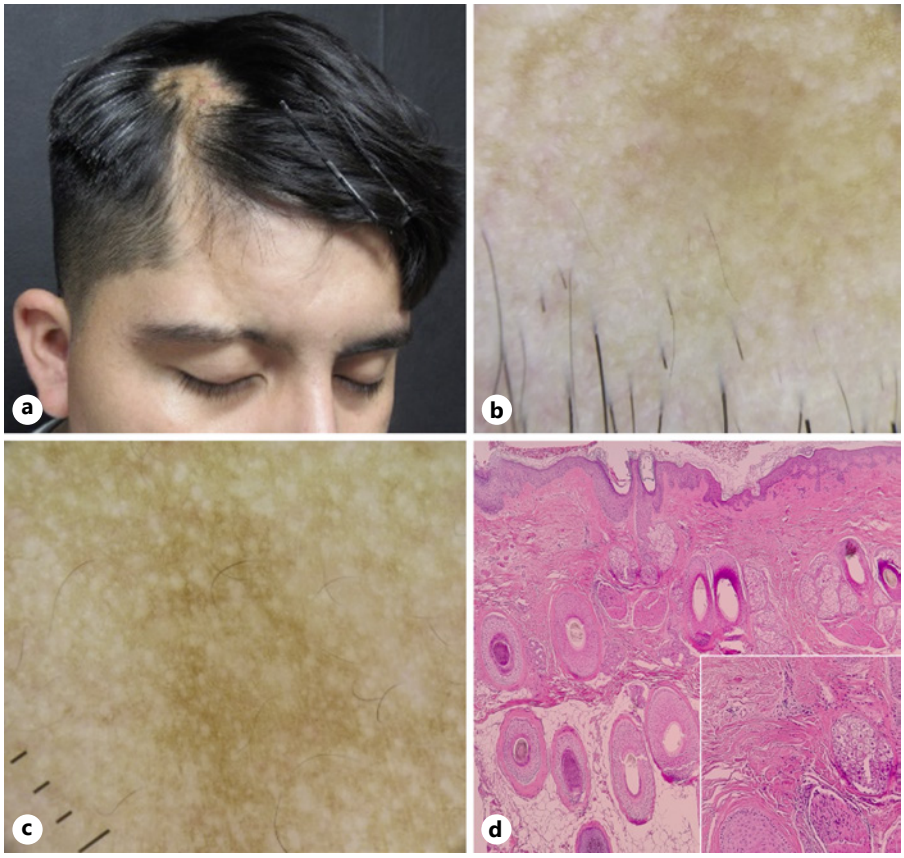

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Fig. 3. LSCS. **a** Clinical image: linear paramedian scarring alopecic patch. **b** Trichoscopy: small white patches, loss of follicular openings, vellus hairs, and broken hairs. **c** Trichoscopy: scattered brown dots, atrophic patches, and vellus hairs. **d** Histopathological findings: thickened hyalinized collagen, perivascular, and periadnexal lymphocytic infiltrate.

include pregabalin or gabapentin [2]. Topical treatments may include corticosteroids and calcineurin inhibitors. For recalcitrant lesions, systemic treatment with hydroxychloroquine, corticosteroids, methotrexate, azathioprine, and mycophenolate mofetil can be considered. Other useful options include intravenous immunoglobulin, dapsone, thalidomide, leflunomide, rituximab, and apremilast [1, 2, 38].

Scleroderma

Scleroderma is classified into systemic sclerosis (SSc) and localized cutaneous sclerosis (morphea). Each group is then subdivided into diffuse or limited; plaque, generalized, linear, pansclerotic, or mixed morphea. Both entities may present with hair loss [16].

Systemic Sclerosis

Epidemiology and Pathogenesis

SSc has a female predominance, with a peak incidence during the third and fifth decades of life [1]. Its pathogenesis is still not fully understood; however, a combination of genetic factors, environmental factors, and autoantibodies (anti-centromere CENP-B, DNA topoisomerase I, RNA polymerase III, and anti-Scl70) plays a fundamental role [19].

Clinical Presentation

SSc has no characteristic scalp findings. Cutaneous lesions such as shiny, thickened, and smooth skin with diffuse hyperpigmentation and hypopigmentation with perifollicular sparing in a "salt and pepper" pattern may be observed [1]. Prominent telangiectasias and diminished hair density are common findings. Diffuse hair loss is frequently observed, which in some cases can lead to irreversible scarring alopecia [39].

Trichoscopy

Polymorphic vessels in the frontal area and telangiectasias are characteristic findings of SSc. Other observed vessels include spider vessels, capillary loops, and arborizing vessels. Additional features include avascular areas, ivory-whitish discoloration, and salt and pepper pigmentation [39].

Histopathology

Histopathology varies according to chronicity. Early skin lesions mainly show lymphocytic inflammatory infiltrates, epidermal atrophy, and thickened collagen bundles. In contrast, the later stages are characterized by marked scarce inflammatory infiltrates, reduced follicular units and sebaceous glands, and increased numbers of myofibroblasts [40, 41].

Differential Diagnosis

Diseases characterized by diffuse skin thickening should be ruled out. Scleredema presents with a "woody" induration predominantly in the face, neck, chest, and proximal arms. Contrary to SSc, in which sclerodactyly is a prominent feature, distal extremities are usually spared. Other entities such as graft-versus-host disease, eosinophilic fasciitis, scleromyxedema, scleredema of Buschke, lipodermatosclerosis, amyloidosis, and nephrogenic systemic fibrosis should be ruled out [42, 43]. However, most of them do not affect the hair scalp.

Treatment

Therapeutic options mainly include topical corticosteroids, topical calcineurin inhibitors, and phototherapy (broadband ultraviolet A, narrowband ultraviolet A1, and narrowband ultraviolet B) [1]. Systemic treatments such as corticosteroids, methotrexate, mycophenolate mofetil, and cyclophosphamide may be used [44].

Linear Scleroderma en coup de sabre

Epidemiology and Pathogenesis

Linear scleroderma en coup de sabre (LSCS) predominantly affects children and young adults, mostly males. The mean age at diagnosis is 13.6 years. Most patients (67%) are diagnosed before the age of 18 years [16, 45]. Its pathogenesis is not fully known; however, autoimmune mechanisms and some environmental factors, such as local trauma, are involved [46].

Clinical Presentation

Typically, a well-defined linear alopecic patch is observed on the paramedian forehead or midline, following Blaschko's lines [16]. It is usually unilateral with a shiny, atrophic, hyperpigmented, or hypopigmented appearance. It may also extend into the frontal scalp, causing irreversible scarring alopecia (shown in Fig. 3a) [1].

Trichoscopy

Trichoscopy in cases of LSCS shows fibrotic beams, small white patches, and branching vessels with a lilac ring [47]. Other features include loss of follicular openings, a whitish skin surface, black dots, broken hairs, pili torti, scattered brown dots and globules, and short thick linear and branching tortuous vessels [45, 48] (shown in Fig. 3b–c).

Histopathology

Histological examination shows a "squared punch biopsy" appearance at low power. Histological features vary according to chronicity. In the early stage, interface dermatitis and intense perivascular and periadnexal lymphocytic inflammatory infiltrates are present (shown in Fig. 3d) [1]. Later stages feature diffuse dermal fibrosis, an absence of eccrine sweat glands, and pilosebaceous units [46, 49].

Differential Diagnosis

The diagnosis of LSCS is often made according to the clinical and histological findings. However, the differential diagnosis must include lupus profundus, linear panniculitis of the scalp, progressive hemifacial atrophy, and DLE [5, 50].

Treatment

A combination of topical and systemic therapies is often required to achieve favorable clinical and cosmetic results. Intralesional and topical corticosteroids, topical calcineurin inhibitors, and topical calcipotriene should be considered [1]. Systemic therapy is usually required to achieve a complete response. Methotrexate has a reported improvement rate of 100% after 2 months of treatment[61]. Phototherapy, oral steroids, antimalarials, and mycophenolate mofetil can also be used [1, 16, 51].

Miscellaneous Conditions

Other autoimmune diseases may also be associated with hair manifestations. Rheumatoid arthritis (RA), similar to other autoimmune systemic conditions, can cause telogen effluvium presenting as diffuse nonscarring alopecia [2, 52]. Furthermore, an association between AA and RA was reported in some studies [2]. In a large Taiwanese cohort, patients with RA had a 2.64-fold higher risk of developing AA, especially those of younger age (20–40 years) [53]. Additionally, drug-related alopecia is a frequent clinical scenario [1]. Medications commonly used, such as methotrexate and leflunomide, may cause telogen effluvium and AA,

respectively [54, 55]. Sjogren's syndrome (SS) can manifest as several associated cutaneous and hair findings. Xeroderma, which is the most common dermatologic manifestation, is usually accompanied by dry and lackluster hair [56, 57]. SS has also been associated with FFA, the second most common autoimmune disease of the hair [58–60]. A study of 29 patients with FFA demonstrated that 3% had a positive history of SS [58]. Although a wide spectrum of hair manifestations have been reported, they remain associated features and are not disease-specific.

Conclusion

Most of these diseases lack information about their specific hair manifestations and associated trichoscopic features. More studies are required for clinicians to properly identify one of these diagnoses in patients with scarce disease stigma. Performing a complete dermatological examination including trichoscopy is mandatory in all patients seeking medical attention due to hair loss; an undiagnosed autoimmune disease should be considered as a possible underlying cause. A prompt and accurate diagnosis is fundamental to initiating specific treatment, preventing irreversible scarring alopecia, achieving complete hair regrowth, and improving overall quality of life.

Statement of Ethics

Written informed consent to publish patients' photographs was obtained.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Giselle Rodríguez-Tamez: contribution to the conception and design of the work, drafting the work, final approval of the version to be published, and agreement to be accountable for all aspects of the work. Maira Elizabeth Herz-Ruelas, Minerva Gómez-Flores, and Jorge Ocampo-Candiani: contribution to the acquisition and interpretation of data for the work, drafting the work, final approval of the version to be published, and agreement to be accountable for all aspects of the work. Sonia Chavez-Alvarez: contribution to the conception or design of the work, acquisition and interpretation of data for the work, drafting the work, final approval of the version to be published, and agreement to be accountable for all aspects of the work.

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