Chapter 10

Cicatricial Alopecias

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Additional information is available at the end of the chapter

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Abstract

Primary cicatricial alopecias (PCA) are a rare group of disorders in which the hair follicle is the main target of destructive inflammation resulting in irreversible hair loss with scarring of affected lesions. Inflammation may predominantly involve lymphocytes or neutrophils. Cicatricial alopecias that mainly involve lymphocytic inflammation include lichen planopilaris, discoid lupus erythematosus, pseudopelade (Brocq), central centrifugal alopecia, alopecia mucinosa, and keratosis follicularis spinulosa decalvans. Cicatricial alopecias that are due to predominantly neutrophilic inflammation include folliculitis decalvans and dissecting cellulitis of the scalp. Acne keloidalis, acne necrotica, and erosive pustular dermatosis are cicatricial alopecias with a mixed inflammatory infiltrate.

Keywords: cicatricial alopecia, lichen planopilaris, discoid lupus erythematosus

1. Introduction

Cicatricial alopecias are the result of various diseases of the scalp. It is usually circumscribed but may be widespread [1]. It presents as areas of hair loss in which the underlying scalp is scarred, sclerosed, or atrophic [1, 2]. In early stages, the underlying disease is usually diagnosable clinically and histologically, but in later stages, only scarring may be evident [3–5]. The scarring is the result of the destruction and fibrosis of hair follicles [1, 6, 7]. The primary cicatricial alopecias, which are the focus of this chapter, can be particularly challenging clinically. Several classification schemes for primary cicatricial alopecia exist in the literature. Workshop sponsored by the North American Hair Research Society, a working classification of primary cicatricial alopecias based on the predominant inflammatory cellular infiltrate was developed (**Table 1**) [8].

1—Lymphocytic
Lichen planopilaris
Classic lichen planopilaris
Frontal fibrosing alopecia
Graham-Little syndrome
Chronic cutaneous lupus erythematosus
Classic pseudopelade
Central centrifugal cicatricial alopecia
Alopecia mucinosa
Keratosis follicularis spinulosa decalvans
2—Neutrophilic
Folliculitis decalvans
Dissecting cellulitis
3—Mixed
Acne keloidalis
Acne necrotica
Erosive pustular dermatosis
4—Nonspecific

Table 1. Classification of primary cicatricial alopecias.

2. Lymphocytic cicatricial alopecias

2.1. Lichen planopilaris

Lichen planopilaris is a rare disease characterized by autoreactive lymphocytic destruction of the hair follicle and is the cause of progressive cicatricial alopecia. Perifollicular erythema, squams, and keratotic follicular papules are commonly encountered clinical findings of LPP (**Figure 1**) [4, 9]. Although diagnosis can be made clinically and histopathologically in the early stages, it becomes more difficult to diagnose in the late stages due to the absence of specific findings [1, 5]. Lichen planopilaris can be subdivided into three groups including classic lichen planopilaris, frontal fibrosing alopecia, and Graham-Little syndrome [4, 6]. Classic lichen planopilaris presents as scalp hair involvement and is sometimes accompanied by extracranial lichen forms. Frontal fibrosing alopecia is characterized by band-like scarring alopecia of the frontal hairline that usually affects middle-aged women. Graham-Little syndrome, also known as Graham-Little-Piccardi-Lasseur syndrome, is a disease having a triad of cicatricial alopecia, lichen planus spinulosus, and nonscarring hair loss of axillary and pubic area [1].

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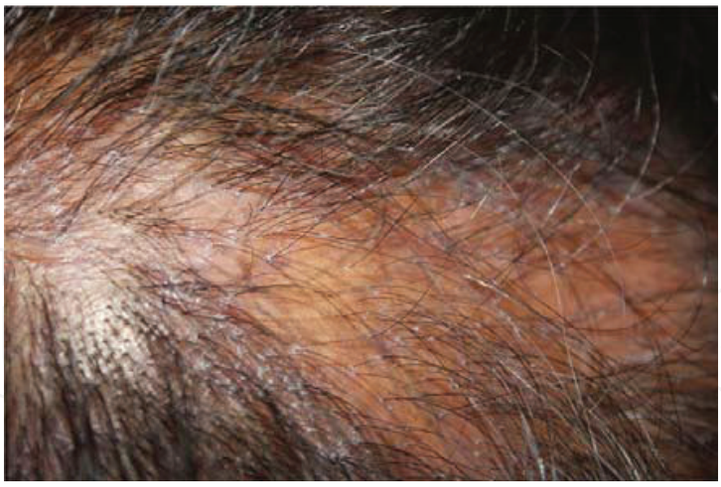

Figure 1. Lichen planopilaris.

2.1.1. Classic lichen planopilaris

It was initially described by Pringle. It is more common in women than in men. Light-skinned individuals are more frequently affected than dark-skinned individuals. Most patients visit a doctor in the first year [1, 4, 10]. Although the etiology of lichen planopilaris is poorly understood, the most widely accepted theory states that it is an autoimmune disorder in which Langerhans cells-activated T lymphocytes destroy keratinocytes. In LPP, most T lymphocytes are located around the bulge area. It is seen that cells in the bulge area are multipotent and are important in generating anagen hair follicle. Destruction of follicular stem cells localized in the bulge area is found to be significant in lichen planopilaris [4, 11]. Contact sensitizers such as metals can enhance a T cell inflammatory reaction. Commonly encountered metals are gold, mercury, and cobalt. The role of infection is determined in the development of LPP. These infections include hepatitis C, HIV, HSV type 2, Helicobacter pylori, and HPV. It was observed that antimalarial agents, beta-blockers, thiazides, ACE inhibitors aggravate lichen planopilaris, and classic lichen planus [1, 3, 4, 12]. Although the pathogenesis of lichen planopilaris shows extensive similarities to the lichen planus, some reports point out differences in immunoreactant deposition. Lichenoid dermatitis is histopathologically detected at the dermoepidermal junction around follicular infundibulum and isthmus. Occasionally similar lichenoid changes may be observed at the papillary dermis between surface epithelium and hair follicles. Mild to severe dyskeratosis and lymphocytic infiltration are encountered. However, epidermal and dermal mucin accumulation seen in DLE are not found [3, 4]. Immunofluorescence evaluation may demonstrate the deposition of fibrinogen, IgM or rarely C3, and IgA at the follicular basement membrane zone of the follicle in a linear pattern. Histopathological findings vary according to disease progression and sometimes it may be difficult to diagnose. It is especially difficult to diagnose in the late stages due to follicular scar. The biopsy site selection is an important process to establish an exact diagnosis. It is recommended to perform biopsy in areas particularly having active lesions. Active inflammatory areas are

squamous, erythematous, and also symptomatic [3, 13, 14]. First signs of LPP include alopecia and pruritus. Rarely pain, burning, and seborrheic dermatitis are observed. The most frequent clinical forms are perifollicular erythema and affinitative, violescent brown, hyperkeratotic follicular papules and flat, atrophic, polygonal-sided alopecic plaques are subsequently appeared following the aggregation of papules. Scalp lesions may be single or multiple, focal, or wide. They are most frequently involved in the vertex and parietal area. Tiny alopecic patches slowly proceed and bind with other patches causing reticular pattern. Pull test is positive in the active periods [1, 4, 15]. Distinctive diagnosis of classic lichen planopilaris is carried out with DLE, pseudopelade of Brocq, folliculitis decalvans, keratosis pilaris spondiloza, alopecia mucinosa, and seborrheic dermatitis. The lack of pustules supports the distinction from folliculitis decalvans. In the clinical manifestation, histopathologic and immunohistochemical properties of LPP facilitate the differentiation from other diseases.

LPP is difficult to treat. Local and systemic therapies are used. Local therapies are initially selected in most cases since they are relatively safe. The aim of therapy is to reduce subjective symptoms and to prevent inflammation and progression [4, 15]. Topical and intralesional corticosteroids are often chosen as the first-line therapy. However, best regimen of treatment for topical corticosteroids in LPP is not known. High potent corticosteroids are mainly selected. If there is no response to corticosteroid therapy, other alternative drugs should be considered. Intralesional corticosteroid injections reduce inflammation significantly. However, atrophy can occur. In progressive patients, prednisone, an oral corticosteroid, can be given at 1 mg/kg/day over 2–4 months. Relapse may occur following the withdrawal of systemic corticosteroid therapy. Hydroxychloroquine has an immunomodulatory property and is well tolerated. Effects of treatment are initiated within 2–3 months and maximal clinical efficacy may take up to 6–12 months. 400 mg/day of oral hydroxychloroquine therapy is recommended after liver function test, complete blood count, and ophthalmologic examination. Adverse reactions are rare, but include abdominal pain, anorexia, nausea, myalgia, skin hyperpigmentation, and ophthalmologic damage. Smoking may decrease efficacy. Cyclosporine is effective in lichen planus. There are reports with mycophenolate mofetil especially in patients unresponsive to corticosteroids and hydroxychloroquine therapy demonstrating its efficacy and lower side effect profile [3, 16]. Other treatment options include retinoids, tetracycline, griseofulvin, thalidomide, dapsone, topical tacrolimus, and minoxidil. However, there effectiveness is still controversial [4, 9].

2.1.2. Frontal fibrosing alopecia

Frontal fibrosing alopecia (FFA), first described by Kossard in 1994, is a form of lichen planopilaris characterized by cicatricial alopecia at the frontoparietal hairline and it occurs primarily in postmenopausal women. Although the disease occurred typically in postmenopausal women, it is rarely seen in men too. A total of 80 cases have been reported so far [17, 18]. Etiopathogenesis of FFA is not fully understood. As most of the cases are postmenopausal, hormone-related triggering mechanisms are thought to play role in the etiopathogenesis. In some cases, successful usage of 5-alpha reductase inhibitors supports the latter. Some reports state that the occurrence of familial cases of FFA points to a possible genetic contribution. The most characteristic clinical feature of frontal fibrosing alopecia is the progressive recession of

the frontal and parietal hairline. All FFA patients exhibit this finding which is required to diagnose [17, 19, 20]. Hairline recession usually occurs symmetrically and bilaterally, giving rise to a band of alopecia between 0.5 and 8 cm from its original site. The progression is relatively slow, and erythema and hyperkeratosis papules indistinguishable from that seen in LPP are common findings. Eyebrow loss or thinning was reported in 62.82% of patients. Despite 50% of lichen planus lesions are observed in lichen planopilaris, the prevalence is only 5% in FFA [3, 17, 21]. Histopathological and immunofluorescence evaluations are similar to classic lichen planopilaris. However, it is harder to diagnose in the late stages. Causes of cicatricial alopecia should be considered in the differential diagnosis of FFA. Clinical features of FFA differ from other alopecias by its histopathological features [1, 4, 22]. Precise, effective treatment options are limited. There are no randomized clinical studies. Corticosteroids are proper approach in the early stages. Systemic corticosteroids can be administered in the dose of 0.5–3 mg/kg per day for a period of 3–18 months. Some studies demonstrated that topical corticosteroids were not effective. Intralesional corticosteroid treatment used in the advanced stages may even worsen the disease rather than improvement. Topical minoxidil revealed controversial results. Mono- or combination therapy of finasteride produced successful results. Other treatment options including griseofulvin, isotretinoin, tacrolimus, pimecrolimus, cyclosporin, hydroxychloroquine do not show promising results [3, 19, 23].

2.1.3. Graham-Little syndrome (Piccardi-Lasseur syndrome)

It was first described by Piccardi in 1914 and the components of the disease were described by Graham-Little in 1915. It has a triad of cicatricial alopecia of the scalp, noncicatricial alopecia of axillae and pubic region and keratotic follicular papules over body and extremities [24]. Patients are generally females aged 30–70 years. The rate of incidence is four times higher in women. Its exact etiology is not known, but it is thought to be a variant of lichen planopilaris [24, 25]. Cellular immunity seems to play a role in the clinical manifestation. However, in a number of cases, hepatitis B vaccination and genetic patterns were thought to be the causes [26, 27]. In the early stages, alopecic patches-like lesions, perifollicular erythema occur in the periphery of scalp [1, 22]. Atrophy is not observed even there is no loss in the hair follicles in the axillae and pubic region. Follicular keratosis occurring mostly over body and extremities is rarely observed on the eyebrows. Histopathological findings include follicular orthokeratotic hyperkeratosis in the early lesions, perifollicular infiltrate of lymphocytes, and loss in pilosebaceous unit. Similar to other cicatricial alopecias, fibrosis is observed in the advanced stages [24, 25]. Treatment options may include topical and systemic corticosteroids, and cyclosporine [24, 28].

2.2. Discoid lupus erythematosus

Cutaneous lupus erythematosus is a widespread disease group. It may be categorized into three main entities: acute, subacute, and chronic. Discoid lupus erythematosus (DLE), is often related to the cicatricial alopecia. Discoid lupus erythematosus most often affects women in 4th and 5th decade of life. Its etiology is not fully understood [4, 29]. It seems that disease occurs following the exposure to UV light of sensitive individuals accompanied by the increase in

keratinocyte apoptosis and the induction of reactive T cell or immunocomplex-mediated response [30, 31]. Compared to other lupus erythematosus forms, the course is benign and transformation to systemic lupus erythematosus is less than 10%. Generalized forms of discoid lupus tend to transform into systemic forms [1, 30, 31].

Approximately 20% of men with DLE and 50% of women exhibit cicatricial alopecia. Lesions are clinically transformed from definite macules and papules into adhesive squams followed by discoid plaques (coin-like shape). Plaques may be painful and itchy. In the course of time, these lesions transform into atrophic plaques having peripheral hyperpigmentation, depigmentation in the middle. Exposure to sun light and trauma may induce the disease [1, 30, 32]. Lesions evolved to squamous cell carcinoma over years. Scalp hair involvement occurred during the first year of disease. Patients generally consult a physician with complaints in hair loss increase and itching. Rarely stinging, burning or scalp sensitivity may occur. Lesions are generally at the vertex localization. Coupled with the centrifugal progression of lesions, plaques containing follicular plugs are formed. Telangiectasia and atrophy are observed on plaques. Approximately 35% of DLE patients manifest a positive antinuclear antibody. Lupus band test is positive in 90% of DLE lesions [4, 31, 32]. Histopathology of DLE reflects its clinical presentation. Epidermis is generally atrophic. Vacuolar degeneration in basal keratinocytes and thickening in the basal membrane are confirmed. Lymphocytic infiltrate is observed around veins and adnexal structures in the dermis. Mucin accumulation is the most significant indicator of cutaneous lupus erythematosus. Collagen and elastic fibrils may get damaged. Lymphocytic infiltrate may diffuse to the subcutaneous fatty tissue. In more than 90% of biopsies IgG and C3 depositions are demonstrated immunopathologically. It is hard to presume the prognosis and course of DLE. Newly diagnosed patients should be evaluated in terms of SLE. Complete physical examination, routine blood tests, urine analysis and anti-dsDNA antibodies are examined. If it is negative, it is regarded as skin-limited form [3, 29, 31, 33].

Differential diagnosis should consider lupus vulgaris, actinic keratosis, sarcoidosis, rosacea, psoriasis, lichen planus granuloma annulare, tinea faciei, and seborrheic dermatitis. Clinically, histopathological and immunological tests are favorable in differentiating from other diseases [1, 4].

First step of the therapy is to recommend sun protection. First-line therapy options include corticosteroids and antimalarial agents. Corticosteroids are administered in forms of potent topical corticosteroids or intralesional triamcinolone acetonide (4–10 mg/mL, monthly injections). Most frequently used antimalarial drug, hydroxychloroquine should be initially started in doses of 200–400 mg daily. Pediatric posology is 4–6 mg/kg. Basal ophthalmological examination and complete blood count should be performed. Side effects are rare and include abdominal pain, anorexia, nausea, myalgia, skin hyperpigmentation, hematologic changes, and ophthalmological damage. Oral corticosteroids can be used until efficacy has been proven (1 mg/kg, tapering in 8 weeks). Oral retinoids can be tried in unresponsive cases. For this purpose, acitretin (50 mg per day) or isotretinoin (40 mg per day) are used. Moreover, topical immunomodulatory agents (e.g., tacrolimus), thalidomide, dapsone, and oral vitamin C may be considered. If none of these treatments are found to be effective, oral immunosuppressant therapies (mycophenolate mofetil, methotrexate, and azathioprine) are initiated [31, 34–36].

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2.3. Classic pseudopelade of Brocq

It was first described by Brocq in 1888. It is frequently characterized as slowly progressing disease with cicatricial alopecic patches without chronic inflammation [37–39]. The cause is not known. There are many debates on accounting the disease. While some researchers believe that Pseudopelade of Brocq is clinicopathologically different disease, some reported that it the last phase of primary cicatricial alopecias. Consequently, successful determination of the disease is not available until this date. The epidemiology is not known. Although Pseudopelade of Brocq may occur in both genders, it is more frequent in women over 40 years of age. It is very rare in pediatric population. Although its etiopathogenesis is not fully understood, the possible causes include genetics, autoimmunity, and infections [1, 38]. Pseudopelade of Brocq is a chronic, and insidious form of primary cicatricial alopecia. There are usually no symptoms. Mild itching may be present. Three clinical forms were identified by Brocq including scattered small plaques, large plaques, and a combination of both. Vertex involvement is frequently observed in all three forms. Patients rarely have squams. Small plaques showed confetti-like scattering. Classic Pseudopelade of Brocq involves scalp, however, beard involvement occurs too. The course of disease is slow and progressive but some cases rarely exhibit fast progression [3, 38]. Biopsy is significant to diagnose histopathologically. Two deep punch biopsies along clinically active alopecic areas should be taken. To differentiate from other cicatricial alopecias patients should undergo both routine and direct immunofluorescence examination. Pseudopelade of Brocq does not have pathognomonic histopathological properties. In the early lesions, there is mild perivascular and perifollicular lymphocyte infiltrate. However, interphase changes are not present. Sebaceous glands are diminished or absent. Follicular epithelial atrophy is present two deep punch biopsies along clinically active alopecic areas should be taken two deep punch biopsies along clinically active alopecic areas should be taken [40, 41]. As alopecia develops, atrophy becomes evident in the infundibular epithelium. Direct immunofluorescence findings are usually negative. In the advanced stages, atrophic epidermis is seen on the dermis containing follicular fibrotic bands extending to the subcutaneous tissue [42, 43]. Differential diagnosis should include alopecia areata, lichen planopilaris, discoid lupus erythematosus, and central centrifugal alopecia.

Treatment objective is to prevent clinical remission and progression of the disease. Unfortunately, the progression still continues even after treatment [38, 44, 45]. Choice of therapy depends mainly on activity, extent, and tolerance of the disease. In patients having less than 10% scalp involvement, topical or intralesional corticosteroid therapy is the first choice. There are reports stating the efficacy of topical tacrolimus and topical minoxidil therapies. In addition, there are also reports indicating that systemic prednisolone, hydroxychloroquine, isotretinoin therapies are effective [36, 43, 44].

2.4. Central centrifugal cicatricial alopecia

It was first described by LoPresti et al. and also referred to as hot comb alopecia and follicular degeneration syndrome [46]. It is the most common form of cicatricial alopecia in African American females. Although its etiopathogenesis is not fully understood, the causes include usage of hot comb, styling practices, chemical agents, infections, autoimmune diseases, and

genetics [47, 48]. Clinically disease initiated at the vertex and middle of the scalp, as it progresses, it can cause centrifugal baldness. Symptoms such as itching or pain are often absent or mild. Most of patients are adult black women [48, 49]. Histopathologic features include perifollicular lymphocytic infiltrate, and fibrosis in the early stages. Terminal follicle count is diminished. In the advanced stages, sebaceous gland and follicle loss are observed. Dyskeratosis and epidermal mucin accumulation are absent. Perifollicular erythema and follicular keratosis is usually absent. Pigment incontinence is minimal. Histopathology in the last stage is similar to other cicatricial alopecias [48, 50]. Moreover, chronic transitional alopecia, androgenic alopecia, alopecia areata, trichotillomania, folliculitis decalvans should be excluded [1, 49, 51].

Treatment objective is to stop progression rather than hair growth. Regrowth is not possible due to the formation of scar tissue. This is related to styling methods and even though this relation is not confirmed, natural hair care techniques not causing trauma are recommended. Hair care method involving chemicals and techniques causing traction should be eliminated [3, 49–53]. Until the disease is stabilized, daily topical corticosteroids usage followed by three times a week posology is recommended. 10 mg/ml doses of intralesional corticosteroids are applied once a month for at least 6 months. The therapy is then continued depending upon the symptoms. Especially in severe cases, oral anti-inflammatory drugs such as tetracycline are applied for at least 6 months. In order to reduce itching and desquamation, seborrhoeic dermatitis-like therapy (shampoos containing zinc pyrithione and/or ketoconazole) is recommended. Antimalarial agents, minoxidil, thalidomide, cyclosporine, mycophenolate mofetil, vitamins, and several herbal treatments are found to be effective. The treatment should be continued at least 6 months and following enhancement, it should be lasted for a year until the remission. Besides, hair transplantation may be performed. Wigs can also be used [1, 36, 43, 49].

2.5. Alopecia mucinosa

It is also known as follicular mucinosis and characterized by the follicular papillary erythema and squam formation in the perifollicular zone [4, 54]. All age groups can be affected and its initiation can go to the infiltrate period. Its etiopathogenesis is not fully understood. Antigenic stimulus T cell-related follicular response formed in hair follicles is thought to increase the folliculotropic response.

Occurrence of lymphoproliferative diseases in 30% of adult patients is an important step in the etiopathogenesis. Its course includes sharp-edged erythematous squamous plaques. The most significant involvement occurs in the head and neck. Alopecia is seen when lesion affects hair follicles. Dysesthesia and anesthesia can be observed in lesions [1, 55, 56]. Clinically, acne-formed, hypopigmented, eczematous plaques and endured nodule forms are reported. Clinically there are 3 types. Primary or benign type occurs in young patients and it is the only lesion tending to improve in several years. Secondary or malignant type occurs in the elderly and it is characterized by many lesions. Third type is chronic benign form having properties of both [4, 55, 57]. The most malignity associated with alopecia mucinosa is mycosis fungoides. Other malignities include leukemia, Hodgkin lymphoma, renal carcinoma, lymphosarcoma. Its

pathology reveals several mucin accumulation in follicular epithelium and sebaceous tissues, perivascular, and perifollicular lymphocyte infiltrates. Dyskeratosis and lameller fibrosis are absent [55, 57].

Differential diagnosis is performed by alopecia areata, telogen effluvium, lichen planopilaris, morphea, tinea capitis, subcutaneous panniculitis-like T cell lymphoma, and dissecting folliculitis.

The most effective therapy in benign type alopecia mucinosa is intralesional steroid treatment. There are a wide range of therapy options. These are topical oral antibiotics, topical retinoids, steroids, dapsone, methotrexate, immunosuppressive agents, nitrogen mustard, and PUVA. In the malignity-associated alopecia mucinosa direct malignity treatment should be performed. Long term follow-ups are needed in chronic alopecia mucinosa [3, 36, 57, 58].

2.6. Keratosis follicularis spinulosa decalvans

Keratosis follicularis spinulosa decalvans (KFSD) is also known as keratosis pilaris decalvans. It was first described by Siemens in 1926. It is characterized by scalp follicular hyperkeratosis and photophobia. The end point is atrophy and cicatricial alopecia. Transition depending on X and sporadic cases also occur. It is thought to be a part of keratosis pilaris atrophicans [59–61]. Its etiology is not known. It is usually initiated around facial with follicular hyperkeratosis in the early life. Later on, it spreads on scalp, hair, and eyelashes. Patch-like alopecia on scalp, hair, and eyelashes occurs and it is followed by cicatrices is observed on alopecic areas. Reddish-brown telangiectasia on lesions can be seen. Moderate itching is present. *Staphylococcus aureus*-related pustule infections can worsen the disease. The disease is generally more severe in men [4, 62, 63]. Histopathologic examination revealed intrafollicular and perifollicular edema and neutrophil infiltrate. As the disease progresses, mucin accumulation around the top of follicles and perivascular and perifollicular lymphocyte infiltration are detected [3, 61, 62].

Differential diagnosis should include KID syndrome, lichen planopilaris, and folliculitis decalvans.

Treatment should be done especially in childhood where the disease is active. Unfortunately, there is no specific treatment. Topical and intralesional corticosteroids, oral retinoids, and dapsone can be used [63, 64].

3. Neutrophilic cicatricial alopecias

3.1. Folicullitis decalvans

Folicullitis decalvans is a disease occurred in pustules on scalp. The cause is not known. Some researchers blame isolation of *S. aureus* in the pustular lesions as its etiology. Immune interaction between microorganisms and the host is thought to be the most significant factor in folliculitis development. Its epidemiology is not fully known. It is one of most important causes of cicatricial alopecia. The prevalence is same in both genders [65–67]. Clinically

initial lesion is erythema follicular pustule and papule. These can be painful and itchy. Later, new pustules are observed and accumulated to form pustular milier abscess (**Figure 2**). Consequently, cicatricial alopecia plaques have been formed [67]. Tufted folliculitis is characterized by having more than one hair on dilated orificial follicles. It is often seen in folliculitis decalvans [67, 68]. Concurrently DLE, LPP, acne keloid, and tinea capitis can occur during the course. In order to diagnose folliculitis decalvans, biopsy must be taken from pustules. Although pathognomonic findings are absent in the histopathological evaluation, follicular neutrophilic pustules in the early stages, plasma cells in chronic period are seen. As a response to follicular damage, foreign matter granules can be formed and consequently common fibrosis occurs. Careful histological and clinical evaluation should be carried out even after the diagnosis since the disease can overlap with other alopecias. Diagnosis should take into consideration of dissecting folliculitis, acne keloid, erosive pustular dermatosis, acne necrotica, DLE, CCCPA, LPP, and pseudopelade [67, 68].

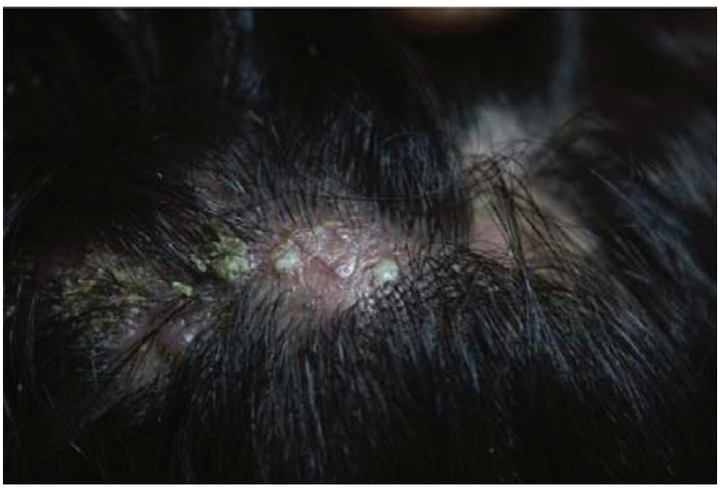

Figure 2. Folliculitis decalvans; pustules on the scalp are noticed.

Bacterial cultures should be taken from each patient and antibiotic resistance should be determined prior treatment. Long-term antibiotics targeting *S. Auerus* must be used. Tetracycline, coloxylin, erythromycin is used as first-line therapy. Antibiotic treatment is initiated as in acne and reduced according to the response. The disadvantage of antibiotic therapy is recurrence. Clindamycin and rifampicin is used in combination in unresponsive patients. Other effective choice of therapy is isotretinoin. It can be administered intralesionally in fast progressive cases. Other treatments include dapsone, hydroxychloroquine, adalimumab, infliximab. Wigs can be recommended as cosmetic camouflage in patients with cicatricial alopecia [36, 69, 70].

3.2. Dissecting cellulitis

Described by Hoffman in 1908, it is also known as perifolliculitis capitis abscedens et suffodiens. It forms follicular occlusion triad along with acne conglobate and hidradenitis

suppurativa. If pilonidal cysts are added to the picture, they all referred as tetrad. Dissecting cellulitis most commonly affects young black men with an average age of 18–40 years. Although its etiopathogenesis remains shrouded in mystery, *S. aureus*-induced neutrophilic response is the cause. It seems genetically transferred disease since familial cases have been reported [71, 72]. Initial lesion is generally follicular pustules at the vertex. Later, these are transformed into painful nodules. Accumulated nodules form tubular bridges. Seropurulent discharge can occur on nodules. Skin can be covered by crut and squams. Hypertrophic and keloid scar atrophy and cicatricial alopecia may occur in patients treated insufficiently. Although spontaneous remission occurs, relapse is frequent. Cervical and occipital lymphadenopathy may be observed [1, 3, 72].

In its histopathology, perifolliculitis formed by lymphocyte around follicles, histiocyte and polymorphous nuclear cells is present. Superficial fat tissue and abscess formation may be observed in dermis. Intensive fibrosis occurs in the late stages [71, 72].

Medical treatment of dissecting folliculitis involves high doses of isotretinoin. Antibiotics such as minocycline, tetracycline, erythromycin, clindamycin along with zinc sulfate, dapsone, colchicine, corticosteroids can be recommended as other treatment options. In persistent cases, surgical interventions such as excision and grafting may be considered [73–76].

4. Mixed cicatricial alopecias

4.1. Acne keloidalis

Acne keloidalis was first described in 1869 by Kohn. This disease is also known as folliculitis keloidalis nuchae or folliculitis keloidalis. The term acne keloidalis is somewhat of a misnomer since the condition is neither a kind of acne nor keloidal in nature. Acne keloidalis is most often seen in African Americans. The prevalence in American football players is 15%. Mechanical trauma, infections, autoimmunity, and drugs are the causes in its etiopathogenesis [76–78]. It is clinically developed in the early stages on the occipital scalp and on the nape of the neck as reddish-brown colored papules with smooth surface. Occasionally, these papules transformed into nodules and plaques. Pustules and abscesses are rarely occurred. Intrafollicular and perifollicular lymphocytes and plasma cell infiltrates are histopathologically involved in the early lesions. In the advanced stages, follicular destruction develops. Sebaceous glands disappear. Differential diagnosis should include folliculitis decalvans, acne necrotica, and dissecting folliculitis [1, 76].

First-line therapy of acne keloidalis is prevention. Preventing trauma and infections is important. In mild cases, combination of potent topical corticosteroids with topical antibiotics is advantageous. Oral tetracycline may be added to the monthly injections of intralesional corticosteroids. Partial response can be obtained by cryotherapy and laser therapy. Effective lasers include carbon dioxide, 1064 nm Nd:YAG or 810 nm diode. Surgical excision may be needed for common keloidal plaques. Surgical approaches include excision with primary closure or secondary intention healing. Deep surgical excision is more efficient. Surgical excision is an effective option [36, 79, 80].

4.2. Acne necrotica

Acne necrotica is also known as folliculitis necrotica. It is a mysterious disease not understood by many dermatologists. Infections are routinely mentioned in its etiology but evidence cannot be demonstrated. Drugs and food allergies are also thought to be the cause. Mechanical factors such as itching only spread the disease. Most of patients are women. Lesions are generally observed on the scalp along face and hairline. They are rarely occurred on nose and cheeks [4, 81, 82]. Initial lesions are umbilicated follicular papules. Not long after, they transformed into pustules. Consequently, varioliform scars may develop. Initially perivascular and perifollicular lymphocytic infiltrate, subepidermal edema is apparent. In the advanced stages, necrosis is observed in the follicular epithelium and epidermis [3, 81]. Neutrophiles can be seen in superficial dermis. Differential diagnosis should also include folliculitis decalvans, dissecting folliculitis, colitis, eczema herpeticum, and molluscum contagiosum [81, 82].

Treatment with oral tetracycline, antistaphylococcal antibiotics may be effective. They should be used in long term. In patients not having complete response, topical or intralesional corticosteroid can be added. Isotretinoin treatment may prolong the remission period [4, 43].

4.3. Erosive pustular dermatosis

The disease was first described in 1979 by Pye et al. and about 100 cases have been reported so far [83]. The disease most commonly occurs in elderly and females. Sun damage, local trauma (surgery, cryotherapy, herpes zoster), and autoimmunity are blamed in its etiology. Lesions with crusts and pustules on atrophic skin are clinically observed. The number of pustules can vary remarkably, and in some cases they are absent. Pain and pruritus in the lesions are not observed. However, cicatricial alopecia may develop in the advanced stages. Histopathology is uncharacteristic and not very helpful in confirming the diagnosis [84, 85]. Histopathological examination is crucial to exclude other diseases. Histopathology shows subcorneal pustules, epidermal atrophy, and erosions. In addition, these findings can be accompanied by a polymorphous dermal inflammatory infiltrate and in some cases leukocytoclastic vasculitis might be present. The differential diagnosis should consider tinea capitis, Gram-negative folliculitis, pyoderma gangrenosum, DLE, pemphigus vulgaris, and SCC. High-potency topical steroids reduce the inflammation significantly [1, 85].

Steroids must be used more than 6 months for better responses. Other treatment options include tacrolimus, dapsone, calcipotriol, and acitretin. Sun protection is reported to be effective since disease etiology includes actinic damage [86, 87].

5. Conclusion

Cicatricial alopecia forms an important group of disorders that end up with scarring and persistent hair loss. An elaborate physical examination, skin biopsies and blood tests can be helpful in order to establish the accurate diagnosis and to suggest the most appropriate treatment for the hair loss. Many patients do not respond to the first treatment they receive

and the condition frequently relapses when treatment is stopped. Some clinics offer surgical treatment, such as scalp reduction surgery and hair transplantation, but it may not be suitable for all patients. Patients often have significant psychosocial impact and management of these patients should address not only their physical but also psychological aspects.

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