Scarring Alopecia

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KEYWORDS

• Cicatricial alopecia • Hair loss • Ethnic hair • Traction alopecia
• Central centrifugal cicatricial alopecia • Discoid lupus erythematosus • Dissecting cellulitis
• Folliculitis decalvans

KEY POINTS

- Although the biochemical composition of human hair is remarkably similar, hair morphology does differ.
- African hair shaft and pigmented scalp have unique features that challenge diagnosis in scarring alopecia.
- The association of 2 or more hair disorders is common in Black patients. Therefore, it is imperative to understand their findings thoroughly to establish a good diagnosis.

BLACK SCALP AND AFRICAN HAIR SHAFT: THEIR DIFFERENCES AND PITFALLS IN DIAGNOSIS

Based on macroscopic characteristics, human hair has traditionally been classified into 3 major groups (African, Asian, and Caucasian).[1] Although the biochemical composition of hair from all 3 groups is remarkably similar; hair morphology does differ.[2] African hair shaft has been described as curlier, drier, and more susceptible to chemical and physical damage.[1,2] There are 4 types of African hair shaft recognized: straight, wavy, helical, and spiral-being the last the most frequent kind of hair. In addition, it seems elliptical in cross-section, with a high degree of irregularity in the diameter along the hair shaft, with frequent twists and random changes in direction.[1–3] It is common to observe knots, longitudinal fissures, and areas of breakage[2,3](**Fig. 1**). Overall, hair density in Black people may be less than in other ethnicities. For example, in a 4-mm-diameter punch, African American specimens showed 22 follicles on average, compared with the 36 follicles per 4-mm-diameter in Caucasians.[4] Moreover, studies have shown that African hair grows more slowly than Caucasian and Asian hair.[1,5] In Blacks, the hair follicle is curved and exits the epidermis at an oblique angle relative to the skin (**Fig. 2**). These factors could favor diseases such as pseudofolliculitis barbae.[3]

Similarly, patients with dark skin phototypes have unique trichoscopic patterns, sometimes challenging the diagnosis. A typical finding is a pigmented network that reflects the rete ridge melanocytes surrounding hypochromic areas of the suprapapillary epidermis.[6] Disruption of the pigmented network can be seen in conditions affecting the interfollicular skin, such as discoid lupus erythematosus, and in secondary scarring alopecias.[7] In addition, some inflammatory scalp disorders can lead to pigment incontinence appearing as blue-gray dots in the skin of color patients. Visualization of the acrosyringeal and follicular openings has been described as the "starry sky" pattern, that is, the presence of multiple small pinpoint white dots (0.2–0.3 mm) regularly distributed on the darker skin background.[6,8] Scarring alopecias can show an irregular distribution of these small pinpoint white dots, interconnected with irregular white patches representing follicular scarring.[7] Erythema is a common finding on the black scalp but vascular patterns are hard to see because of the overlying pigmented skin.

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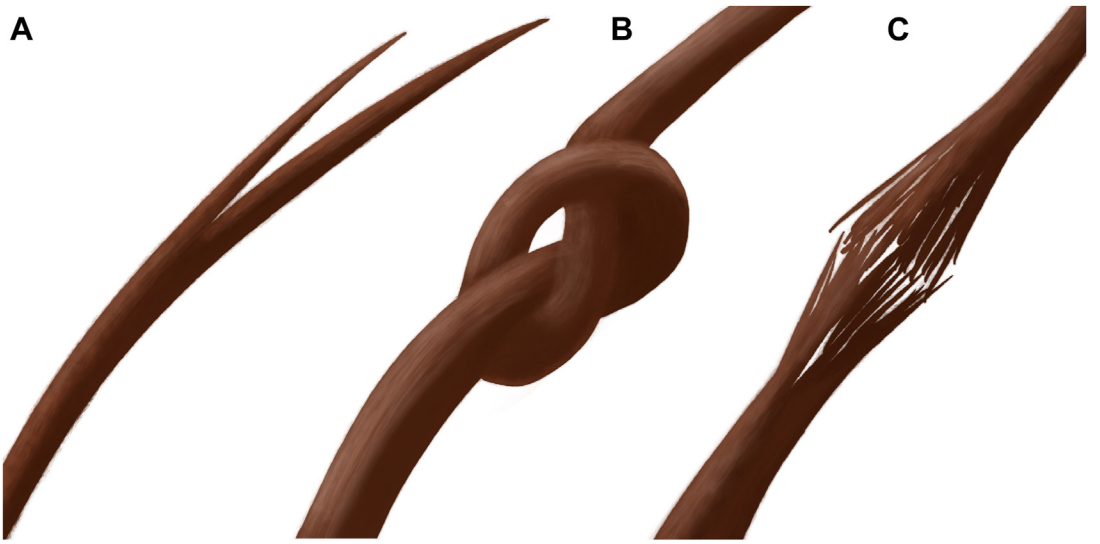

Fig. 1. Black hair shaft illustration showing longitudinal fissures (*A*), knots (*B*), and points of breakage (*C*). (Courtesy of J Larrondo, MD, MSc, Winston-Salem, NC.)

Moreover, clinical assessment could underestimate erythema in black scalp patients when facing inflammatory disorders.[6] Immersion fluid is sometimes helpful to enhance erythema in trichoscopy of skin of color patients (**Fig. 3**). Of note, the association of 2 or more hair disorders is common in Black patients.[2] Therefore, a thorough evaluation will help establish these diagnoses.

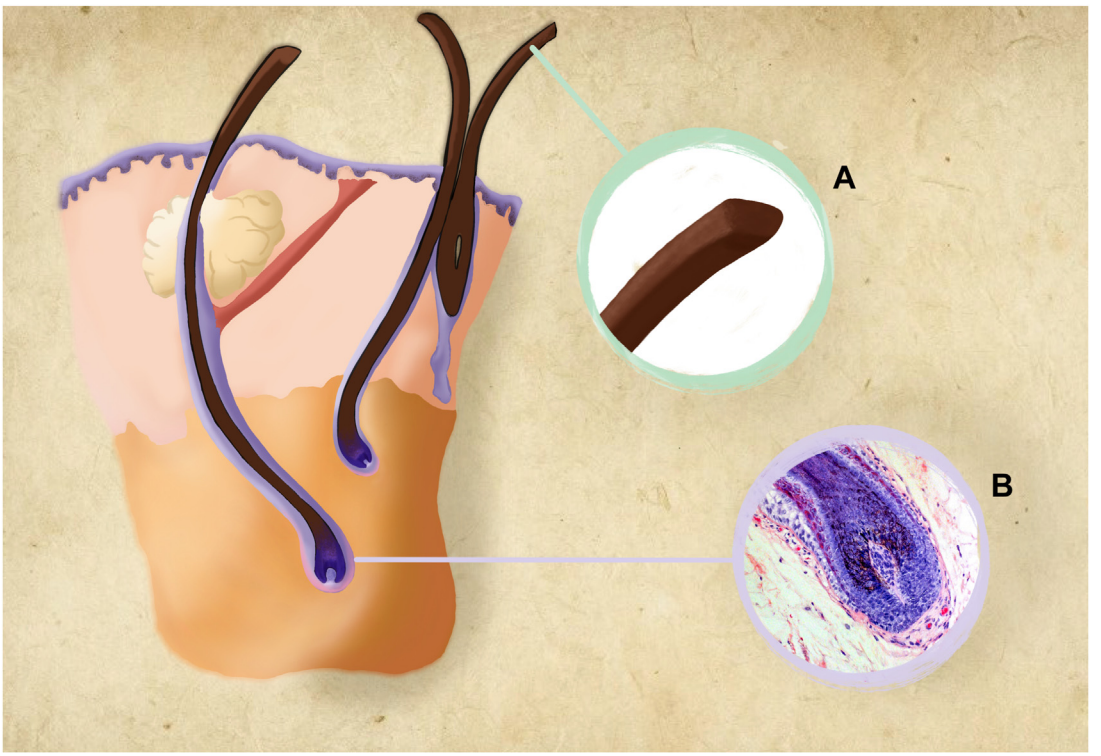

Fig. 2. Black hair follicle illustration showing flattened and elliptical hair shafts (*A*), with a marked hair bulb retrocurvature (*B*). (Courtesy of J Larrondo, MD, MSc, Winston-Salem, NC.)

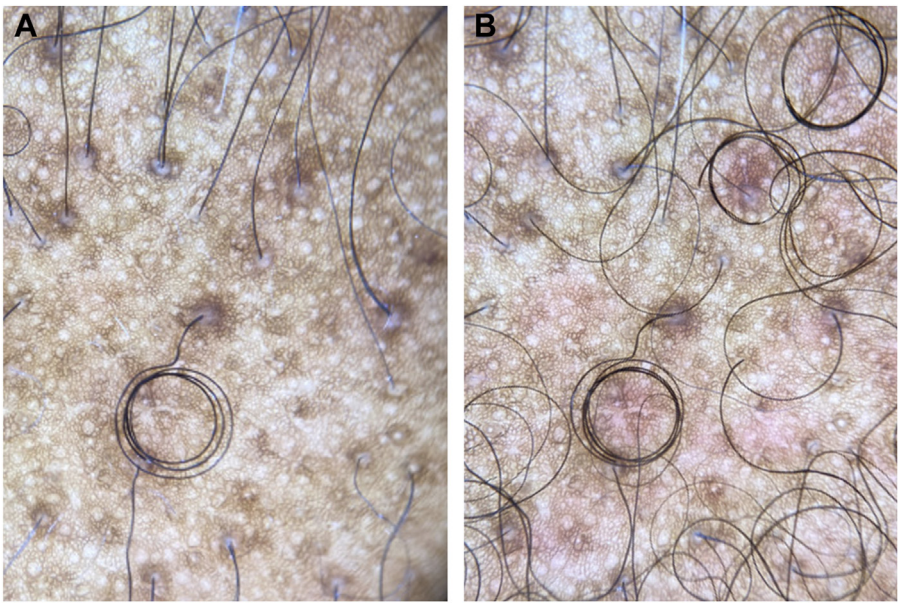

Fig. 3. Trichoscopic evaluation before (*A*) and after immersion fluid (*B*), enhancing erythema in a CCCA case.

TOPOGRAPHIC DIFFERENTIAL DIAGNOSIS IN SCARRING ALOPECIA

Frontal Scalp

Traction alopecia

Traction alopecia (TA) is a mechanical form of hair loss caused by prolonged or repetitive tension on the hair shaft. It has been reported across different ethnicities, affecting men, women, and children.[9,10] Moreover, TA is one of the most prevalent forms of alopecia in patients of color, affecting up to one-third of adult women of African descent.[10] High-tension hairstyling and the concomitant use of chemicals or heat may increase the risk.[11] TA is considered a biphasic form of hair loss, with the early disease being nonscarring and reversible; meanwhile, chronic disease issues scarring alopecia (**Table 1**).[12]

Clinical view

Hair loss may be limited to a minimally decreased hair density in the early stages. Perifollicular papules, erythema, or pustules can be found in areas of highest tension, usually asymptomatic.[11] However, the risk of TA increases with symptomatic traction from hairstyles, including pain, stinging, pustules, or crusting.[10] Hair loss can occur in any area depending on the hairstyle's configuration and the pressure-induced bulk.[13,14] It is also helpful to inquire about nocturnal hair-care practices because various techniques used to maintain hairstyles while sleeping can increase or induce traction.[14] Marginal TA, one of the most common patterns, affects the frontal and temporal scalp above the ears. It leaves a margin of vellus hairs marking the preexisting hairline, also known as the "fringe sign"[15] (**Fig. 4**A). Nonmarginal TA can occur anywhere throughout the scalp at the site of the installment of the hairstyles with tension. An example would be using volumizers such as volumizing scrunchies or clips in women wearing the hijab.[16] Linear, horseshoe, or stippled hair loss patterns should also alert the clinician about nonmarginal TA. Trichoscopy can show black dots, broken hairs at different levels, and follicular pustules in acute lesions.[11] The presence of hair casts is a sign of ongoing or persistent traction[6,11] (**Fig.5**). In chronic lesions, there is loss of follicular openings, pinpoint white dots irregularly distributed, white patches, and vellus hairs prevailing over terminal hairs[17] (**Fig. 4**B).

Histopathologic findings

In early TA, histopathology shows a usual number of terminal hairs with increased catagen/telogen count, preserved sebaceous glands, trichomalacia (distorted hair shafts), and pigmented casts.[12] Chronic TA shows a decrease in the total number of follicles with follicular dropout. The vellus follicles outnumber the terminal hairs, and sebaceous glands are preserved with minimal inflammation.[13]

Pitfalls and diagnostic clues

Acute forms of TA can mimic alopecia areata or trichotillomania. Chronic marginal TA may be confused with frontal fibrosing alopecia (FFA) or female pattern hair loss. **Table 1** summarizes the clinical characteristics of FFA and TA.

Table 1
Main findings of disorders in the frontal scalp

	Acute Traction Alopecia[11–17]	Chronic Traction Alopecia[11–17]	Frontal Fibrosing Alopecia[17,19–21]
Clinical presentation	Papules, pustules, or erythema in areas of tension. Fringe sign +	Decreased hair density in areas of traction. Increased vellus hairs. Fringe sign +	Pruritus. Hairline recession. Loss of follicular openings. Eyebrow involvement. Lonely hair sign +
Dermatoscopic features	Black dots, broken hairs, and follicular pustules. Hair casts.	Loss of follicular openings, white patches. Vellus > terminal hair. Hair casts.	Loss of follicular openings, absence of vellus, white patches. Perifollicular erythema, peripilar casts.
Histopathology	Normal number of terminal hairs. Increased catagen/telogen. Preserved sebaceous glands. Trichomalacia and pigmented casts.	Decreased number of terminal hairs with follicular dropout. Vellus > terminal hair. Preserved sebaceous glands. Minimal inflammation.	Perifollicular lichenoid lymphocytic infiltrate at the infundibulum and isthmus with concentric fibrosis. Diminished number of sebaceous glands.

Treatment

Education is a fundamental part of therapy. The clinician must be vigilant and proactive in the hair-care practices of patients who may be at risk. Mirmirani and Khumalo proposed 2 important slogans: "Tolerate pain from a hairstyle and risk hair loss" and "No braids or weaves on relaxed hair" as a general rule for patients to avoid high-risk hairstyling.[11] Minimizing chemical and thermal treatment is also recommended.[14] Medical treatment of early cases includes topical and intralesional corticosteroids applied to the affected areas and oral antibiotics for their anti-inflammatory properties.[11] Topical and oral minoxidil can also be considered in the therapeutic arsenal. Advanced stages of TA, characterized by scarring alopecia, are less likely to respond to medical therapy. Therefore, hair restoration surgery may be an ideal option for advanced cases.[12]

Frontal Fibrosing Alopecia

FFA is considered a variant of lichen planopilaris. Most patients are postmenopausal white women.[18] However, FFA can also affect Black patients, even with familial cases being reported, suggesting, among other factors, the possibility of genetic inheritance.[19] One study showed that Black patients with FFA may have higher rates of coexisting entities such as central centrifugal cicatricial alopecia (CCCA), systemic lupus erythematosus, and alopecia areata.[20] In addition, study data have shown that Black patients may present with earlier onset FFA than other ethnicities.[20,21]

Fig. 4. TA clinical presentation showing the "fringe sign" (*A*, *B*). Trichoscopy shows loss of follicular openings, pinpoint white dots irregularly distributed, and vellus predominance (*C*).

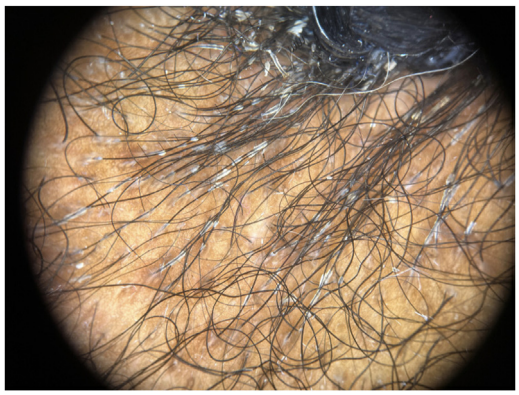

Fig. 5. Dermoscopy in TA. The presence of white to brown cylindrical structures (hair casts) along the hair shaft is a sign of active traction.

Clinical view

Hairline recession is a typical characteristic of FFA and occurs more frequently on the frontotemporal region of the scalp but upper periauricular and occipital localizations are not uncommon.[20,21] The band of alopecia is often readily distinguishable from the sun-damaged skin of the forehead. Alternatively, one may see a lighter band of skin behind where the hairline began that can be highlighted using Wood's light.[22] Black patients are more likely to have vertex/central and occipital scalp involvement.[20] The itch is a common complaint among skin of color patients with FFA; pain or a burning sensation is often reported. Patchy or complete eyebrow involvement and the presence of the "lonely hair sign" are often clues to diagnosis[23] (**Fig. 6**A). Compared with White patients, higher rates of facial hyperpigmentation and less perifollicular hyperkeratosis/scales are found.[20] Trichoscopy typically shows loss of follicular openings, absence of vellus hairs in the hairline, preserved honeycomb pigmentary network, and white patches. Perifollicular erythema and peripilar casts can also be seen[17] (**Fig. 6**B).

Histopathologic findings

Histologic features may be indistinguishable from lichen planopilaris (LPP) on hematoxylin and eosin sections. Typically, lichenoid interface dermatitis affects the infundibulum/isthmus and concentric perifollicular fibrosis with a partial or total absence of sebaceous glands.[24] FFA sometimes can show a more pronounced follicular apoptosis and clefting between the follicular epithelium and the fibrosis zone compared with LPP.[25]

Pitfalls and diagnostic clues

FFA can be misdiagnosed in Black patients due to overlapping features with TA. Literature reports have shown that many black patients with FFA had a history of hairstyles associated with traction and chemical or heat to straighten the hair.[21] In addition, some of those patients also showed signs of TA, making the diagnosis difficult (see **Table 1**).

Treatment

The goals of treatment are to stop disease progression and provide symptomatic relief. Topical treatments include topical minoxidil, corticosteroids, or topical calcineurin inhibitors. Systemic anti-inflammatory therapies using drugs such as hydroxychloroquine, doxycycline, oral corticosteroids, oral retinoids, and mycophenolate mofetil were reported to be useful.[26,27] 5-alpha reductase inhibitors (finasteride and dutasteride) seem to be effective in stabilizing disease progression,[28] with oral dutasteride being the most effective therapy for patients with FFA compared with other systemic therapies (hydroxychloroquine, doxycycline, and isotretinoin) or no systemic treatment in 224 patients.[29] The most effective regimen was 5 to 7 capsules of dutasteride 0.5 mg/wk.[29] The use of Nd: YAG (1064 nm), nonablative, once a month for 3 months (14 J/cm^2, spot size 5 mm, pulse duration 3 milliseconds) in 5 patients as an adjuvant therapy has also been reported. Four out of five patients reported improvement in at least one symptom (pain, pruritus, burning) and improvement in at least 3 out of 8 clinician-evaluated signs. One patient had worsening of pain. In addition, 2 patients with lichen planus pigmentosus of the face improved after treatment. No major side effects were reported.[30]

MIDDLE SCALP

Central Centrifugal Cicatricial Alopecia

CCCA remains the leading cause of scarring alopecia in women of African descent. It predominantly affects middle-aged women of African ancestry, with a prevalence of 2.7% to 5.7% in that population.[31] Hairstyles and hair-care practices have long been suspected of the development of CCCA but the available evidence is conflicting.[32] Mutations in the PADI3 gene have been identified in women with CCCA,[33] and it is uncommon in men and children (**Table 2**).[34,35]

Clinical view

Symptoms can range from pruritus, tenderness, dysesthesias, or burning.[31] However, many patients are asymptomatic,[36] making a silent and insidious progression and leading to late presentation of the patient for medical care. CCCA commonly presents scarring on the vertex or crown that spreads centrifugally, often symmetrically (**Figs. 7A and 8**A). The severity of central

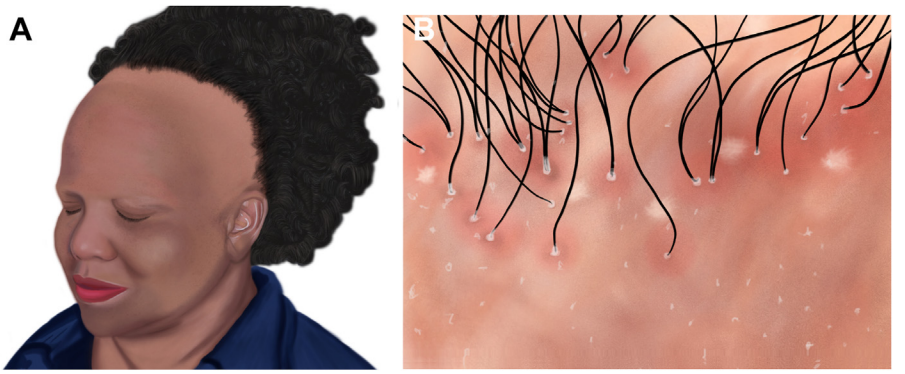

Fig. 6. FFA illustration showing frontotemporal hairline recession and complete eyebrow involvement (*A*). Trichoscopic illustration shows the absence of vellus hairs, peripilar casts, perifollicular erythema, and white patches (*B*). (Courtesy of J Larrondo, MD, MSc, Winston-Salem, NC.)

Table 2
Main features of scarring alopecia in the middle scalp

	Clinical Presentation	Dermatoscopic Features	Histopathology
CCCA[17,31,39]	Pruritus, tenderness, dysesthesias, or burning. Vertex or crown scarring with centrifugal expansion. Hair breakage.	Loss of follicular openings, irregularly distributed pinpoint white dots, white patches, and hair diameter variability. Peripilar gray/white halos.	PDIRS in affected and unaffected follicles. Follicular miniaturization. Varying degrees of perifollicular lymphocytic inflammation, and concentric fibrosis.
Lichen planopilaris[7,17,50,53]	Pruritus, burning, or pain. Focal, multifocal, or diffuse areas of scarring alopecia.	Loss of follicular openings, peripilar casts, small hair tufts, and white patches. Blue-gray dots in a targetoid pattern.	Perifollicular lymphocytic infiltrate around infundibulum and isthmus. Absent/diminished sebaceous glands. Concentric perifollicular fibrosis.
Discoid lupus erythematosus[7,17,55,74]	Burning, pruritus, or tenderness. Central erythema or hypopigmentation and peripheral hyperpigmentation. Follicular plugging.	Loss of follicular openings, perifollicular hyperkeratosis, keratotic plugs, red dots, and large arborizing vessels. Blue-gray dots in a speckled distribution.	Vacuolar interface dermatitis. Superficial and deep perivascular and periadnexal lymphocytic infiltrate. Diffuse deposition of mucin in the dermis and subdermis.
Fibrosing alopecia in a pattern distribution[61,62]	Pruritus, pain, or dysesthesia. Hair loss in a male or female pattern distribution, usually with a slowly progressive course.	Loss of follicular openings, peripilar casts, perifollicular erythema. Hair shaft variability and predominance of single hair follicles.	Lichenoid inflammation affecting single terminal and vellus follicles. Hair follicle miniaturization. Perifollicular lamellar fibrosis.

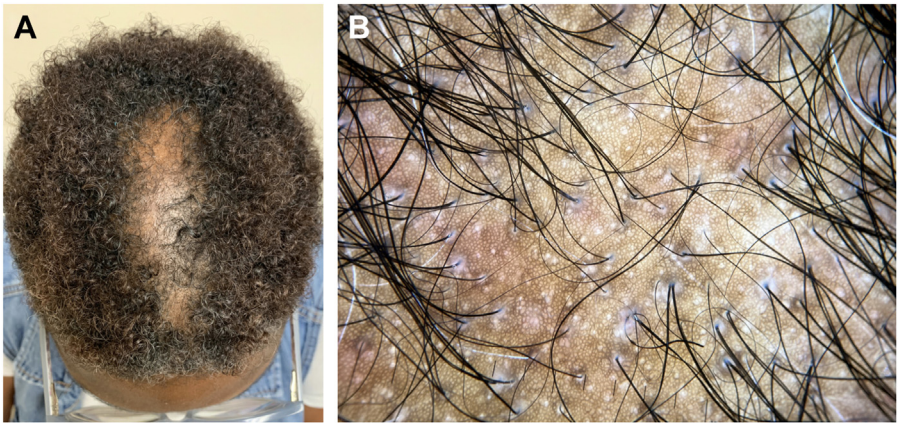

Fig. 7. CCCA clinical presentation (*A*). Trichoscopy shows peripilar gray/white halos, irregularly distributed pinpoint white dots, and hair diameter variation (*B*).

hair loss is graded according to a previously validated photographic scale[37] (**Fig. 9**). Hair loss can be associated with scaling, crusting, follicular papules, or pustules. In addition, hair breakage has been reported as a possible early clinical presentation.[36] Trichoscopic features include the loss of follicular openings and irregularly distributed pinpoint white dots/white patches. A preserved honeycomb pigmentary network, variation in hair diameter, perifollicular erythema, and occasionally black dots and broken hairs.[17] In addition, the presence of peripilar gray/white halos is a specific and sensitive sign of the diagnosis[38] (**Figs. 7B and 8**B).

Histopathologic findings

The earliest histologic finding is the premature desquamation of the inner root sheath (PDIRS).[39] However, PDIRS is a nonspecific feature in heavily inflamed follicles in other primary cicatricial alopecias.[40] In CCCA, PDIRS can be detected in affected and unaffected follicles. Variably dense lymphocytic perifollicular inflammation at the infundibulum and isthmus with concentric onion-like follicular fibrosis, fragmented hair shafts in the dermis, follicular miniaturization, and focal preservation of the sebaceous glands are common findings.[39]

Pitfalls and diagnostic clues

Histopathologically, LPP remains the principal differential diagnosis. The absence of follicular apoptosis and only mild/absent lichenoid inflammation favors CCCA over lichen planopilaris.[25] LPP is unlikely to show PDIRS in noninflamed follicles.[39] However, in some cases, the inflammatory infiltrates in CCCA and LPP are not only histologically similar but also immunophenotypically

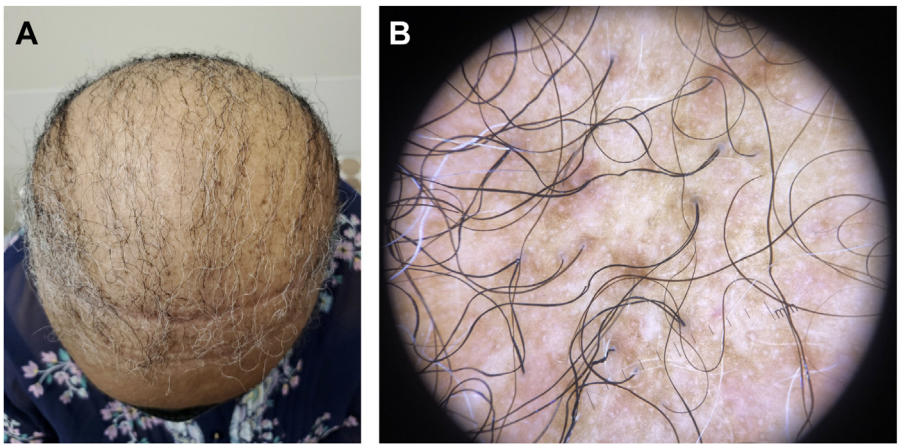

Fig. 8. Severe CCCA with extensive scalp involvement (*A*). Trichoscopy shows loss of follicular openings, peripilar white/gray halos, irregularly distributed pinpoint white dots, and white patches (*B*).

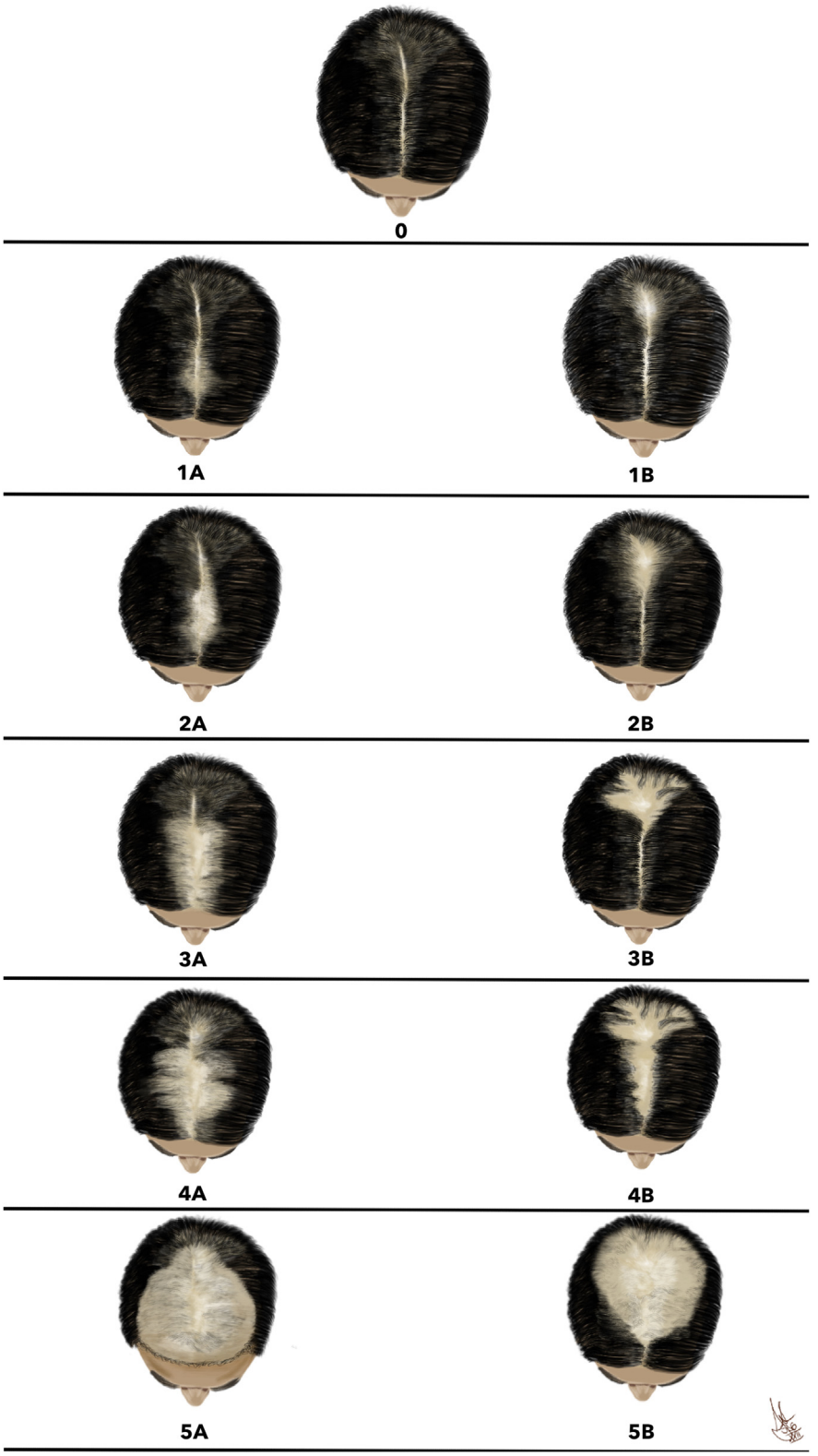

Fig. 9. Illustration adapted from the central scalp alopecia photographic scale in African American women. (*From* Olsen E, Callender V, Sperling L, et al. Central scalp alopecia photographic scale in African American women. Dermatol Ther 2008;21(4):264 to 7.)

indistinguishable.[41] **Table 2** summarizes the main features of scarring alopecia in the middle scalp.

Treatment

The treatment goals in CCCA are to halt the disease progression, control symptoms, and hopefully establish some hair regrowth. Despite the lack of solid evidence directly associating hair-care practices, the removal of potentially harmful hair-care practices is encouraged; minimal trauma and infrequent use of hair chemicals and heat are recommended by some clinicians.[42,43] The use of antiseborrheic shampoos may help decrease pruritus and scaling. Many researchers have reported treating with topical and intralesional corticosteroids, calcineurin inhibitors, and minoxidil.[31] Topical treatments are commonly used daily, with topical corticosteroids usually tapered to 3 days/wk once control of symptoms is achieved.[43] Intralesional corticosteroids ranging in strength from 2.5 to 10 mg/mL may be used every 4 to 8 weeks for at least 6 months.[44,45] The target area of treatment should be at the periphery of areas of hair loss, including normal appearing areas to prevent the progression. Dermoscopy can be a helpful tool to assess clinically unapparent areas of activity.[46] Systemic anti-inflammatory therapies include oral tetracyclines, antimalarials, mycophenolate mofetil, and cyclosporine. The usual regimen is 6 to 9 months for active inflammation or where topical treatment has been unsuccessful. In addition, antiandrogen and 5-alfa-reductase inhibitors have been used with success.[43] Research has highlighted the efficacy of platelet-rich plasma (PRP) therapy in patients with refractory CCCA. However, the investigators also noted a reduction in the follicular density 6 months after treatment, supporting the need for maintenance therapy.[47] Another report of 2 stabilized cases of CCCA found an increase in hair density during the monthly PRP sessions. Nevertheless, during the 6-month-interval sessions, both patients showed a noticeable decrease in follicular density.[48] An expert opinion states that PRP may be more advantageous in women with concurrent CCCA and androgenetic alopecia.[42] Surgical correction via hair transplantation is a possible option for patients with a stabilized condition for 9 to 12 months and the absence of inflammation histologically.[31]

Lichen planopilaris

A chronic lymphocytic disorder that usually affects the scalp but may also compromise the hair on the face and body. Most patients are caucasian women in their early fifties[49] but it can occur in men and women of all racial groups, including individuals of color.

Clinical view

LPP presents with focal or multifocal patches or diffuse areas of scarring alopecia on the vertex or parietal scalp, often quite symptomatic with pruritus, burning, and tenderness[50] (**Fig. 10**A). Disease can be indolent or slowly progressive but rarely involves the entire scalp. Less typical pigmentary findings in Black patients include hypopigmented macules and patches of varying sizes on the scalp, face, and trunk.[51] Moreover, reticulated hyperpigmentation on the scalp has been reported.[52] Trichoscopic features show loss of follicular openings, peripilar casts, and a preserved honeycomb pattern. Occasionally, there are blue-gray dots in a targetoid pattern corresponding to pigment incontinence.[7] Small, irregularly shaped, whitish areas lacking follicular openings and small hair tufts can be seen[17] (**Fig. 10**B).

Histopathologic findings

LPP shows areas of follicular dropout and an absent/diminished number of sebaceous glands. Perifollicular lichenoid/interface lymphocytic

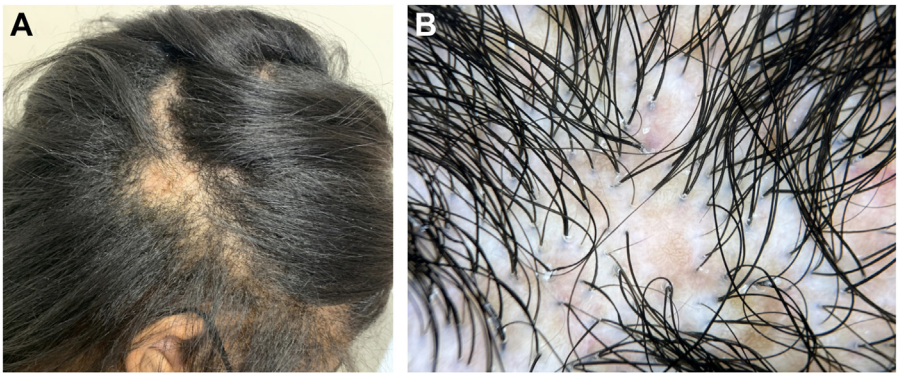

Fig. 10. LPP presenting as a focal patch of scarring alopecia (*A*). Trichoscopy shows loss of follicular openings, peripilar casts, and small hair tufts (*B*).

infiltrate involving the infundibulum and isthmus, and concentric fibrosis involving the permanent portion of the hair follicle may be observed.[53]

Pitfalls and Diagnostic Clues

LPP differs from discoid lupus erythematosus (DLE) by the presence of follicular hypergranulosis, diminished elastic fibers in a wedge shape around the infundibulum, and more frequent colloid bodies.[54]

Treatment

The treatment goals include controlling symptomatology and halting the progression of the disease. High-potency topical corticosteroids, calcineurin inhibitors, and intralesional corticosteroids can be used to target areas of active disease and are the mainstay of treatment of primary symptoms.[55] Additional therapies may be used based on the amount of inflammation and/or involvement, as well as the velocity of progression. These include oral tetracyclines, hydroxychloroquine, mycophenolate mofetil, cyclosporine, azathioprine, dapsone, and isotretinoin.[56] Limited evidence supports the therapeutic potential of Janus kinase (JAK)-1/2 and JAK-1/3 inhibitors for treating recalcitrant LPP.[57,58] Other adjunctive therapy includes low-level light therapy[59] and platelet-rich plasma,[60] awaiting placebo-controlled trials to understand their benefits entirely.

Fibrosing Alopecia in a Pattern Distribution

Fibrosing alopecia in a pattern distribution (FAPD) is a progressive form of scarring alopecia characterized by patterned hair loss similar to androgenetic alopecia but with trichoscopic and histopathologic signs of both lichen planopilaris and androgenetic alopecia.[61] This form of hair loss has been described in Caucasians, patients of African descent, and Hispanics.[62] There are no prevalence data for this form of hair loss in Black patients, which is not well represented in the literature.

Clinical view

FAPD presents with hair loss in a centroparietal distribution, usually with a slowly progressive course. Typically resembles male or female pattern alopecia. A closer look may reveal the pattern of "pink goosebumps" in the patterned area.[63] Some patients may complain of pain, dysesthesia, and scalp pruritus.[61] Trichoscopy is fundamental for suspecting the diagnosis. FAPD shows trichoscopic signs seen in androgenetic alopecia, such as hair diameter variability and predominance of single hair follicles.[61] In addition, features such as loss of follicular openings, peripilar casts, and perifollicular erythema are seen.[61,63] In higher phototypes, similar features found in CCCA, such as peripilar white halos, a honeycomb pigmented network, and scattered small white patches, have been described, making diagnosis challenging.[61,62]

Histopathologic findings

FAPD shows combined features of both LPP and androgenetic alopecia (AGA). Histopathology shows an increased number of vellus hairs, concentric perifollicular lamellar fibrosis, and a decrease in sebaceous glands. Variably dense, perifollicular lymphocytic infiltrate is also seen, and both terminal and vellus follicles are affected.[61,63]

Pitfalls and diagnostic clues

In the author's opinion, CCCA and FAPD may sometimes be indistinguishable in skin of color. One distinction would be the presence of a lichenoid infiltrate and an interface dermatitis of the follicular epithelium in FAPD.[61] However, although uncommon, CCCA can also show mild follicular lichenoid inflammation and follicular apoptosis in active stages. In addition, early-stage CCCA that presents hair thinning may be mistaken for AGA.[64]

Treatment

FAPD is a chronic and progressive scarring condition, and the treatment goal, as in other scarring alopecias, is to stop the disease progression. Therapy aims to decrease inflammation and block miniaturization. Anti-inflammatory agents include topical, intralesional corticosteroids, and oral hydroxychloroquine.[61] Hair growth promoters such as topical minoxidil 5% or oral minoxidil can be used as adjunctive treatments.[65,66] Antiandrogen therapy and 1 mg daily of oral finasteride have been shown to stabilize the progression of hair loss.[67]

Discoid lupus erythematosus

DLE is a form of chronic lupus erythematosus that commonly affects the scalp, and about one-third of cases are associated with scarring alopecia.[68] DLE has a higher incidence and prevalence in Black individuals.[69] Women are more affected than men, and the disease is more common in adults than children.[70]

Clinical view

Patients may be asymptomatic or describe burning, pruritus, or tenderness in affected areas. In addition, patients may report worsening after ultraviolet (UV) light exposure.[70] Areas of involvement typically include the scalp but it may occur on any other body part, especially in sun-

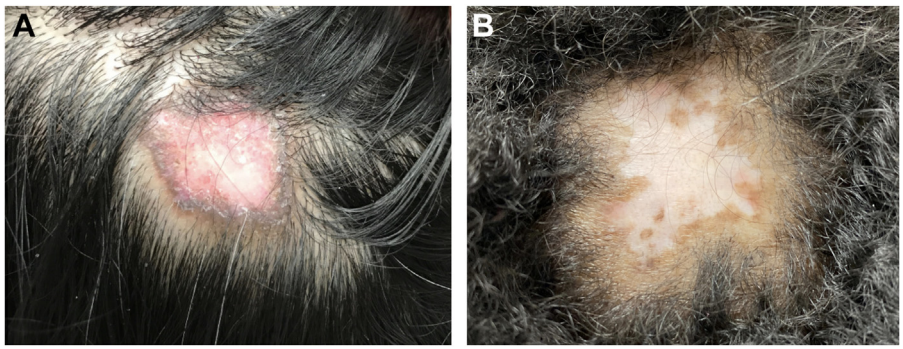

Fig. 11. An active DLE case shows a well-demarcated erythematous patch with scales and peripheral hyperpigmentation (*A*). A well-established patch of alopecia with central hypopigmentation and mild erythema (*B*).

exposed areas. Erythema, scale, and pigmentary changes are more pronounced in DLE than in other forms of cicatricial alopecia.[71] It usually presents as well-demarcated annular lesions with central hypopigmentation and/or erythema, follicular plugging, and peripheral hyperpigmentation (**Fig. 11** A and B). Of note, DLE cases presenting as hyperpigmented patches on the scalp without evident hair loss have also been described.[72] Trichoscopic features include loss of follicular openings, perifollicular hyperkeratosis, interfollicular scales, keratotic plugs, follicular red dots, and large arborizing vessels (**Fig. 12**). In higher phototypes, the loss of pigmentation with disruption of the honeycomb pattern, white patches or a reduction/absence of pinpoint white dots, blue-gray dots distributed in a speckled pattern, and blue-white veil-like features can be appreciated.[7,73]

Histopathologic findings

In DLE, vertical and horizontal sections are helpful for the diagnosis. DLE lesions typically show an interface dermatitis with vacuolar degeneration involving the dermo–epidermal junction, epidermal atrophy with hyperkeratosis, follicular plugging, a diminished number of sebaceous lobules, pigment incontinence, and thickening of the basement membrane.[74] In addition, the presence of mucin in the dermis and subdermis in a diffuse pattern and an infiltrate of plasma cells in a perivascular and periadnexal location are strong pointers to DLE.[75]

Pitfalls and diagnostic clues

Folliculitis decalvans (FD), psoriasis, inflammatory tinea capitis, and especially LPP are differential diagnoses of DLE. In histopathologically inconclusive cases, direct immunofluorescence (DIF) can help establish the diagnosis. In DLE, DIF may show immunoglobulin G (IgG) and complement C3 (C3) along the dermo-epidermal junction in 70% to 95% of cases.[54] Another finding that may help to distinguish alopecic DLE from LPP is the presence of groups of CD123+ plasmacytoid dendritic cells (clusters of at least 5 cells) in discoid lupus, whereas in LPP these are arranged as single interstitial cells.[76]

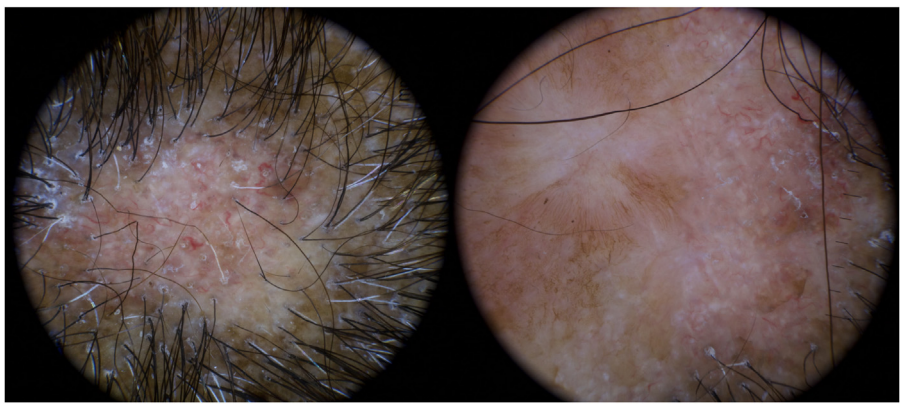

Fig. 12. Trichoscopy of DLE shows loss of follicular openings, pigmentary network disruption, large arborizing vessels, perifollicular hyperkeratosis, and white patches.

Table 3
Main characteristics of disorders in the posterior scalp

	FD[7,55,80]	Dissecting cellulitis[7,55,91]	Acne Keloidalis nuchae[8,55,100]
Clinical presentation	Pain, pruritus, and burning sensation. Purulent folliculitis with scales, erythema, and crusts. Hair tufting.	Pain or tenderness. Multiple boggy scalp nodules, abscesses, and sinus tracts.	Fibrotic papules and pustules. Nodules or plaques of scarring. Hair tufting.
Dermatoscopic features	Loss of follicular openings, peripilar casts, scaling, pustules, and hair tufting.	Black dots, broken hairs, 3D yellow dots, and short regrowing hairs. Milky-red areas and cutaneous clefts.	Broken hairs Ingrown hairs Hair tufting
Histopathology	Mixed inflammatory infiltrate (neutrophils, lymphocytes, histiocytes, and plasma cells). Polytrichia (4–6 follicles), and fragmented hair shafts. Perifollicular concentric fibrosis.	Mixed cell infiltrate affecting the lower follicle. Increased telogen count, trichomalacia. Late-onset disease: Diminished or absent sebaceous glands, follicular dropout, chronic granulomatous infiltrate, fragmented hair shafts and dermal fibrosis.	Dense infiltrate of neutrophils, lymphocytes, and plasma cells distributed around the isthmus and the lower infundibulum. Late-onset disease: Chronic granulomatous inflammation, follicular dropout, fragmented hair shafts, and dense dermal fibrosis without keloidal features.

Treatment

Prompt diagnosis and early therapy are crucial to prevent irreversible hair loss. The general recommendation includes photoprotection and smoking cessation.[72] Early DLE may be managed with topical and intralesional corticosteroids (triamcinolone acetonide 10 mg/mL every 4–6 weeks). Topical calcineurin inhibitors can also be helpful, especially for areas with thinning or atrophy.[77] Systemic agents include oral antimalarial agents, mycophenolate mofetil, methotrexate, retinoids, dapsone, and thalidomide.[3,77] In addition, oral corticosteroids (prednisone 10–20 mg/d, tapering down over time, or dexamethasone as a minipulse of 0.1 mg/kg on 2 consecutive days or the week) can be used as bridging therapy.[1,72]

Posterior Scalp

Folliculitis decalvans

FD is a highly inflammatory neutrophilic cicatricial alopecia characterized by chronic inflammation, hair tufting, and follicular destruction. It is more frequent in young and middle-aged adults.[55] Cause has not been completely elucidated. An alteration in the host immune response may play a role in the onset of the disease, triggered by a dysbiosis of normal hair microbiota (**Table 3**).[78,79]

Clinical view

Occipital and vertex are the main affected areas. Lesions are often symptomatic with pain, pruritus, and burning sensation. Typical lesions include purulent folliculitis with tufts of hairs, scales, erythema, and perifollicular crusts[80] (**Fig. 13** A and B). Patients may also present concomitant features of LPP, calling this variant FD lichen planopilaris phenotypic spectrum. Some authors consider this a continuum in the evolution from neutrophilic inflammation to chronic lymphoid-plasmacytic inflammation.[81] Trichoscopy typically shows hair tufting—6 or more hairs emerging together—which corresponds to the fused outer root sheaths at the infundibulum level, usually surrounded by a band of yellowish scales.[8] Other findings include focal disruption of the interfollicular pigmentary network, irregular white patches, crusts, scaling,

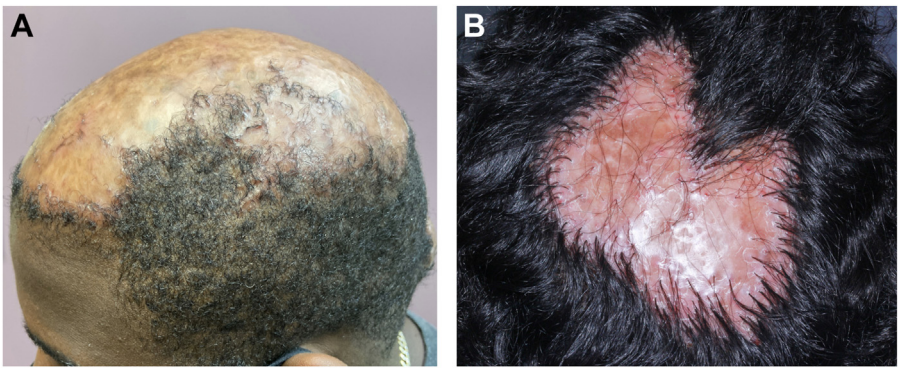

Fig. 13. FD clinical presentation. (*A*) A severe case with extensive scarring, scaling, and erythema. (*B*) Hair tufts, scaling, and perifollicular crusts.

follicular pustules, and elongated loop-like and coiled vessels in a concentric perifollicular arrangement[80,82] (**Fig. 14**).

Histopathologic findings

Histologic findings include intense inflammation around the upper portion of affected follicles, especially at the level of the lower infundibulum.[83] Early lesions include intrafollicular and perifollicular neutrophilic infiltrate, loss of sebaceous glands, and fused outer root sheaths of 4 to 6 follicles at the infundibulum, causing polytrichia. Advanced lesions show a mixed cell inflammatory infiltrate, including neutrophils, lymphocytes, histiocytes, and plasma cells.[80] Hair shaft granulomas with foreign-body giant cells and follicular and interstitial dermal fibrosis can be found.[55]

Pitfalls and diagnostic clues

Very active forms of lymphocytic cicatricial alopecia are in the differential diagnosis and can mimic the FD pattern. A biopsy from a pustular or papular area is less likely to provide useful histopathological information[83] than a sample guided by dermoscopy. A helpful place for biopsy would be a hair tufting area surrounded by thick white, yellowish scales.[84]

Treatment

FD can be an aggressive, resistant, and relapsing disorder making treatment challenging. Due to the host immune response triggered by an altered microbiota, eradicating infectious agents, specifically *Staphylococcus aureus*, has been the mainstay therapy.[55] However, the presence of biofilms could explain, in part, the chronicity and recurrences after appropriate antibiotic treatments. One study isolated gram-negative bacteria in 11 out of 34 FD cases.[79] Therefore, bacterial cultures with antibiotic sensitivities should be obtained in every case. Different regimens have been recommended to reduce the bacterial load of staphylococci effectively. The combination of clindamycin 300 mg and rifampicin 300 mg twice daily systemically for a 10-week course has been commonly used, achieving the most prolonged remission. Other useful treatment regimens include oral doxycycline 100 mg daily for 3 to 6 months, oral minocycline 100 mg daily for 3 to 6 months, and oral azithromycin 500 mg 3 times

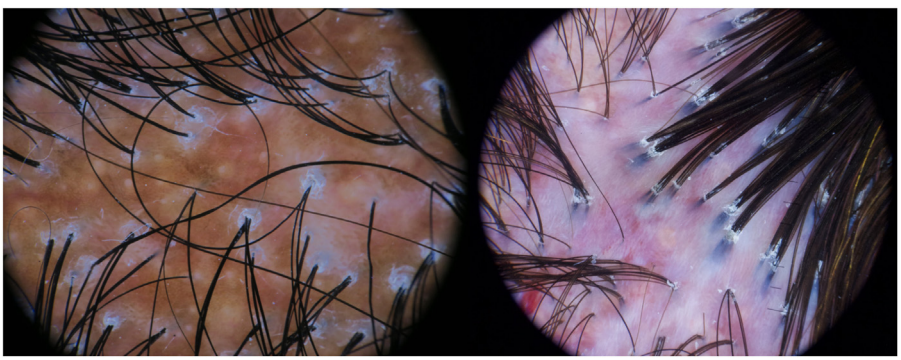

Fig. 14. Trichoscopy in FD shows perifollicular hyperkeratosis, hair tufting, and irregular white patches.

a week for 3 months.[85] Iorizzo M and colleagues reported the successful use of adalimumab in 23 FD refractory cases; 2 patients discontinued the treatment because of insufficient improvement. The regimen was 160 mg at week 0, 80 mg at week 2, and 80 mg every other week.[86] Topical antibiotics and intralesional corticosteroids can be combined with oral regimens. Intralesional triamcinolone acetonide (10 mg/mL) injected into the surrounding hair areas every 4 to 6 weeks can help slow the progression and reduce symptoms.[55] Other therapies include external beam radiation, isotretinoin, human immunoglobulin, infliximab, PRP, and photodynamic therapy.[87–89]

Dissecting cellulitis

Dissecting cellulitis of the scalp (DCS) is a neutrophilic cicatricial alopecia, considered a part of the follicular occlusion disorders that may progressively lead to scarring alopecia and significant morbidity.[55] This occlusion causes a buildup of keratin and other cellular debris, leading to rupture and exposure of the inner follicle and dermis to the environment, with the subsequent inflammatory response. It is more commonly seen in young men of African or Hispanic descent, although it can also occur in Caucasians, women, and children.[55,90]

Clinical view

Occipital and vertex are usually affected. The inflammatory lesions begin as multiple, tender, boggy scalp nodules and sterile abscesses on the occipital scalp. Multifocal lesions can merge to form cerebriform ridges. Lesions often interconnect, forming sinus tracts with overlying permanent alopecia.[55,91] High-frequency ultrasound has been used to characterize inflammatory lesions in DCS[92] (**Fig. 15**). The lesions are sterile; however, secondary bacterial infection may occur most commonly with coagulase-negative staphylococci.[91] Trichoscopic findings on early stage lesions typically show features of noncicatricial alopecia with regularly distributed pinpoint white dots, enlarged plugged follicular openings (3D yellow dots), black dots, broken hairs, and short regrowing hairs.[17] Inflammatory areas can show erythema, yellow/violaceous structureless areas, arborizing vessels, and giant capillaries. End-stage lesions show loss of follicular openings, confluent ivory-white areas, and cutaneous clefts containing hair shafts.[7,91]

Histopathologic findings

Early lesions show a dense mixed inflammatory infiltrate surrounding the lower half portion of the follicle. The infiltrate may contain individual giant cells or collections of epithelioid cells. There is an increased catagen/telogen hair count, dilated infundibula plugged with keratin and sebum, and trichomalacia. Long-standing lesions can show chronic granulomatous infiltration with sinus tracts and fragmented dermal and subdermal hair shafts in the dermis and subdermis. Partial to complete

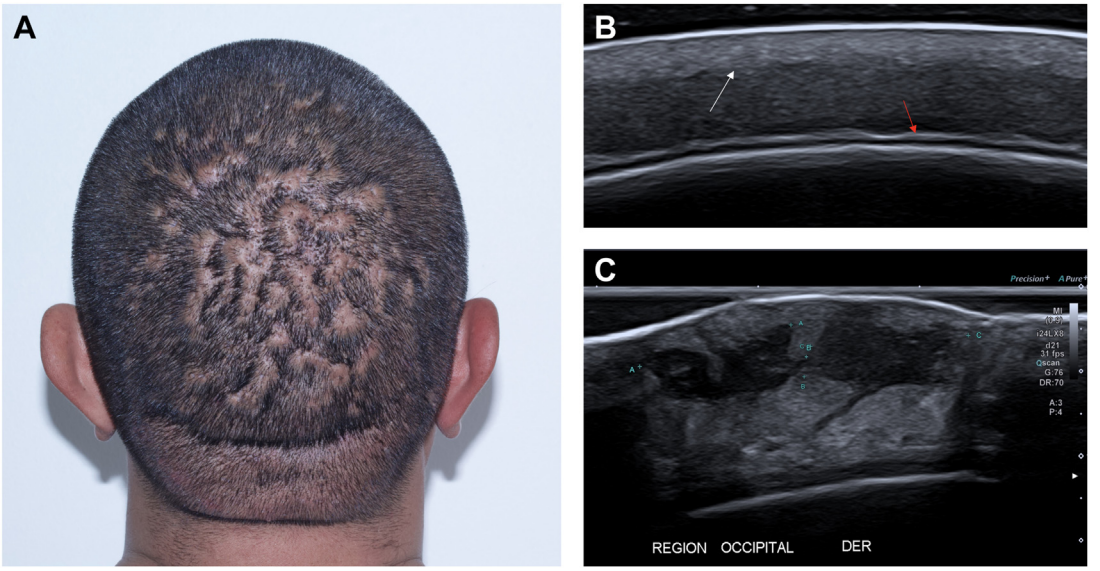

Fig. 15. DCS clinical presentation (*A*). (B) A healthy scalp ultrasound with typical thickness and echogenicity. The *white arrow* shows the hypodermal interface; the *red arrow* indicates epicranial aponeurosis. DCS ultrasound shows thickening of the dermis and subcutaneous planes, with loss of definition of the dermo–hypodermal interface, increased echogenicity of subcutaneous adipose tissue, and presence of confluent cystic lesions with the appearance of merging pseudocysts (*C*). *Courtesy* of C Whittle, MD, Santiago, Chile.

loss of sebaceous glands, follicular dropout, and dermal fibrosis may also be present.[91,93]

Pitfalls and diagnostic clues

Early DSC lesions can be patchy emulating alopecia areata in their clinical and trichoscopic findings. Therefore, a scalp biopsy can help establish the diagnosis. A good place for biopsy should include features such as the 3D yellow dots and any areas with black dots.[91] DSC sometimes can clinically and histologically mimic inflammatory tinea capitis. Therefore, a complete workup that includes trichoscopy, histology, and fungal culture should be performed to rule out fungal infections.[94]

Treatment

DCS treatment is often challenging. For limited disease or milder presentation, intralesional corticosteroids and oral antibiotics (tetracyclines, cloxacillin, erythromycin, cephalosporin, and clindamycin) are the standards of care.[55] Incision and drainage is often common step in treating abscesses and fluctuant nodules.[95] In moderate-to-severe cases, oral isotretinoin (0,25–1 mg/kg) has been suggested.[90,96] Secondary line treatments include antitumor necrosis factor α agents, most commonly adalimumab. Oral corticosteroids can be used as adjunctive therapy at low doses or as a bridge to more definitive treatments.[96] For refractory cases, destruction of the hair follicle with CO_2, long-pulse non-Q-switched ruby, and 800-nm pulsed diode have been reported. Other options include oral dapsone, zinc sulfate, colchicine, and photodynamic therapy.[55] Surgical treatment has not been well established. Staged excisions of sinus tracts can help achieve control in refractory patients with draining nodules and sinus tracts without full scalp involvement.[97]

Acne Keloidalis Nuchae

Acne keloidalis nuchae (AKN) is a chronic idiopathic inflammatory scarring condition that mainly occurs in men of African descent.[55] It has been infrequently reported in Caucasians and other ethnic groups.[98] Pathophysiology is not fully understood. Contributing factors involve androgens, autoimmunity, infection, trauma, genetics, and ingrown hairs causing a foreign-body-like immune reaction.[2,55,98] Isolated reports show that AKN can be induced by drugs, such as cyclosporine, diphenylhydantoin, and carbamazepine.[55]

Clinical view

Patients sometimes complain of pruritus and burning sensation.[55] Early lesions are characterized by dome-shaped, fibrotic papules and pustules on the occipital scalp and nape. Secondary infections can result in abscess formation. Hair tufting may also be present.[98,99] In long-term lesions, the papules may coalesce to form hypertrophic scars resembling keloids (**Fig. 16**). Trichoscopy shows broken hairs, tufted hairs,

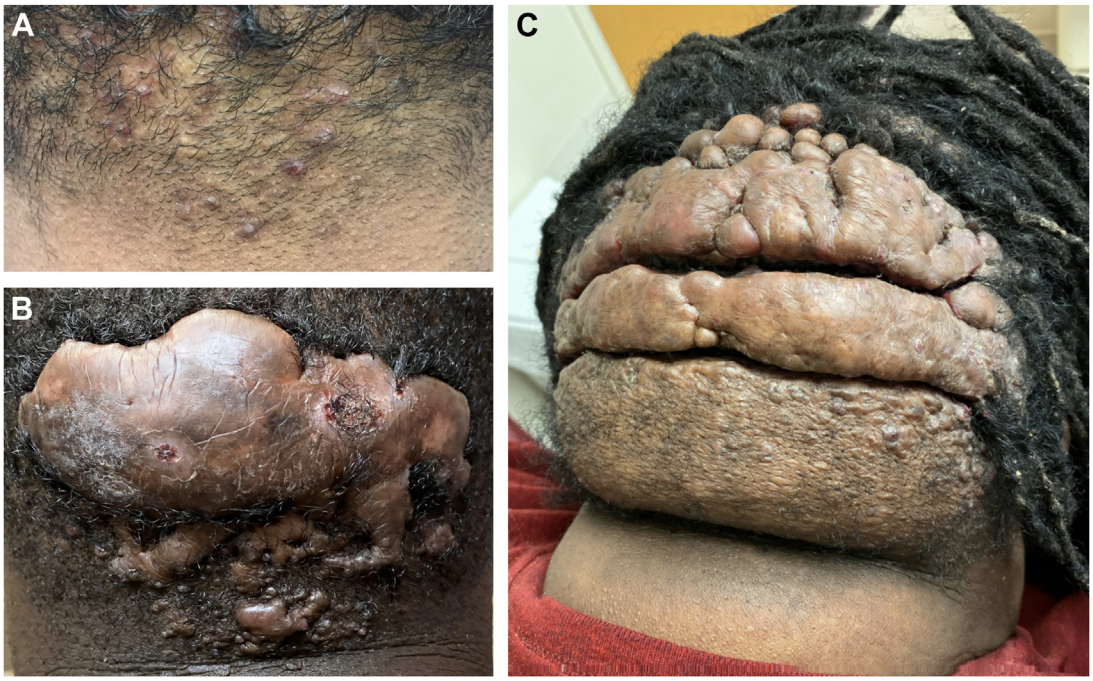

Fig. 16. Clinical spectrum of AKN, starting as fibrotic papules on the occipital scalp (*A*). Papules may coalesce to form hypertrophic scars resembling keloids (*B-C*).

ingrown hairs, and peripilar casts.[8] Late-stage lesions show loss of follicular openings and irregular pinpoint white dots.

Histopathologic findings

Early lesions show a dense infiltrate of neutrophils, lymphocytes, and plasma cells distributed around the isthmus and the lower infundibulum.[100] Complete disappearance of sebaceous glands is associated with inflamed or destroyed follicles. Late lesions show chronic granulomatous inflammation, follicular dropout, fragmented hair shafts, and dense dermal fibrosis without keloidal features.[100,101]

Pitfalls and diagnostic clues

Vertical sections are more appropriate for diagnosis. AKN may be associated with FD, CCCA, or androgenetic alopecia.[98] AKN and FD histologic findings may be identical. Therefore, clinical correlation is advised.[101]

Treatment

AKN is a chronic and recurrent condition; an early treatment may prevent future scarring. Preventive measures include using loose-fitting shirts without occlusive collars and avoiding frequent haircuts with close shaving of the occipital scalp.[2] Early disease is often treated using or combining topical, intralesional corticosteroids, and cryotherapy.[55] When pustules are present, cultures should be obtained to guide the antibiotics regimen. Surgical excision and laser treatment (CO_2, 1064-nm Nd: YAG, 810-nm diode) have been described in refractory cases.[98]

CLINICS CARE POINTS

- African hair shafts have been described as curlier, drier, and more susceptible to chemical and physical damage than other ethnicities.
- Clinical assessment could underestimate erythema in black scalp patients when facing inflammatory disorders.
- The association of two or more hair disorders is common in black patients. Therefore, an excellent clinical-pathological correlation can help us to establish the diagnosis.

DECLARATION OF INTERESTS

J. Larrondo, has no relevant disclosures. Amy J. McMichael, Consulting: Lilly, Janssen, Pfizer, Arcutis, Almirall, Abbvie, Galderma, Bristol Meyers Squibb, Sanofi-Genzyme, UCB, Revian, Johnson & Johnson, L'oreal, and Nutrafol.

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