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Common Hair Disorders

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Table 1.
Scarring Alopecia

Diagnosis	Clinical Features	Histopathological Features	Trichoscopy	Management	Other
CCSA (follicular degeneration syndrome, hot comb alopecia)	Circular area on the vertex of the scalp that is smooth and shiny with notable follicular dropout and a few short hairs remaining in the hair loss area; tenderness, burning, pain, or itching in the affected area; scaling, pustules, and crusting	Histopathology depends on the stage of the lesion; lymphocytic folliculitis, perifollicular granulomatous inflammation with hair shaft foreign body giant cells, and destruction of the follicular units replaced by fibrous scar tissue; premature desquamation of the inner root sheath and eccentric epithelial atrophy	Reduced hair density with hair shaft variability, pinpoint white dots, peripilar white halos, and pigmented asterisklike macules	Topical steroids can be applied daily or 3 times weekly; intralesional corticosteroids usually are injected once monthly or every 2 mo for at least 6 mo; anti-inflammatory agents including tetracycline and minocycline, antimalarials, thalidomide, and cyclosporine; minoxidil foam 5%; advise patients to avoid all hair care practices that involve relaxers, dyes, heat, and traction hairstyles	Most common cause of scarring hair loss in black women; cause is unknown, multifactorial; hair grooming practices have been implicated in the development of CCSA; the relationship between these hair practices and CCSA is unclear, as studies have produced conflicting results
Chronic cutaneous lupus erythematosus	Erythematous macules or papules, may grow in size to become discoid plaques that eventually heal with an atrophic scar and changes in pigment	Hyperkeratosis, dilated keratin-filled follicles, vacuolar degeneration of the basal keratinocytes with epidermal atrophy, dermal mucin, and a lymphoplasmacytic periadnexal and perivascular dermal infiltrate; in long-standing cases, thickened basement membrane and a bandlike scarring of the dermis; DIF can be helpful in diagnosis	Prominent follicular plugging (large yellow dots), white patches, large arborizing vessels	Topical and/or intralesional corticosteroids or calcineurin inhibitors; antimalarial drugs: hydroxychloroquine 200 mg BID	Autoantibody anti-RNP IgG correlated strongly with the activity of the patients; ANAs are often negative in chronic cutaneous lupus erythematosus

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Diagnosis	Clinical Features	Histopathological Features	Trichoscopy	Management	Other
Folliculitis decalvans	Painful perifollicular papules and pustules on vertex and occipital regions of the scalp; pain, itching, or burning sensations in the affected area	Active lesions show a suppurative folliculitis, followed by disruption of the follicular wall and a resulting mixed cell dermal infiltrate; end-stage lesions are characterized by interstitial dermal fibrosis and fibrous tracts in place of hair follicles	Multiple upright hairs can be seen emerging from a single ostium (doll's hair sign) as well as follicular scaling; in scarred areas, there will be pinkish white patches without any follicular markings	Topical antibiotics, including clindamycin and fusidic acid, may be used in conjunction with oral antibiotics; to control symptoms and reduce inflammation, topical and intralesional corticosteroids may be used; for severe and rapidly progressing cases, oral corticosteroids may be considered; oral isotretinoin and dapsone have been shown to be effective	Rare scalp disorder of superficial inflammation that affects both males and females; unknown etiology, however *Staphylococcus aureus* has been cultured from lesions
Frontal fibrosing alopecia	Atrophy of the scalp in frontal and bitemporal hairline; decreased number of hairs in eyebrows; the edge may appear moth-eaten; single "lonely" hairs may persist in the bald areas	Histologic features are identical to lichen planopilaris	Absent follicles, white dots, tubular perifollicular erythema and scale	Dutasteride 0.5 mg/d; calcineurin inhibitors on atrophic scalp and eyebrows; topical corticosteroids	Postmenopausal women

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Diagnosis	Clinical Features	Histopathological Features	Trichoscopy	Management	Other
Lichen planopilaris	White, atrophic, scarring patches of hair loss on the scalp without any hair follicles present; in active lesions the hair follicles surrounding the hair loss region are usually erythematous with macule and scale; positive pull test; Graham-Little-Picardi-Lasseur syndrome variant: triad of scarring scalp alopecia, nonscarring of the axilla and groin, and follicular lichen planus elsewhere on the body	Active lesion consisting of a lichenoid lymphocytic infiltrate affecting the infundibulum of the hair follicle; hypergranulosis of the infundibulum and often changes of lichen planus in adjacent epidermis; if a biopsy is taken at a site of chronic scarring, it will most likely reveal a wedge-shaped infundibular scarring, which can be highlighted with an elastin stain	Identical to frontal fibrosing alopecia and includes the absence of follicular openings, cicatricial white patches, peripilar casts, blue-gray dots, perifollicular erythema and scale	Corticosteroids, both topical and intralesional; hydroxychloroquine 200 mg BID if no response is appreciated after initial treatment, immunomodulating agents such as cyclosporine and mycophenolate mofetil may be considered; third-line therapies with limited evidence: systemic retinoids, griseofulvin, tetracycline, thalidomide, tacrolimus, pioglitazone, and minoxidil	Autoreactive T lymphocytes destroy hair follicle cells, including keratinocytes; appears to be most common in white females; 17%–28% of patients present with other forms of lichen planus, including nail or mucous membrane involvement

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Table 1. (continued)

Diagnosis	Clinical Features	Histopathological Features	Trichoscopy	Management	Other
Traction alopecia	Can occur anywhere tension is applied to the hair; more common in frontal and bitemporal scalp and less common in occipital scalp	Early traction alopecia: trichomalacia (a defect of the hair shaft that is characterized by soft, fragile, swollen hairs; may result from physical injury to the follicles), an increased proportion of the hairs in telogen and catagen phases, a normal number of terminal follicles, pigment casts within follicular canals, histology is indistinguishable from trichotillomania; advanced traction alopecia: the diagnosis can be confirmed if there is miniaturization and follicular dropout but the presence of sebaceous glands	Vellus hairs, mobile hair casts, and white dots	Management includes prevention, treatment of early traction alopecia, and suggestions for advanced traction alopecia; prevention: education should include high-risk hair care practices; early traction alopecia: loosening hairstyles, avoiding heat and other high-risk hair practices, topical or intralesional corticosteroids if inflammation is present, submicrobial doses of oral antibiotics, minoxidil foam 5%; advanced traction alopecia: hair transplants can be considered	Most common in black women; caused by repeated pulling or tension of the hair from hairstyling or hair care; late diagnosis can lead to scarring

Abbreviations: CCSA, central centrifugal scarring alopecia; DIF, direct immunofluorescence; BID, 2 times daily; RNP, ribonucleoprotein; ANA, antinuclear antibody.

Table 2.

Nonscarring Alopecia

Diagnosis	Clinical Features	Histopathological Features	Trichoscopy	Management	Other
Alopecia areata	Discrete patches of hair loss but can present as loss of the entire scalp (alopecia totalis) or loss of all body hair (alopecia universalis); nail findings include geometric pitting (multiple small superficial pits in transverse or longitudinal lines), geometric punctate leukonychia (white spots in a linear pattern), and trachyonychia (sandpaper nails)	Peribulbar and lymphocytic inflammatory infiltrate surrounding anagen follicles, commonly described as a "swarm of bees"; miniaturization and increased catagen/telogen follicles; late lesions show dilated infundibula; identical findings can be seen in syphilitic alopecia (but often shows more plasma cells in infiltrate)	Yellow dots, black dots, broken hairs, tapering hair, exclamation mark hairs, and short vellus hairs	Mainstay of therapy is corticosteroids, but treatment varies according to the patient's age, extent of disease, and location of affected areas; topical steroids are often used as a first-line agent; intralesional corticosteroid injections; oral steroids, JAK inhibitors (ruxolitinib, tofacitinib); methotrexate; minoxidil foam 5%, anthralin; topical immunotherapies (DPCP and squaric acid); if hair regrowth is not sufficient for the patient, camouflage with a wig or hairpiece is an option	Mix of genetic, autoimmune, and environmental factors; scalp is the most commonly affected site representing 90% of cases; genome-wide association studies have pointed to NKG2D ligands in disease pathogenesis
Androgenetic alopecia	Gradual thinning on frontal, vertex, and bitemporal scalp; female presentation with widening of the part line and sparing of the frontal hairline	Miniaturized hair follicles with decreased percentage of anagen hairs; mild superficial follicular inflammatory infiltrate; enlarged sebaceous glands; increased fibrous tract remnants	Hair shaft diameter variation of >20% of hair shafts; pearly white dots; peripilar sign	FDA approved: minoxidil foam 2% or 5%, oral finasteride, and low laser light; non-FDA approved: oral dutasteride and hair restoration surgery; spironolactone 100–200 mg/d in women; hormone replacement therapy	Most common form of human hair loss; affects up to 80% of men and 50% of women; 5-α-reductase, 3-β-hydroxysteroid dehydrogenase, and 17-β-hydroxysteroid dehydrogenase have all been found in increased concentration in the scalp of patients with androgenetic alopecia

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Table 2. (continued)

Diagnosis	Clinical Features	Histopathological Features	Trichoscopy	Management	Other
Telogen effluvium	Diffuse shedding affecting the whole scalp; positive pull test	Increased percentage of hairs in telogen phase (flame thrower–like appearance); usually 5%–15% of follicles are in telogen phase but increases to 25%–30% in this disorder	Empty follicles and decreased hair density	Recovery is spontaneous and takes up to 6 mo after the inciting event; reassurance and counseling; minoxidil foam 5%; spironolactone 100–200 mg/d for chronic forms	Physical or emotional stress causes an increase in the number of hair follicles that are in telogen phase (eg, high fever, surgery, bereavement, exposure to heavy metals, thyroid disorders, anemia, chronic illness, vigorous weight reduction, malignancy, HIV infection, drugs, anorexia nervosa)
Trichotillomania	Patchy bald spots	Can show identical findings as traction alopecia	Broken hair shafts of different lengths with longitudinal fraying, with some broken hairs coiled from high amounts of traction; flame hairs	Both behavioral modification techniques and pharmacotherapy (small studies have shown treatment effects for clomipramine, *N*-acetylcysteine, and olanzapine); a referral to a psychiatrist can be helpful; use of a hair growth window can aid in diagnosis	A form of traumatic alopecia affecting 1%–3% of the population in which the patient pulls out his/her own hair; can involve any part of the body but is most common on the scalp, eyebrows, and eyelashes; an impulse control disorder in which the patient cannot resist the urge to pull the hair, leading to noticeable hair loss; commonly associated with psychiatric comorbidity and functional impairment

Abbreviations: JAK, Janus kinase; DPCP, dyphencyprone; NKG2D, natural killer group 2D; FDA, US Food and Drug Administration; HIV, human immunodeficiency virus.

Practice Questions

1. **A 40-year-old woman presents to the clinic with a burning sensation and tenderness on the scalp. At physical examination you notice erythematous papules and pustules on the vertex scalp. The most likely diagnosis is:**
   a. alopecia areata
   b. CCSA
   c. folliculitis decalvans
   d. lichen planopilaris
   e. traction alopecia

2. **A 60-year-old woman presents with receding hair loss on the frontal and bitemporal scalp. She has noticed hair loss on the eyebrows. She has a history of oral ulcers. On physical examination there is mild erythema and perifollicular scales on the frontal hairline. A hair pull test is positive in this area. The most likely diagnosis is:**
   a. androgenetic alopecia
   b. chronic cutaneous lupus erythematosus
   c. frontal fibrosing alopecia
   d. telogen effluvium
   e. trichotillomania

3. **A 5-year-old girl with a history of seasonal allergies and eczema presents with recurrent patchy hair loss on the scalp of 6 months' duration. Her mother has noticed rapidly progressive hair loss affecting the whole scalp. On trichoscopy, you find yellow dots, broken hairs, and tapering hairs. The most likely diagnosis is:**
   a. alopecia areata
   b. androgenetic alopecia
   c. telogen effluvium
   d. traction alopecia
   e. trichotillomania

4. **A 30-year-old white woman with a history of obsessive-compulsive disorder presents to the clinic with hair loss for the last 3 years. She says she has noticed worsening of the hair loss when she is under stress. She also bites her nails. On physical examination you identify an irregular patch of alopecia with broken hairs on the occipital scalp. The most likely diagnosis is:**
   a. alopecia areata
   b. androgenetic alopecia
   c. lichen planopilaris
   d. traction alopecia
   e. trichotillomania

5. **A 45-year-old black woman who has a family history of hair loss in her mother presents with tenderness and burning sensation on the vertex scalp. She reports the hair loss was worse after she got a hair relaxer 6 months prior. She uses braids on her scalp and she has not had a relaxer since then. The most likely diagnosis is:**
   a. CCSA
   b. chronic cutaneous lupus erythematosus
   c. folliculitis decalvans
   d. lichen planopilaris
   e. trichotillomania

Fact sheets and practice questions will be posted monthly.