Alopecia

Alopecia classified into:

- ***Non-cicatricial Alopecia*** **:** No clinical sign of tissue inflammation, scarring, or atrophy of skin .
- ***Cicatricial alopecia*** **:** Evidence of tissue destruction such as inflammation, atrophy, and scarring is apparent.

Classification of acquired alopecia

Can occur **globally** or **focally** : (Causes of hair loss)

Diffuse (global) hair loss

Non-scarring

1- **Failure of follicle production**
2- **Hair shaft abnormality**
3- **Abnormality of cycling (shedding)**
- Telogen effluvium
- Anagen effluvium
- Loose anagen syndrome
- Alopecia areata

Focal (patchy, localized) hair loss

Non-scarring

3- Production decline :
- Triangular alopecia
- Pattern hair loss (androgenetic alopecia)

4- Hair breakage :
- Trichotillomania
- Traction alopecia
- Infection (tinea capitis)
- Primary or acquired hair shaft abnormality

5- Unruly hair.

6- Abnormality of cycling :
- Alopecia areata
- Secondary syphilis (alopecia areolaris) ("moth-eaten" appearance in beard or scalp).

Scarring (cicatricial) alopecia

1- Lymphocytic :
- Chronical cutaneous (discoid) lupus erythematosus
- Lichen planopilaris (LPP)
- Classic pseudopelade of Brocq
- Central centrifugal cicatricial alopecia
- Alopecia mucinosa
- Kertosis folliclaris spinulosa decalvans

2- Neutrophilic :
- Folliculitis decalvans
- Dissecting folliculitis (cellulitis)

3- Mixed :
- Folliculitis keloidalis
- Folliculitis necrotica
- Erosive pustular dermatosis

4- Non-specific :
- Trauma.
- Chemical or physical burn.
- Surgical wound
- Neoplasm (Basal cell carcinoma ,Squamous cell carcinoma)

Scarring (cicatricial) alopecia

- Primary cicatricial alopecia (PCA) results from damage or destruction of the hair follicles stem cells by:
  - Inflammatory (usually noninfectious) processes
  - Infection: e.g., "kerion" tinea capitis, necrotizing herpes zoster
  - Other pathologic processes: surgical scar, primary or metastatic neoplasm.
- Manifestations: Effacement of follicular orifices in a patchy or focal distribution, usually in scalp or beard.
- The end result is effacement of follicular orifices & replacement of the follicular structure by fibrous tissue.
- Scarring is irreversible. Therapies are ineffective.

TELOGEN EFFLUVIUM

- Telogen effluvium (TE) is the transient increased shedding of normal club (telogen) hairs from resting scalp follicles.
- Secondary to accelerated shift of anagen (growth phase) into catagen and telogen (resting phase)
- Results in increased daily hair loss and, if severe, diffuse thinning of scalp hair.

Etiology:

A reaction pattern to a variety of physical or mental stressors :

- **Endocrine :**
  1- Hypo- or hyperthyroidism ;
  2- postpartum ;
  3- discontinuation or changing type of estrogen containing drugs
- **Nutritional:**
  1- Deficiency: biotin, zinc, iron, essential fatty acid
  2- Rapid weight loss,
  3- caloric or protein deprivation,
  4- excessive vitamin A ingestion
- **Physical stress :**
  1- Febrile illnesses,
  2- catabolic illnesses (e.g., malignancy, chronic infection),
  3- major surgery,
  4- major trauma,
  5- acute or chronic psychological stress
- **Psychological stress :**
  1- Anxiety,
  2- depression,
  3- bipolar disorder
- **Intoxication :** Thallium, mercury, arsenic
- **Drugs :**
  1- Antimitotic agents (dose dependent): cancer chemotherapy, benzimidazoles.
  2- Antihypertensives: captopril
  3- Anticoagulants
  4- CNS drugs: lithium, valproic acid
  5- Cholesterol-lowering drugs
  6- Colchicine
  7- Cytostatic drugs
  8- Interferon
  9- Penicillamine
  10- Retinoids: vitamin A excess, retinoids (isotretinoin, acitretin, indinavir)
  11- Selective serotonin reuptake inhibitors
  12- Inflammatory scalp disease: Seborrheic dermatitis, erythroderma
- **Idiopathic :**
  No obvious cause is apparent in a significant number of cases.

ANAGEN EFFLUVIUM

- **Etiology:** See below
- **Onset** is usually rapid & extensive.
- **Pathogenesis:** Occurs after any insult to hair follicle that impairs its mitotic/metabolic activity. Rapid growth arrest or damage to anagen hairs that skip catagen and telogen phases and are shed.
- More common and severe with combination chemotherapy than with the use of a single drug. Severity is generally dose dependent.
- **Manifestations:**
  Scalp hair : loss is diffuse, extensive; also: eyebrows/lashes, beard, etc.
  Nails: show transverse banding or ridging.
- Regrowth is usually rapid after discontinuation of Chemotherapy

Etiology:

Anagen cycle disrupted causing varying degrees of hair follicle dystrophy :

- **Radiation therapy to head .**
- **Alkylating agents:**
  busulfan, carboplatin, carmustine, BCNU, chlorambucil, cisplatin, dacarbazine, estramustine, fotemustine, ifosamide, lomustine, mechlorethamine, nitrogen mustard, melphalan, oxaliplatin, procarbazine, streptozocin, temozolomide, thiotepa.
- **Intoxications:**
  mercury, boric acid, thallium, colchicine.
- **Severe protein malnutrition.**

Alopecia areata

- A localized loss of hair in round or oval areas with no apparent inflammation of the skin. Most common on scalp.
- Nonscarring; hair follicle intact; hair can regrow.
- **Clinical findings:** Hair loss ranging from solitary patch to complete loss of all terminal hair.
- **Prognosis:** good for limited involvement. Poor for extensive hair loss.
- **Management:** intralesional triamcinolone effective for limited number of lesions.

PATHOGENESIS

- **Chronic organ-specific autoimmune disease,** mediated by autoreactive T cells affecting hair follicles and nails.
- **Associated autoimmune disorders:** Autoimmune thyroid disease in adults.
- Follicular damage occurs in **anagen** followed by → rapid transformation to **catagen** and to → **telogen**; then to → **dystrophic anagen status**.
  While the disease is active, follicles unable to progress beyond early anagen and do not develop normal hair.

CLINICAL MANIFESTATIONS

Age: Any age , but peak between 20 – 50 years

Sex : affect both sexes.

Duration of Hair Loss
Gradual over weeks to months.

Skin Symptoms
Disfiguring bald patch.

Associated Findings
- Autoimmune thyroiditis.
- Down syndrome.
- Autoimmune polyendocrinopathy-candidiasis–ectodermal dysplasia syndrome.

Skin Findings
- Usually none.
- Possibly minimal erythema in area of hair loss.

Hair

Round patches of hair loss.
- Single or multiple. May coalesce.
- It's often **sharply defined.**
- **Normal-appearing skin** with follicular openings present .
- **"Exclamation mark" hairs** (Fig. 32-8) : Diagnostic broken- off stubby hairs (distal ends are broader than proximal ends) seen at margins of hair loss areas.
- **Scattered, discrete areas of alopecia** (Fig. 32-9) or **Confluent with total loss of scalp hair** (Fig. 32-10), or **generalized loss of body hair (including vellus hair).**
  Diffuse AA of scalp (noncircumscribed) gives the appearance of thinned hair; can be difficult to differentiate from pattern hair loss of telogen effluvium, hair loss with thyroid disease.
- With regrowth of hair, **new hairs** are fine, often white or gray.

Sites of Predilection
- Scalp (most commonly)
- Any hair-bearing area : Beard, eyebrows, eyelashes, pubic hair.
  - **Alopecia areata (AA):** Solitary or multiple areas of hair loss.
  - **AA totalis (AAT):** Total loss of terminal scalp hair.
  - **AA universalis (AAU):** Total loss of all terminal body & scalp hair.
  - **Ophiasis:** Bandlike pattern of hair loss over periphery of scalp .

Nails
- Fine pitting ("hammered brass") of dorsal nail plate.
- mottled lunula,
- trachyonychia (rough nails),
- onychomadesis (separation of nail from matrix).

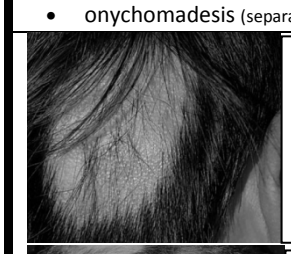

FIGURE 32-8
Solitary lesion
The short, broken-off hair shafts (so-called exclamation point hair) appear as very short stubs emerging from the

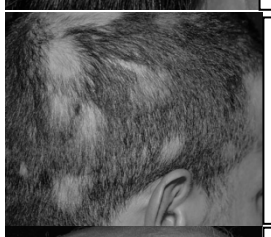

FIGURE 32-9
multiple, extensive lesions
Multiple, confluent, involved sites on the scalp with "exclamation point hairs."

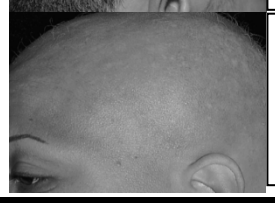

FIGURE 32-10
AA totalis

DIFFERENTIAL DIAGNOSIS

Nonscarring Alopecia :
- White-patch tinea capitis,
- Trichotillomania,
- Early scarring alopecia,
- Pattern hair loss,
- Secondary syphilis (alopecia areolaris) ("moth-eaten" appearance)

LABORATORY EXAMINATIONS

- **Serology** :
  1- ANA (to rule out SLE);
  2- rapid plasma reagin (RPR) test (to rule out secondary syphilis).
- **KOH Preparation:** Rule out tinea capitis.
- **Dermatopathology :**
  Acute lesions show :
  - peribulbar, perivascular, and outer root sheath mononuclear cell infiltrate of T cells and macrophages;
  - follicular dystrophy with abnormal pigmentation and matrix degeneration.

MANAGEMENT

No curative treatment is currently available. Treatment for AA is unsatisfactory.

Treatment directed at inflammatory infiltrate and growth inhibitor factors produced by inflammation.

1- General measures :
- **Psychological support (very important).**
- Persons with extensive scalp involvement such as AAT may prefer to wear a wig or hairpiece.
- Makeup applied to eyebrows is helpful. Eyebrows can be tattooed.

2- Glucocorticoids
- **Glucocorticoids *Topical*** :
  Superpotent agents not usually effective.
- ***Intralesional Injection*** :
  Few and small lesions of AA can be treated with intralesional triamcinolone acetonide, 3–7 mg/mL, which can be very effective temporarily.
- ***Systemic Glucocorticoids :***
  May induce regrowth, but AA recurs on discontinuation; risks of long-term therapy therefore preclude their use.

3- Systemic Cyclosporine
Induces regrowth, but AA recurs when drug is discontinued.

4- Induction of Allergic Contact Dermatitis -
By Dinitrochlorobenzene (DNCP) , but local discomfort due to allergic contact dermatitis and swelling of regional lymph nodes poses a problem.

5- Oral PUVA (Photochemotherapy) :
Entire body must be exposed,
in that the therapy is believed to be a form of systemic immune suppression.

- **Patchy alopecia :**
  - Intralesional corticosteroids : Up to 2 mL injected/session and repeated at intervals.
  - Potent topical steroid (1-2×/day).
  - Topical anthralin (0.1%-2.0% once daily) : Wash off after 10-20 min, steadily increase contact duration, switch to higher dose if no significant irritation.
  - Minoxidil lotion (5%) twice daily.
- **Extensive or rapidly progressive alopecia.**
  - Contact immunotherapy.
  - Systemic corticosteroids. Benefits are uncertain and must be weighed against risk of systemic corticosteroid therapy.
  - Wig or hairpiece.
- **Alopecia totalis/universalis**
  - Contact immunotherapy.
  - Topical/systemic steroids.
  - Wig or hairpiece.

COURSE

- Spontaneous remission is common in patchy AA but is less so with AAT or AAU.
- Poor prognosis associated with onset in childhood, loss of body hair, nail involvement, atopy, family history of AA.
- If occurring after puberty, 80% regrow hair. With extensive AA, AAT, AAU, <10% recover spontaneously.
- Recurrences of AA, however, are frequent.
- Systemic glucocorticoids or cyclosporine can induce remission of AA but do not alter the course.