**Classification of acquired alopecia**

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

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

### Alopecia

**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

**Hair breakage**

- Trichotillomania
- Traction alopecia
- Infection (tinea capitis)
- Primary or acquired hair shaft abnormality

**Unruly hair**

**Anomalies of cycling**

- Alopecia areata
- Secondary syphilis (alopecia areolaris)
- “woolly-aten” appearance in beard or scalp

**Telegen 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 until normal rhythm is re-established.

**Etiology**

- **Endocrine**:
  - Hypo- or hyperthyroidism
  - Postpartum
  - Discontinuation or changing type of estrogen containing drugs

- **Nutritional**:
  - Deficiency: Biotin, zinc, iron, essential fatty acid
  - Rapid weight loss
  - Caloric or protein deprivation
  - Excessive vitamin A ingestion

- **Physical stress**:
  - Febrile illness
  - Catabolic states (e.g., malignancy, chronic infection)
  - Major surgery
  - Major trauma
  - Acute or chronic psychological stress

- **Psychological stress**:
  - Anxiety
  - Depression
  - Bipolar disorder

- **Intoxication**:
  - Thallium, mercury, arsenic

- **Drugs**:
  - Antibiotic agents (dose dependent): cancer chemotherapy, benzimidazoles.
  - Antihypertensives: captopril
  - Anticatabolites
  - CNS drugs: lithium, valproic acid
  - Cholesterol-lowering drugs
  - Colchicine
  - Corticosteroids
  - Interferon
  - Pancrelinase
  - Retinoids: vitamin A excess, retinoids (sotretinoin, acitretin, isotretinoin)
  - Selective serotonin reuptake inhibitors
  - Inflammatory scalp disease: Seborrheic dermatitis, erythrodema
  - Lipidopathy

**Alopecia areata**

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

**Focal (patchy, localized) hair loss**

**Non-scarring**

1. **Production decline**:
   - Triangular alopecia
   - Pattern hair loss (androgenic alopecia)

2. **Hair breakage**:
   - Trichotillomania
   - Traction alopecia
   - Infection (tinea capitis)

3. **Unruly hair**

4. **Abnormality of cycling**: Alopecia areata

**Cicatricial (cicatrical) 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 focal or paticular 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.

**Scarring (cicatrical) alopecia**

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**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 follicles.

**Clinical manifestations**

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- Erosive pustular dermatosis
- Chemical or physical burn
- Tinea capitis
- Hair shaft abnormality (so-called "waxy-aten" appearance)

**Differential diagnosis**

- Non-scarring Alopecia
  - White-pocket tinea capitis
  - Trichotillomania
  - Early scarring alopecia
  - Pattern hair loss
  - Secondary syphilis (alopecia areolaris)

- Laboratory examination

**Course**

- Spontaneous remission is common in patchy AA but not so with AAT or AAU.
- Better 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.