

Hair Conditions: Alopecia, Dandruff, and Seborrheic Dermatitis

This presentation explores three common hair and scalp conditions that affect millions worldwide: alopecia (hair loss), dandruff, and seborrheic dermatitis. We'll examine their causes, symptoms, diagnosis methods, and treatment options, along with their psychological and social impacts.



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Alopecia Areata

Epidemiology & Pathophysiology

Affects 2% of the global population. Onset peaks in teens/young adults; no gender predilection. Associated with autoimmune diseases (e.g., thyroiditis, vitiligo).

Clinical exam: Exclamation mark hairs (tapered, broken hairs at margins). Dermoscopy: Yellow dots (follicular openings with keratin), black dots (destroyed hairs). Biopsy: Peribulbar lymphocytic infiltrate ("swarm of bees").

Clinical Variants & Diagnosis

Alopecia totalis: Total scalp hair loss.

Alopecia universalis: Loss of all body hair.

Ophiasis: Band-like hair loss along the occipital scalp

(poor prognostic sign).









alopecia totalis

alopecia universalis

Ophiasis



Treatment & Prognosis

First-line: Intralesional corticosteroids (triamcinolone 2.5–5 mg/mL). Topical: High-potency steroids (clobetasol), minoxidil 5%,. Systemic: JAK inhibitors (baricitinib, approved by FDA in 2022), oral steroids (short-term)., PRP (platelet-rich plasma).

Spontaneous regrowth in 50% within 1 year; poorer prognosis with ophiasis, nail changes, or childhood onset.

Androgenetic Alopecia (AGA)

Epidemiology

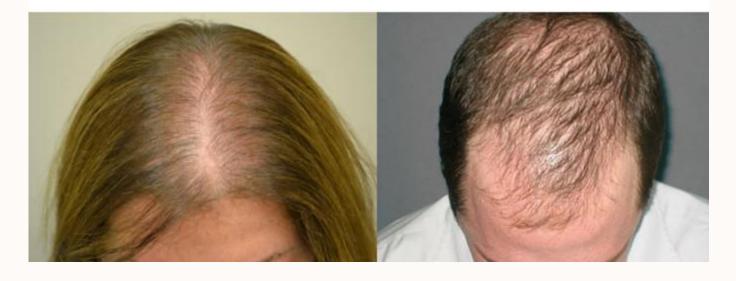
Affects 50% of men by age 50 (Norwood-Hamilton scale) and 40% of women (Ludwig scale).

Diagnosis

Male pattern: Receding temples, vertex thinning.

Female pattern: Diffuse thinning with preserved frontal hairline. Dermoscopy: Hair diameter variability (>20% difference), peripilar signs (brown halos).

Androgenetic Alopecia-Male and Female Pattern





Androgenetic Alopecia Treatment Options

Topical Treatments

Minoxidil 5% (stimulates anagen phase):
Applied twice daily to dry scalp. Mechanism:
Potassium channel opener that increases
blood flow and prolongs the growth phase.
Results typically seen after 4-6 months of
consistent use. Women may use 2% or 5%
formulations. Side effects include initial
shedding, scalp irritation, and unwanted hair
growth in adjacent areas.

Procedural Interventions

Low-level laser therapy (LLLT):
Photobiomodulation increases ATP
production and stimulates stem cells in hair
follicles. Available as in-office treatments or
home devices (combs, helmets). Treatment
requires 2-3 sessions weekly for at least 6
months.

Hair transplantation: Follicular Unit Extraction (FUE) involves harvesting individual follicular units, while Follicular Unit Transplantation (FUT) removes a strip of tissue. Modern techniques achieve natural-looking results with 90-95% graft survival rates. Candidacy depends on donor hair availability and quality.

Oral Medications

Finasteride (1 mg/day): Inhibits 5-alpha reductase, reducing DHT production by up to 70%. Primarily for men; contraindicated in women of childbearing potential due to teratogenicity. Efficacy: Stops progression in 90% and stimulates regrowth in 65% of men. Side effects may include sexual dysfunction (2-4% of patients).

Spironolactone (for women): Anti-androgen that blocks androgen receptors. Dosage: 50-200 mg daily. Requires monitoring of electrolytes and blood pressure. Contraindicated during pregnancy due to potential feminization of male fetuses.

Emerging Therapies

Platelet-rich plasma (PRP) injections, microneedling, and topical prostaglandin analogs show promising results in preliminary studies. Combination therapy approaches often yield superior outcomes compared to monotherapy.

Traction Alopecia



Epidemiology



Common in individuals with tight hairstyles (e.g., braids, weaves, ponytails). Early stages reversible; chronic traction leads to permanent fibrosis.



Pathology

Mechanical stress \rightarrow perifollicular hemorrhage \rightarrow inflammation \rightarrow follicular drop-out.



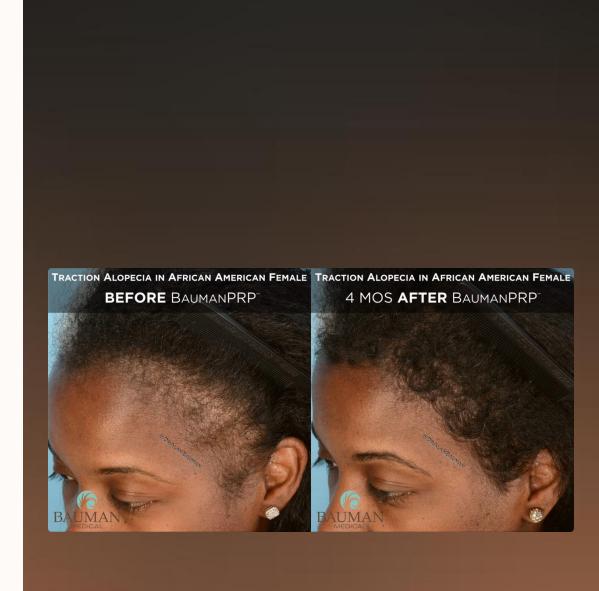
Prevention

Avoid tight hairstyles; use satin/silk hair coverings. Educate on early signs (folliculitis, hair breakage).



Treatment

Early stage: Topical steroids, minoxidil. Late stage: Hair transplant; camouflage (wigs, fibers).



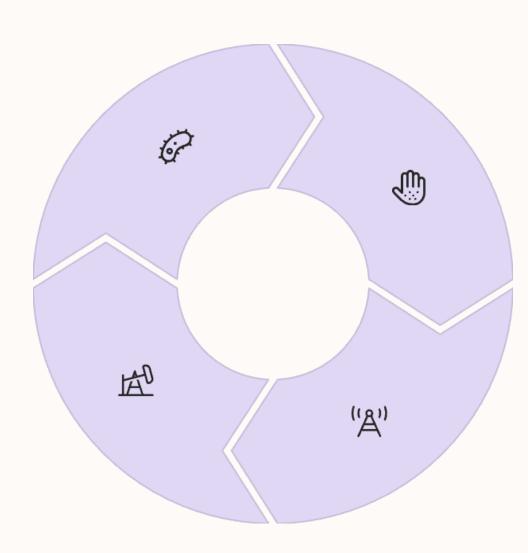
Dandruff (Pityrias is Capitis): Pathophysiology

Malassezia Yeast Proliferation

Lipophilic yeast (e.g., M. globosa, M. restricta) metabolize sebum triglycerides → oleic acid

Contributing Factors

Seborrhea (oily scalp), infrequent shampooing, cold/dry climates

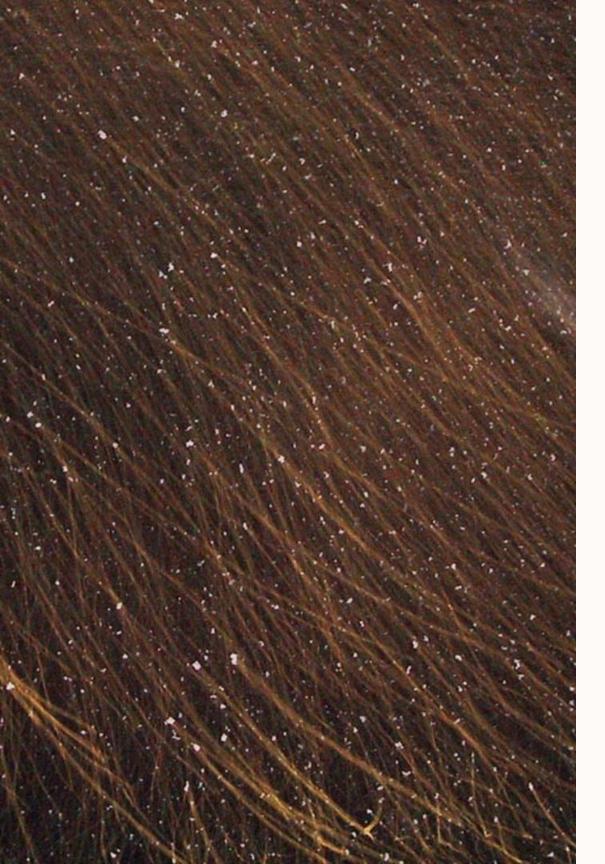


Skin Barrier Disruption

Oleic acid penetrates stratum corneum → inflammation

Hyperproliferation

Accelerated cell turnover leads to visible flaking



Dandruff: Clinical Features and Management

Clinical Features

- Flakes: White or gray, loosely adherent
- Symptoms: Mild pruritus; no significant erythema

Differential Diagnosis

- Dry scalp: Smaller flakes, non-greasy, worsens in winter
- Psoriasis: Thick silvery scales, Auspitz sign (bleeding when scraped)

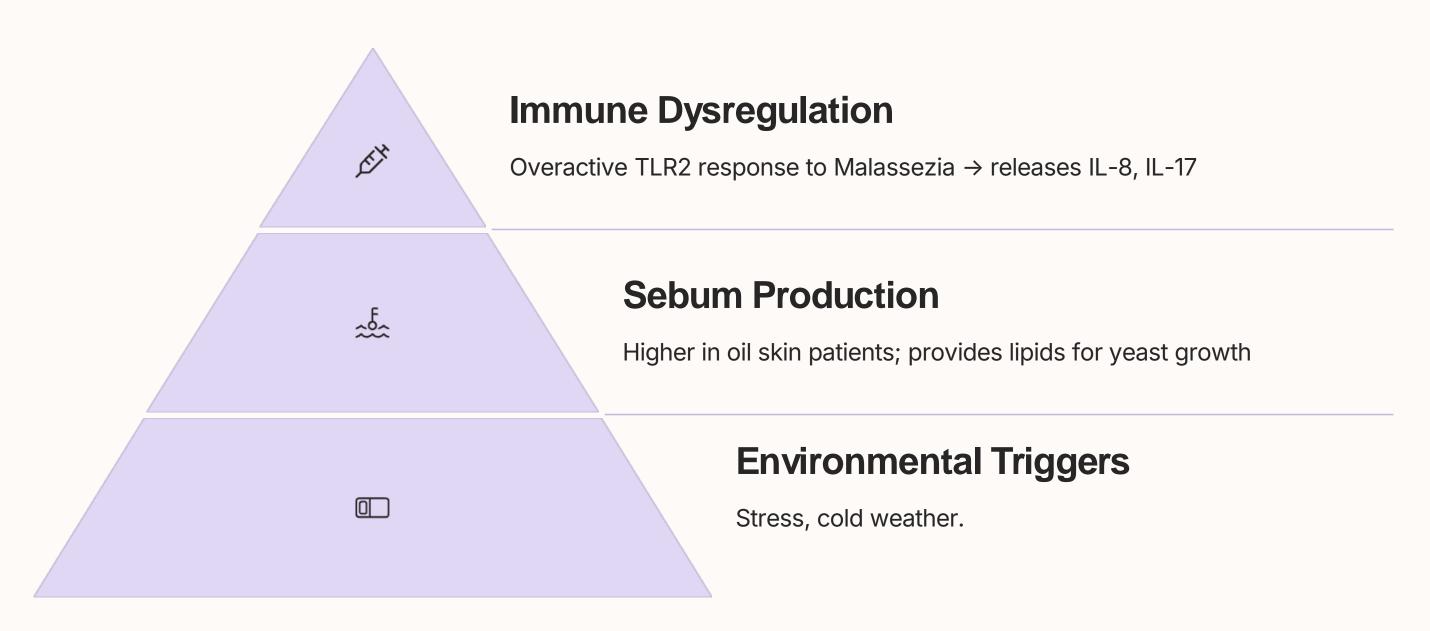
First-line Management

- Zinc pyrithione: Antifungal + anti-inflammatory
- Ketoconazole 2%: Targets Malassezia
- Salicylic acid: Exfoliates scales

Adjunctive Treatments

- Tea tree oil (5% shampoo), coconut oil (moisturizes)
- Frequency: Use medicated shampoo 2–3× weekly; rotate agents to prevent resistan

Seborrheic Dermatitis: Pathogenesis



Seborrheic Dermatitis: Clinical Presentation and Treatment



Scalp Presentation 🜈

Erythematous patches with yellow, greasy scales.



Extracranial Sites

Nasolabial folds, eyebrows, chest.



Infants

"Cradle cap" (resolves by 12 months; rarely itchy).



Treatment Options

- Shampoos:
 Ketoconazole
 2%, ciclopirox
 1%, selenium
 sulfide 2.5%
- Topical steroids: Clobetasol solution (shortterm to avoid atrophy)
- Calcineurin

 inhibitors:
 Tacrolimus 0.1%
 (for face/eyelids)



Severe Cases

Oral antifungals (itraconazole), phototherapy (UVB).



Maintenance

Lifelong management; flareups linked to stress/seasonal changes.

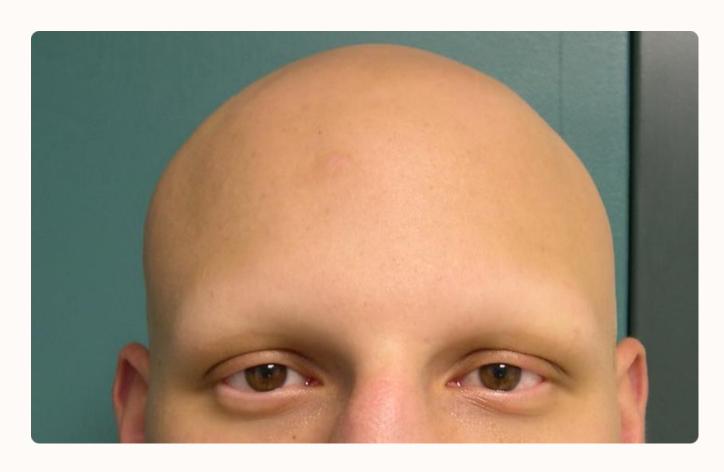




Comparative Analysis of Scalp Conditions

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Psychological and Social Impact



Alopecia Impact

40% of patients report anxiety/depression; support groups crucial.



Dandruff/SD Impact

Social stigma; "snowy shoulders" affect self-confidence.

Folliculitis: Pathophysiology & Subtypes

Infectious Folliculitis

Bacterial:

- Staphylococcus aureus: Most common; methicillin-resistant strains (MRSA) require culture-guided therapy
- Pseudomonas aeruginosa: "Hot tub folliculitis" due to biofilm in poorly chlorinated water.

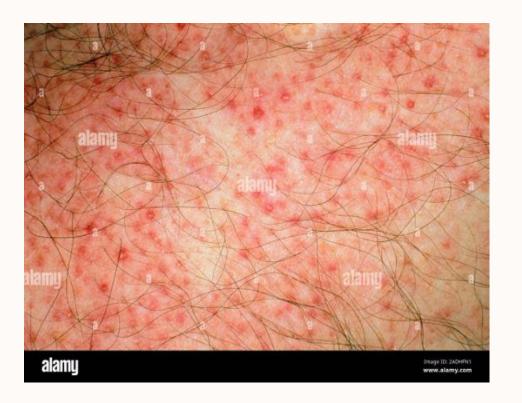
Fungal:

- Malassezia furfur: Causes pityrosporum folliculitis (pruritic papules on chest/back); diagnosed via KOH prep showing hyphae and spores
- Dermatophytes: Tinea barbae in beard area; requires oral antifungals (terbinafine)

Viral: Herpes simplex virus (HSV) folliculitis in immunocompromised patients

Non-Infectious Folliculitis

- Mechanical/Irritant: Pseudofolliculitis barbae (curly hair regrowth causing ingrown hairs); common in Black individuals
- Eosinophilic Folliculitis: HIV-associated; eosinophil-rich infiltrate on biopsy



Folliculitis: Clinical Presentation & Management

Key Clinical Features

• Papules/pustules with erythematous base; may coalesce into furuncles or carbuncles

Mimics: Acne vulgaris, rosacea, HSV infection, scabies

Topical Therapy

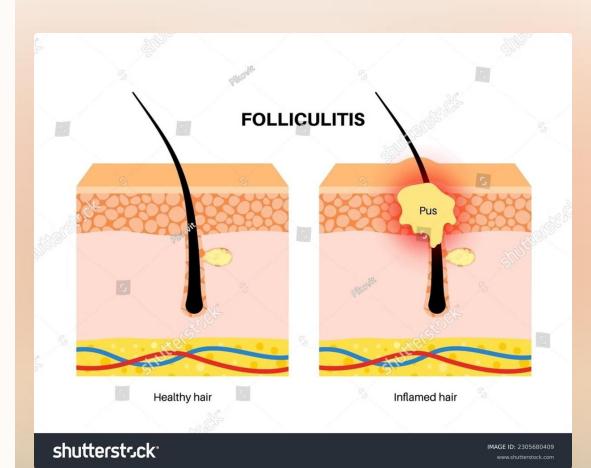
- Bacterial: Mupirocin 2% BID × 10 days; clindamycin 1% for acne-like presentations
- Fungal: Ketoconazole 2% shampoo daily; econazole cream

Oral Therapy

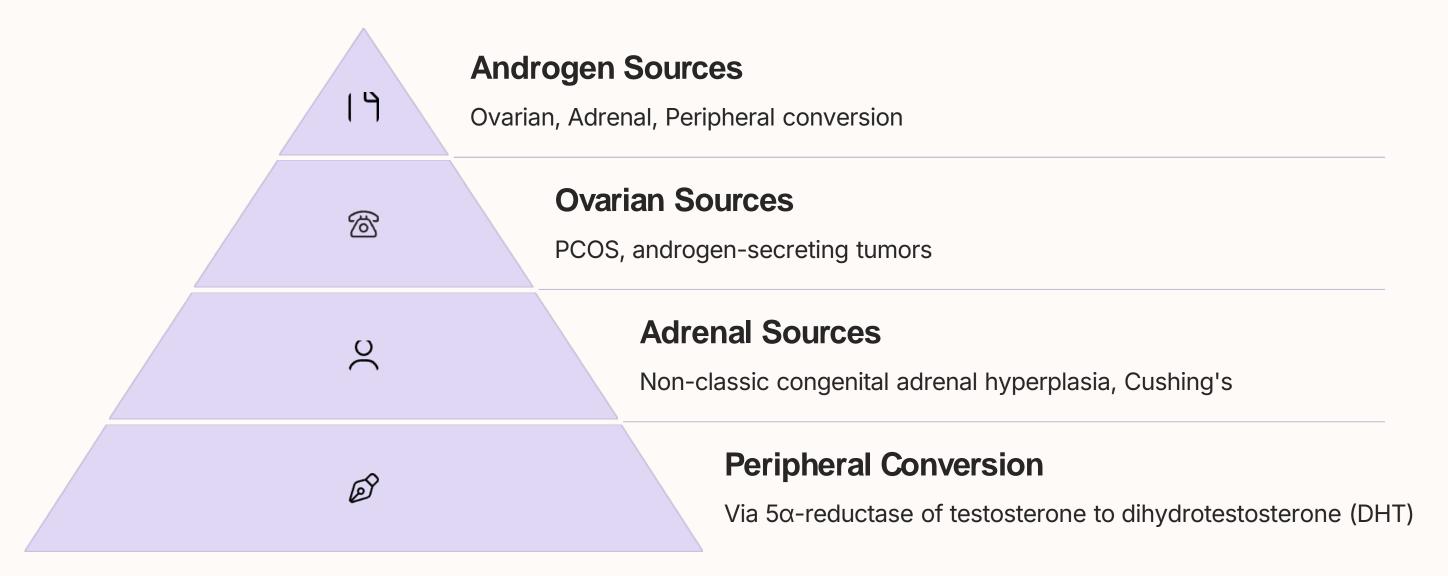
- Severe bacterial: Cephalexin 500 mg QID × 7 days; doxycycline 100 mg BID for MRSA
- Fungal: Itraconazole 200 mg daily × 2 weeks

Prevention Strategies

- Shave with single-blade razors, avoid tight clothing, disinfect hot tubs
- Patient Education: "Shave with the grain," use benzoyl peroxide wash post-shaving



Hirsutism: Androgen Physiology & Etiology



PCOS Diagnosis (Rotterdam Criteria) requires 2/3: Oligo/anovulation, clinical/biochemical hyperandrogenism, polycystic ovaries on ultrasound.

Hirsutism: Diagnostic Workup



Initial Lab Tests

Total testosterone (>200 ng/dL suggests tumor),, LH:FSH ratio (>2 in PCOS)



Secondary Lab Tests

17-OH progesterone, prolactin



Imaging

Pelvic ultrasound (ovarian stroma pearls), adrenal CT/MRI.



Differential Diagnosis

PCOS, Congenital adrenal hyperplasia, adrenal/ovarian tumors, idiopathic hirsutism, medications



Hirsutism: Treatment Algorithms

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First-Line Therapy

Combined Oral Contraceptives (COCs): Suppress ovarian androgens; improve menstrual regularity



Topical Eflornithine: Inhibits hair follicle ornithine decarboxylase; apply BID

Cosmetic: Laser (diode, alexandrite, ND-YAG)

Psychosocial Considerations: Screen for depression/anxiety; cultural sensitivity (e.g., hijab-wearing patients may prioritize facial hair).



Spironolactone 50–200 mg/day (monitor potassium; avoid in pregnancy)

Finasteride (5α-reductase inhibitor; off-label use)



Metformin for insulin resistance Weight loss (5–10% reduces androgens)





Trichotillomania: DSM-5 Diagnostic Criteria

- 1 Recurrent Hair Pulling

 Despite attempts to stop
- Tension and Relief Cycle
 Tension before pulling,

pleasure/relief after

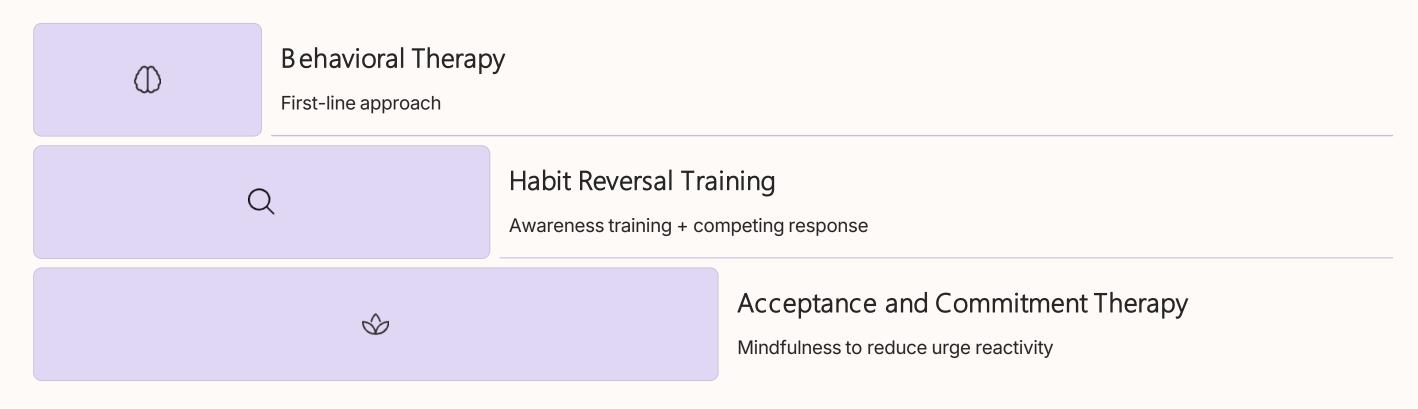
3 Significant Impact
Clinically significant distress or impairment

Trichotillomania is a mental health disorder characterized by the recurrent, compulsive urge to pull out one's hair, leading to noticeable hair loss. It is classified as an obsessive-compulsive and related disorder



Am I a Victim of Trichotillomania?!

Trichotillomania: Evidence-Based Management



Pharmacologic Options:

SSRIs

Fluoxetine 20–80 mg/day for comorbid anxiety/depression

Pediatric Focus: Parental reinforcement + "BFRB Kids" support groups

N-acetylcysteine

1200–2400 mg/day modulates glutamate; emerging evidence

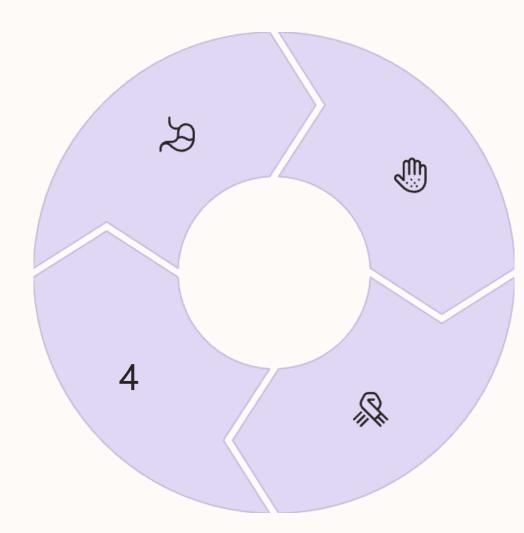
Trichotillomania: Complications

Trichobezoar

Surgical emergency if causing bowel obstruction

Psychological Impact

Social isolation, low self-esteem



Skin Damage

Folliculitis from chronic picking

Scarring Alopecia

Permanent hair loss from repeated trauma

Trichobezoars (hairballs) can form when patients with trichotillomania ingest the hair they pull. These can accumulate in the gastrointestinal tract, potentially leading to serious complications including intestinal obstruction, perforation, and peritonitis. Regular monitoring and patient education about this risk is essential, especially in pediatric populations.

