HOW TO BEST MANAGE ALOPECIA IN 2018

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MOST COMMON TYPES OF ALOPECIA

Non scarring
- Androgenetic alopecia
- Alopecia areata
- Telogen effluvium
- Lupus
- Syphilis
- Trichotillomania

Scarring
- Lichen planopilaris (LPP)
- Frontal fibrosing alopecia (FFA)
- Central centrifugual cicatricial alopecia (CCCA)
- Traction alopecia
- Dissecting cellulitis
- Acne keloidalis nuchae
INITIAL EVALUATION HELPS SIGNIFICANTLY NARROW THIS WIDE DDX
HISTORY

- History of hair loss in the past
- New or changing medications
- Family history of hair loss
- Ethnic styling practices
  - Tight braids or weaves or long term tight ponytails
- Pace of hair loss – gradual or rapid
- Recent stressor, illness or hospitalization in the last 6 months
- Quantity of hair loss
- Pre or post menopausal
- Type of diet
COEXISTING DISEASE

- Thyroid
- Vitiligo
- PCOS
- Lupus (discoid or systemic)
PHYSICAL

- Distribution
  - Frontal, vertex, isolated patches, diffuse, or irregular
- Hair pull test
  - trichogram
- Preserved follicular ostia
- Broken hairs (exclamation point hairs)
- White hairs
- Body hair
  - Eyebrows, eyelashes, beard
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SCARRING (CICATRICIAL) VS. NONSCARRING

- Look for preserved follicular ostia
- Regrowing hairs

*Figure 9: Dermoscopy of the scalp. A) FPHL. There is great variability in the thickness of the hair shaft, hairs emerging individually from*
SCARRING (CICATRICIAL) VS. NONSCARRING

- Look for preserved follicular ostia
- Regrowing hairs
LABORATORY EVALUATION

• For diffuse, non-scarring alopecia:
  • Hgb/Hct
  • Ferritin – must be > 40ng/dl for hair regrowth
  • TSH
LABORATORY EVALUATION

- Biopsy – *mostly reserved for scarring alopecias*
  - Two, 4 or 5 mm punch biopsy samples preferred
  - one for vertical sectioning and one for horizontal (can be put in same bottle)
NON SCARRING ALOPECIAS

- Primary
  - Androgenetic alopecia
  - Alopecia areata
  - Telogen effluvium
- Or associated with inflammatory skin disease
  - Seborrhea
  - Psoriasis
  - Lupus
- Infections
  - Tinea capitis
  - Syphilis
- Exogenous
  - Trichotillomania
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ANDROGENETIC ALOPECIA (AGA)

- Genetically determined sensitivity of scalp hair follicles to adult levels of androgens
  - Strong genetic predisposition, but its complicated!
- Miniaturization of hairs in a symmetric “pattern” on the crown, vertex, and frontal regions
  - Conversion of terminal hairs into vellus hairs
  - Affected follicles are more sensitive to dihydrotestosterone (DHT)
- Frequency and severity increase with age
  - 80% of men and 50% of women have AGA by age 80
ANDROGENETIC ALOPECIA

Male Pattern Hair Loss
- Symmetric and progressive
- Some pattern variation
  - Frontotemporal and vertex areas affected to differing degrees

Female Pattern Hair Loss
- Diffuse central thinning of the crown with preservation of the frontal hairline
- Initially might coincide with superimposed telogen effluvium
- Early-onset or severe disease should prompt workup for pathologic hyperandrogenism
ANDROGENETIC ALOPECIA

Male Pattern Hair Loss
- Norwood Classification

Female Pattern Hair Loss
- Sinclair Scale
ANDROGENETIC ALOPECIA TREATMENT

- Male Pattern Hair Loss
  - Topical minoxidil and oral finasteride (Propecia) are FDA approved
TOPICAL MINOXIDIL

- 2% solution, 5% solution, 5% foam

- 1mL applied to scalp/affected areas BID

- Adverse reactions:
  - Mild scalp dryness and irritation → common
  - Allergic contact dermatitis → rare

- Minoxidil-induced hair loss is often associated with shedding of telogen hairs and a paradoxical worsening of hair loss at 4-6 weeks
  - Resolves with continued therapy

- Continued therapy is necessary to maintain response
FINASTERIDE

- Type II 5α-reductase inhibitor
  - Blocks conversion of circulating testosterone to DHT

- 1mg po daily (Propecia)

- Halts hair loss in 90%
- Partial hair regrowth in 65%

- Allow 6 months to see response, max response seen at 1-2 years

- Continued therapy is necessary to maintain response
FINASTERIDE

- Side effects:
  - Reversible loss of libido, reduced volume of ejaculate fluid, and erectile dysfunction
    - 3.4 – 15.8% of men\(^1\)
  - Post-finasteride syndrome
  - Falsely decreased serum PSA
    - Adjust the measured serum PSA upwards by 40-50% for prostate CA screening \(^2\)
  - Increased frequency of high-grade prostate carcinoma
    - Direct induction or detection bias? \(^1\)
POST-FINASTERIDE SYNDROME

• Post-finasteride syndrome
  • Long-lasting sexual dysfunction
    • True persistent sexual dysfunction is controversial
    • Studies have shown it can last 5.4 months after stopping Rx
    • Incidence unknown

• Psychiatric effects (depression)
  • Relationship has not been defined
  • Multiple case series demonstrating depression in men treated with finasteride
    • Range from mild to severe

• Possible MOA:
  • Decreased testosterone
  • Decreased plasma and neurosteroid levels
    • Decrease dopamine, block GABAergic receptors, etc
ANDROGENETIC ALOPECIA TREATMENT - WOMEN

- Minoxidil 2% solution and 5% foam are FDA approved
- Oral contraceptives to suppress ovarian androgen production
  - Superior oral option for women of childbearing potential
- Spironolactone 100-200 mg/daily
  - Class D teratogen
  - Irregular menses, breast tenderness, hyperkalemia
- Oral finasteride
  - Dose of 1mg/day = ineffective in women
  - 2.5 to 5mg po daily are more effective (62% and 81% improvement, respectively)
  - Also teratogenic (abnormal male fetus genitalia, including hypospadias)
  - SE: GI upset, decreased libido, dry skin, acne
- Oral dutasteride in postmenopausal females only
DUTASTERIDE

- Inhibits BOTH type 1 and type II 5α reductase
- Off-label use for men that do not respond to finasteride
- Use only in postmenopausal women

- T1/2 = 5 weeks!
  - 6-8 hours for finasteride
  - Side effects are longer-lasting and more difficult to reverse
  - Reduced sperm count and sperm motility, potentially irreversible
LOW-LEVEL LIGHT THERAPY

• Laser hair removal can paradoxically trigger hair growth in surrounding skin
  • Photo-biostimulation of hair growth
• Patient combs a device (650-700nm) through the hair 2-3 times per week
  • Or wears a laser cap daily
• $200-3000

• Limited studies, lots of skepticism
HAIR TRANSPLANT

• From 1950s-1990s, hair transplants had a very unnatural appearance
  • “plugs” → 3-4mm grafts containing 15-30 hair follicles
• Current transplants use grafts containing 1-4 follicles
  • Individual follicular units
  • Taken from the occipital scalp → elliptical donor harvesting
• Recreate the hairline with grafts
• Patients are encouraged to continue minoxidil and/or oral finasteride after procedure
• Given progressive nature of AGA, another transplant session will likely be needed in 5-10 years
TELOGEN EFFLUVIUM

- Increased shedding of otherwise normal telogen hairs in response to a pathologic or normal physiologic change in health status.
TELOGEN EFFLUVIUM

- Hair loss happens ~3 months after systemic event
  - Length of the telogen phase
- No inflammation in pure TE
- Thinning typically involves entire scalp and may be seen in other regions of body (pubic hair, axillae)
- Gentle hair pull test will show many telogen hairs
  - Telogen hair = white bulb on the end (club hair)
- Trichogram will show >15% telogen hairs
  - 15-20% is suggestive, >20% is diagnostic
TELOGEN EFFLUVIUM CAUSES

- Shedding of the newborn (physiologic)
- Postpartum (physiologic)
- Chronic telogen effluvium
- Postfebrile (extremely high fevers, ie malaria)
- Severe infection
- Severe chronic illness (HIV, SLE)
- Severe psychological stress
- Postsurgical
- Endocrinopathies (thyroid or parathyroids)
- Crash or liquid protein diets
- Starvation/malnutrition
TELOGEN EFFLUVIUM CAUSES

- Drugs:
  - Discontinuation of OCPs
  - Retinoids or excess vitamin A
  - Anticoagulants (esp heparin)
  - Antithyroid (PTU, methimazole)
  - Anticonvulsants
  - Interferon-α-2b
  - Heavy metals
  - B-blockers
TELOGEN EFFLUVIUM TREATMENT

- Eventually complete hair regrowth is expected
- Can cycle a few times or become chronic (years)
  - Does not lead to baldness
  - If applicable, will reverse if causative illness or medication is eliminated

- If cause is unclear, laboratory workup should include:
  - TSH, T4
  - Hematocrit
  - Ferritin (treat if <40ng/dl)
- If last >6 months, scalp biopsy should be performed
  - r/o AGA
ALOPECIA AREATA (AA)

- Non-scarring patterned alopecia, most commonly presenting as circular areas of alopecia
- Hair-specific autoimmune disease involving T cells
- Prevalence in US of 0.1-0.2%
- Genetic factors play a role in susceptibility and severity
- Chronic relapsing nature of AA and its profound effect on physical appearance make this a distressing and life-changing event for most patients
ALOPECIA AREATA

- During acute phases, many lymphocytes “swarm” the hair bulb, yet the bulb always remains able to produce a new hair (=non-scarring)
  - Follicular stem cells remain viable
ALOPECIA AREATA

• Most commonly presents as round or oval patches of non-scarring hair loss
  • Short “exclamation point” hairs can be seen, particularly on margins
• Other patterns:
  • Totalis – loss of all scalp hair
  • Universalis – loss of all body hair
  • Ophiasis pattern (band-like pattern along periphery)
ALOPECIA AREATA – OPHIASIS PATTERN
ALOPECIA TOTALIS & UNIVERSALIS
ALOPECIA AREATA

- Non-pigmented hairs may initially be spared
  - “graying overnight”
- Hair loss is often rapid
- Nails involved in 10-20%
  - Pitting most common
  - Trachyonichia = sandpaper like roughness
ALOPECIA AREATA - PROGNOSIS

- 80% of patients presenting with a single bald patch, spontaneous regrowth occurs within 1 year.
- Recovery is possible for even alopecia totalis and universalis.
- Poor prognostic factors:
  - Extensive disease
  - Bald patches >1 year
  - Ophiasis pattern of hair loss
  - Nail involvement
  - Onset of AA before puberty
  - Family members with AA
  - Personal or family h/o other autoimmune disease
  - Down syndrome
ALOPECIA AREATA TREATMENT

- Patchy disease –
  - Topical or intralesional steroids
    - Clobetasol solution
    - 2.5-10 mg/kg triamcinolone injected into lesions every 4-8 weeks
  - Topical minoxidil
  - Topical anthralin 0.5-1% cream or ointment daily
ALOPECIA AREATA TREATMENT

- “Dependable and safe treatment for extensive disease has yet to be found, although spontaneous recovery is possible” (Bologna text, 4th ed, p 1174)
- 80% response to high dose steroids
  - 40mg triamcinolone IM monthly
    - Limit to 2-3 months
  - Daily prednisone or dexamethasone, tapered over 6-8 weeks
- 50% will relapse with dose reduction and cessation of therapy
- Long-term maintenance therapy with corticosteroids is rarely justified
- Unfortunately, steroid-sparing meds (azathioprine, mtx) are unreliable
ALOPECIA AREATA TREATMENT

- “Dependable and safe treatment for extensive disease has yet to be found, although spontaneous recovery is possible” (Bologna text, 4th ed, p 1174)

- Simvastatin and ezetimibe
  - Antiinflammatory via HMG CoA Reductase inhibition
  - Case series and case reports in literature to support use in AA
  - Likely not helpful for long-standing disease, may prevent relapses
ALOPECIA AREATA TREATMENT

• “Dependable and safe treatment for extensive disease has yet to be found, although spontaneous recovery is possible” (Bologna text, 4th ed, p 1174)

• Topical immunotherapy (diphencypronone or squaric acid) is an option, but unimpressive
  • Many reliable online protocols, www.naaf.org
ALOPECIA AREATA TREATMENT

• “Dependable and safe treatment for extensive disease has yet to be found, although spontaneous recovery is possible” (Bologna text, 4th ed, p 1174)

• Tofacitinib and ruxolitinib (JAK/STAT pathway inhibitors)
ALOPECIA AREATA TREATMENT

- All patients should be directed to the National Alopecia Areata Foundation
- www.naaf.org
SCARRING ALOPECIA

- Lymphocyte mediated
  - Lichen planopilaris (LPP)
  - Frontal fibrosing alopecia (FFA)
  - Central centrifugal cicatricial alopecia (CCCA)
- Pauci-inflammatory
  - Traction alopecia
  - Pseudopelade of Brocq
- Neutrophil Mediated
  - Folliculitis decalvans
  - Dissecting cellulitis
  - Acne keloidalis nuchae
LYMPHOCYTE MEDIATED ALOPECIA

- Central centrifugal cicatricial alopecia (CCCA)
- Lichen planopilaris (LPP)
- Frontal fibrosing alopecia (FFA)
FRONTAL FIBROSEING ALOPECIA AND LICHEN PLANOPILARIS

• Likely the same disease with different presentation and different disease associations

• Frontal Fibrosing alopecia
  • First described ~20 years ago
  • Incidence seems to be increasing
  • Scarring hair loss of anterior hair line
    • Often associated with loss of the lateral eyebrows
  • Etiology/pathogenesis unknown

• Lichen planopilaris
  • +/- lichen planus on the body
FRONTAL FIBROUSING ALOPECIA AND LICHEN PLANOPILARIS

• Inflammatory scarring alopecia with several different patterns of hair loss
  • Often presents with pruritus and tenderness
  • Most often with several scattered foci of partial hair loss with perifollicular erythema, follicular spines and scarring
• Most common cause of end-stage cicatricial alopecia
FRONTAL FIBROSYING ALOPECIA/LICHEN PLANOPILARIS

- Treatment of limited disease
  - Inject the margin with intralesional triamcinolone (5-10mg/cc)
    - Consider eyebrow injections
  - Fluocinolone oil or Clobetasol solution
  - Topical tacrolimus 0.1% compounded in cetaphil lotion
FRONTAL FIBROSYNG ALOPECIA/LICHEN PLANOPILARIS

- Treatment of extensive or progressive disease
  - Can be very resistant to treatment!
  - Tier 1
    - Hydroxychloroquine 200 mg BID
    - Finasteride/dutasteride (for FFA variant)
  - After 6-12 months if s/s persist go to tier 2

- Tier 2
  - Methotrexate
  - Mycophenylate mofitil
  - Cyclosporine
  - Retinoids

*Pioglitazone 15 mg daily → ineffective
CENTRAL CENTRIFUGAL CICATRICIAL ALOPECIA (CCCA)

- Central hair loss over the superior scalp
- Chronic, progressive disease with eventual burn out
- Predominantly centered on the crown or vertex
- Expands in a roughly symmetric fashion
  - “active” zone is periphery
CENTRAL CENTRIFUGAL CICATRICIAL ALOPECIA (CCCA)

- Responsible for more cases of scarring alopecia in African Americans than all other types of scarring alopecia combined
  - Related to genetics, styling practices
  - Mostly women
- Caustic hair care products and/or hair styles causing traction aggravate disease
  - But this alone cannot fully explain pathogenesis
CCCA TREATMENT

• Topical and/or intralesional steroids:
  • Inject the margin with IL triamcinolone
    • Careful to prevent hypopigmentation
  • Fluocinolone oil or Clobetasol solution
  • Topical tacrolimus 0.1% compounded in cetaphil lotion
• Long-term tetracycline abx (Doxy or mino)

• For resistant disease:
  • Oral rifampin + clindamycin
TRACTION ALOPECIA
TRACTION ALOPECIA

- Related to styling practices
- Tight braids, weaves, or ponytails worn over years
- PERMANENT scarring alopecia
- If caught early and styling practices are changed, some regrowth may occur

- Encourage natural hair styles and educate
- Treatment: rogaine; change in styling practices; wigs
SCARRING ALOPECIA- NEUTROPHIL MEDIATED

- Acne keloidalis nuchae
- Dissecting cellulitis
ACNE KELOIDALIS NUCHAE
ACNE KELOIDALIS NUCHAE

- Most common in black men, but can occur in women and Caucasians
- Can occur in conjunction with CCCA
ACNE KELOIDALIS NUCHAE

- May be exacerbated by protective headwear or CPAP
- D/c shaving affected area
  - “Mechanical folliculitis”
- Topical and intralesional steroids
- doxycycline or minocycline
- Surgical removal (be wary of)
DISSECTING CELLULITIS
DISSECTING CELLULITIS

- Often part of a follicular occlusion syndrome including acne and hidradenitis
- Deep, boggy, suppurative scalp lesions
- Treatment: tetracyclines, retinoids, intralesional corticosteroids
- End stage: surgical approaches
REFERENCES


