Getting to the Root of Hair Loss Disorders/Alopecia Treatment

Matt L. Leavitt, D.O., FAOCD
Diplomate ABHRS
Founder, Chairman, CEO
Advanced Dermatology & Cosmetic Surgery
Founder, Medical Hair Restoration
Executive Medical Advisor, Bosley
Associate Clinical Professor, NOVA, KCUM
President, Hair Foundation
Chairman, KCU-ADCS Orlando Dermatology Residency

New Wave Dermatology
April 12, 2018
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Disclosures

No relevant disclosures
Objectives

• Discuss the hair cycle
• Describe an evaluation for hair loss
• Distinguish between scarring and non-scarring alopecia
• Compare and contract treatments for pattern vs. disease-related hair loss
Hair Physiology: Two Major Types of Hair

• **Terminal**
  – Large in size
  – Pigmented
  – Frequently medullated
  – Can grow to considerable length
  – Bulb in subcutaneous fat
  – Typical hair of scalp

• **Vellous**
  – Small in size
  – Lightly pigmented
  – Non-medullated
  – Do not grow more than 2 cm in length
  – Hair bulb in reticular dermis
Hair Growth Cycle

- Hair growth generally non-synchronized
- Anagen or Growth Period - 90% of follicles
  - 2-8 year period of activity cellular proliferation
- Catagen – 1-2% of follicles
  - Transition Stage
    - bulbar portion of follicle is almost totally degraded 2-4 weeks
- Telogen or Resting Stage-10% or less (usually 6-8%)
  - 2-4 months
  - “Club” hair
  - Period of almost complete inactivity
Hair Facts

- **Numbers**
  - Average number of hairs on the scalp is estimated to be 100,000.
  - Up to 100-150 hairs are postulated to be shed every day.
  - Approximately 50% loss is usually necessary to detect visible hair loss.
Alopecia

Scarring/Cicatricial
- Primary
- Secondary
  - Infection
  - Malignancy
  - Trauma

Non Scarring
- Focal
- Diffuse
Non-Scarring Alopecia

• Diffuse
  – Even distribution of hair loss in area of loss

• Focal
  – Unifocal or multifocal and limited or widespread
Cicatricial Alopecia

- Visible loss of follicular ostia
- Destruction follicle on H&E exam
- Inflammatory infiltrate targets hair follicle
- Replacement with fibrous tissue
- Progressive permanent hair loss
Scarring Alopecia
1° Scarring Alopecia Classification

1. Lymphocytic
   Chronic Cutaneous Discoid Lupus Erythematosus
   Lichen Planopilaris
   Classic Pseudopelade (Brocq)
   Central Centrifugal Cicatricial Alopecia
   Alopecia Mucinosa
   Keratosis Follicularis Spinulosa Decalvans

2. Neutrophilic
   Folliculitis Decalvans
   Dissecting Cellulitis / Folliculitis

3. Mixed
   Folliculitis Keloidalis
   Folliculitis (acne) Necrotica
   Erosive Pustular Dermatosis

4. Non-Specific
Lymphocytic Scarring Alopecias

Discoid Lupus Erythematosus (DLE)

Females 20-40 years of age

50% of cases have scalp involvement

5-10% of patients with DLE will develop systemic lupus

Extra cranial lesions can help with Dx
Scarring Alopecia 2/2 Lupus

~11 mos s/p 1st transplant
Lichen Planopilaris (LPP)

- Females 40-60 years of age
- Extra cranial lichen planus is present 25%-50% patients
- Etiology is unknown- possible antigenic stimulus
- Three types of lesions are present:
  1) Perifollicular erythematous/violaceous papules & spinous/follicular keratotic papules
  2) Atrophic, smooth shiny patches
  3) Typical lesions of LP- generally not seen
Lichen Planopilaris

- Multifocal/scattered areas throughout the scalp is a typical distribution
- Disease activity is limited to the hair bearing periphery of scarred patches
- Slowly progressive disease that can lead to extensive area of hair loss
Frontal Fibrosing Alopecia

- Specific patterned clinical variant of LPP affecting the frontal hairline
- Postmenopausal women >40 years of age, rarely men
- Progressive alopecia
- Creates a band like area of alopecia 3-8cm wide contrasts with photodamaged skin
- Lesions of LPP can be seen at hairline

Frontal Fibrosing Alopecia

- Eyebrows thinned/absent
- Axillary hair loss has also been reported
- Lesions of extra-cranial lichen planus are not seen
Psuedopelade of Brocq

- Multiple meanings depending on which study or which person referencing
- Primary Psuedopelade of Brocq - Idiopathic
- Secondary Psuedopelade of Brocq - Final stage of several scarring disorders (LPP, DLE)

Psuedopelade of Brocq = LPP?
Pseudopelade of Brocq

- Adult females
- 1-2 cm atrophic, oval-round, ivory plaques: “Footprints in the Snow”
- May see isolated and grouped kinked hairs
- Minimal to no inflammation with few symptoms
Lichen Planopilaris Treatment

Steroids:

- **Topical** – ex. Clobetasol 0.05% soln, oint, foam pulsed: 2 week on, 1 week off
- **Intralesional** – Triamcinolone 4-10 mg/cc
- **Intramuscular** - Triamcinolone) 40-80 mg
Lichen Planopilaris Treatment

Other Treatments:
- Hydroxychloroquine
- Doxycycline
- Finasteride 1-2.5 mg
- Minoxidil 5%
- Pioglitazone
Lichen Planopilaris

- Evaluate for drug-related (gold) and Hepatitis C
Lichen Planopilaris / Frontal Fibrosing Alopecia

s/p 2 eyebrow transplants
Lichen Planopilaris / Frontal Fibrosing Alopecia

s/p 2 hair transplants
Female Pattern Hair Loss

~10 mos s/p 1st hair transplant
Central Centrifugal Cicatricial Alopecia (CCCA)

- Follicular Degeneration Syndrome (FDS), “Hot Comb Alopecia”
- African American women in 30’s-40’s
- Originally thought to be secondary to liquified hot petrolatum and other hair care practices
- Trauma due to long-standing traction
- Etiology is still unknown
- Also associated with pattern CCCA:
  - FDS
  - Pseudopelade
  - Folliculitis decalvans
  - Tufted hair folliculitis
Central Centrifugal Cicatricial Alopecia

- Progressive symmetric centrifugal scarring that starts at the midline/vertex scalp
- “Pins & Needles”, pruritus, tenderness
- Minimal erythema
- Scalp can be smooth and shiny
- Perifollicular hyperpigmentation may be seen
- Incomplete alopecia
Central Centrifugal Cicatricial Alopecia

• Treatment
  – Topical and Intralésional Steroids
  – Calcineurin Inhibitors (i.e. Tacrolimus oint)
  – Tetracyclines (i.e. Doxycycline)
  – Cyclosporine
  – Hydroxychloroquine
  – Hair Transplantation (counsel about realistic expectations)
  – Wigs and Hair Pieces
  – Topical Camouflage Agents (i.e. powders and sprays)
  – Transition to natural hairstyles that limit tension, heat, and chemicals
Treatment
Primary Cicatricial Alopecia

– Lymphocytic
  • Steroid
    ▪ High potency topical steroid – Clobetasol, Betamethasone, Ultravate
    ▪ Oral steroid – Prednisone
    ▪ Intraloesional or Intramuscular steroid - Triamcinolone
  • Topical non-steroidal anti-inflammatory - Tacrolimus oint
  • Low dose antibiotic - Doxycycline, Minocycline
  • Oral retinoids - Acitretin
  • Immunosuppressants – Cyclosporine, Mycophenolate mofetil, Hydroxychloroquine
  • Excision of burned out small localized lesions (koebnerization is possible)
Neutrophilic Cicatricial Alopecias
Folliculitis Decalvans

- Pustular eruption of the scalp that leads to patchy scarring alopecia
- M=F, younger
- Staph. aureus is commonly cultured
- Abnormal host response to Staph aureus??
Peripheral disease activity
Dissecting Cellulitis

- 80% patients are African-American age 18-40
- Initially develop a follicular pustule occipital/vertex scalp that transforms into a painful nodule
- Multifocal disease with interconnecting tracts
- A cerebriform appearance develops
- Depressed, hypertrophic, keloidal scarring
- Tx: Isotretinoin 1mg/kg
  Oral steroids
  Dapsone
  Antibiotics- keflex, bactrim
Treatment
Primary Cicatricial Alopecia

- Neutrophilic
  - Antibiotics
    - Clindamycin/Rifampin for staph
    - Alternative antistaphylococcal antibiotics (top and po)
  - Oral retinoids – Isotretinoin
  - Incision and Drainage
  - Laser Ablation
  - Intraleisonal steroids
  - Antitumor necrosis factor biologic agents
  - Excision of burned out small localized lesions (koebnerization is possible)
Mixed Cell Scarring Alopecias
Acne (Folliculitis) Keloidalis

- Occipital scalp and nape of neck
- Flesh colored discrete papules
- May be pustules, nodules or plaques
- May form abscesses or sinuses

**Treatment:**
- IL steroids
- Oral Antibiotics: tetracycline
- Topical antibiotics (clindamycin) and steroids
Mixed Cell Scarring Alopecias

Folliculitis Keloidalis
# 2° Cicatricial Alopecia

<table>
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<tr>
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<td>Miscellaneous</td>
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Non-Scarring Hair Loss
Differential Diagnosis of Hair Loss

- Diffuse
  - Breakage
    - Anagen effluvium
    - Hair shaft disorder
    - Physical or chemical processing
  - Telogen effluvium
  - Androgenetic alopecia (women)
  - Alopecia areata, totalis or universalis
  - Loose anagen syndrome
Differential Diagnosis: Non-Scarring Cont’d.

- Focal
  - Infection
  - Traumatic
  - Alopecia Areata
  - Hair breakage
  - Androgenetic alopecia (men and women)
  - Developmental
Anagen Effluvium

- Direct toxic insult to rapidly dividing keratinocytes
- Dystrophic hairs from abrupt interruption of hair growth
- Copious hair loss
- Common causes
  - Radiation
  - Plants
    - Lecythis Ollaria
    - Loucaena glauca
  - Chemotherapy
  - Colchicine
  - Heavy metals
    - Thallium
    - Mercury
    - Arsenic
Telogen Effluvium

- History and Exam
  - Visible and rapid progression of hair loss
  - Increase from 10% to 30-50% telogen hairs
    - 150-700 hairs / day
  - Hairs easy to comb out
  - Lags inciting event by approximately 3 months
Causes of Telogen Effluvium

Acute Stress (hemorrhage)
Childbirth (postpartum)
Chronic Systemic Illness
  - Cancer
  - Leukemia
  - Hodgkin’s Disease
  - Tuberculosis
  - Cirrhosis
Crash Dieting

Chronic Iron Deficiency
Psychogenic Stress
Thyroid Disease

Drugs
  - Allopurinol (Zyloprim)
  - Clofibrate (Atromid-S)
  - Cocaine
  - Warafin (Coumadin)
  - Heparin
  - Oral Contraceptives
  - Propylthiouracil

Febrile Illness
  - Influenza
  - Lobar pneumonia
  - Pertussis
  - Scarlet Fever

Source: Women and Hair Loss, Dr. Matt Leavitt, p. 44, 2005
Hair Loss Questionnaire: Hair History

• How long ago did you first notice hair loss?
• Since this started, have you been losing hair at the same rate or at a faster rate?
• How many hairs are you losing daily?
  – Do you feel you have been shedding excessively? Do you feel your scalp is slowly thinning out without losing excessive numbers of hair?
  – Which did you notice first, shedding or thinning?
• Has your hairline or temporal areas receded?
Hair History Continued

• Are you losing hair from the entire scalp, or over the top of the scalp? Does your hair seem dull, brittle or uncombable?

• Is your hair lost from the roots, breakage or both?
  – Do you have symptoms, i.e. itching, scaling, etc.

• Are you losing hair from any other parts of your body?

• Do you have a habit of twirling, scratching or pulling your hair?

• List any family members who have hair thinning, receding or loss?
Hair History Continued

- List all health problems, current or in the past, including any surgeries
- List all allergies
- Do you have a history of:
  - Pregnancy
  - Infertility
  - Thyroid Disease
  - Iron Deficiency
  - Discharge from nipples
  - Diabetes
  - Severe Acne
  - Menstrual Irregularity
  - Prolonged Fever
  - Anemia
  - Ovarian Cysts
  - Excessive Tiredness
  - Increased facial hair
Hair History Continued

- Have you ever been pregnant?
  - Number of pregnancies? Number of children?
  - Any complications during pregnancy?
  - Did you lose any hair during/after pregnancy?
  - Did you lose any hair after any pregnancy or surgery?

- Do you take birth control pills?

- Have you donated blood in past 3 years

- Do you have a vegetarian diet

- Are you too hot? Too cold?
Hair History Continued

- In the past year have you had
  - Surgery
  - General Anesthesia
  - Serious Injury
  - Childbirth
  - Prolonged high fever
  - Weight loss/gain
  - Other
  - Marriage
  - Divorce
  - Death in family
  - New Job
  - New residence
Hair History/Medications

- Cholesterol
- Parkinsons
- Ulcer
- Anti-coagulants
- Gout
- Anti-arthritis
- Drugs from Vitamin A
- Anti-seizure
- Miscellaneous

- Tricyclic Anti-depressants
- Beta Blockers
- Calcium Channel blockers
- Non-steroid Anti-inflammatory
- Aspirin products
- Anabolic steroids
Telogen Effluvium

- Visible telogen shedded hairs
  - Root of hair appears bulbous or onion-shaped
- Hair dull and limp
Telogen Effluvium

- Most frequent cause postpartum (telogen gravidurum)
- $1/3 - \frac{1}{2}$ postpartum mothers report mild-to-moderate hair loss
- Corrects self 6-18 months
- 90% cases correct without intervention
- Greater than 6 months – chronic telogen effluvium
Telogen Effluvium

- Bi-temporal thinning may be present
- Positive Pull Test for telogen (club) hairs
- Shorter, regrowing frontal hairs may be seen in resolving state
- Chronic:
  - Subtle onset in women 40-60’s
  - Continuous shedding with fluctuations
  - Normal appearing densities
  - Shortened anagen phase
Work Up

- CBC, CMP
- TSH
- FSH/LH, Free and Total Testosterone, DHEA-S
- Iron, Iron Binding Capacity, Transferrin
- Ferritin: best test for Iron deficiency
  - Best between 50-70µg/L
Treatment of Telogen Effluvium

- Reassurance -- tendency towards resolution
- Minoxidil 5%
- Clobetasol pulsed, 2 weeks on, 1 week off
- IL triamcinolone 5 mg/cc
- Correct any reversible causes of hair loss
  - Anemia, thyroid abnormality, dietary deficiencies (inadequate protein, low iron, B12, folic acid), medications
- Alternative styling to mask defects
- Gentle hair care
Non-Scarring Androgenetic Alopecia

Male Pattern AGA

Female Pattern AGA
Androgens- Women

- Role in women is not completely understood
- Women can have baldness in the presence of normal and high androgen levels
- Pattern is different in women
- No complete areas of baldness
- Don’t respond equally as men do to anti-androgen therapies
Androgens- Women

- Aromatase enzyme converts testosterone to estrone, estradiol and estrase (E’s)
- The E’s increase SHBG which bind androgens
- Women treated with Aromatase inhibitors experience FPHL
- Aromatase levels are decreased in balding scalp vs non-balding areas
- Women have significantly higher levels of scalp aromatase compared to men
Female Pattern

- **History**
  - Family History (may be difficult to pinpoint)
  - Slow onset, slow progression
  - Rate of loss is generally stable
  - Hair loss is from root
Hair Loss: Physical Examination and Tests

- **Ludwig Scale**: Stage I, II, III
- **Savin Scale**: D 1, 2, 3, 4, 5, 6
- **Scalp**
  - Normal
  - Erythema
  - Papules/pustules
  - Excoriations
  - Scaling
  - Focal hair loss
  - Scarring
  - Follicular plugging
- **Density**
  - Normal
  - Sparse-Crown
  - Sparse-Diffuse
  - Mild thinning-crown
  - Mild thinning-diffuse
Hair Loss: Physical Examination and Tests

- Hair Pull Test: Positive / Negative
- Broken Hairs: Absent / Present
- Laboratory: KOH Exam, Microscopic Trichogram, Hair Shaft Exam
- Diagnosis
Hair Loss: Physical Examination and Tests

- Plan
  - 3 day hair count requested
  - 5 mm punch biopsy
  - Blood tests:
    - CBC with Diff
    - Protime with INR
    - Free testosterone
    - TSH, T4 Free
    - Ferritin
    - HIV
    - DHEAS
Female Pattern: Examination

• Hairline and temporal mostly maintained
  – 90% maintain temple
  – 85% maintain hairline
• Most follow Ludwig Pattern with widening part
Female Pattern: Examination

• Miniaturization
  – Finer, smaller, slower-growing, less pigmented hairs
  – Thinning more common early than noticeable areas of hair loss
  – Ratio of less than 4 terminal hairs to 1 miniaturized hair
  – Telogen
Female Pattern – Ludwig Scale

Type I - Mild
Type II - Moderate
Type III - Severe
Male Pattern Androgenetic Alopecia
Male Pattern Androgenetic Alopecia
Common MPHL Patterns: Norwood Classification
Etiology of Male Pattern Hair Loss

- **Hormones**: Dihydrotestosterone (DHT) is a key factor in the development of male pattern hair loss (MPHL) among genetically predisposed men.
- **Genetics**: Autosomal dominant and/or polygenic. Inherited from either or both parents.
- **Hence the term**: Androgenetic Alopecia.
Role of DHT in Male Pattern Hair Loss

- T is converted to DHT by the enzyme 5a-Reductase
  - Two isozymes of 5a-R, Type I and Type II
  - Type II 5a-R localizes to hair and prostate
- DHT levels are elevated in balding scalp tissue
- Over time, increased DHT causes normal “terminal” hairs to become “miniaturized” hairs, the hallmark of MPHL
Androgens: 5 α Reductase

- Two Isoforms 1 and 2
- Type 1: Sebaceous Gland
  Pilosebaceous Apparatus
- Type 2: Prostate Gland
  Outer Root Sheath
  Dermal Papilla
Progression of MPHL

• Hair cycles are shortened in MPHL
  – Cycles may shorten to as little as 6-8 months
  – Anagen (growing) phase reduced to 6-8 weeks
• Hairs “miniaturize” over time to become progressively thinner, finer, shorter, and less pigmented
• Men may have already lost 50% of hair count before it is noticed

Progression of MPHL

Healthy hair (thick, actively growing, and fully pigmented) → Progressive hair thinning (thinner, shorter, and less pigmented)
Minoxidil

• Medical - Minoxidil
  – Mechanism of Action
    • Non-specific biological response modifier
    • Acts directly on viable sub-optimally functional follicles: potassium channel
Mechanism Of Action

- Hair cycle stimulation
- Opening of potassium channels
- Vascularization (VEGF)
- Cell proliferation

Patient Treatment

• Medical - Minoxidil
  – Benefits
    • Enlargement of miniaturized follicles -- increase diameter
    • Conversion of telogen hair to anagen
    • Prolongs anagen -- slows progression of hair loss
Using Minoxidil: Practical Suggestions

• Apply Minoxidil topical solutions once daily
• Towel dry or blow dry hair before application
• Apply Minoxidil directly on the scalp
• Wash hands after application
• Apply 5 minutes before using styling aids
• Do not shampoo or swim for 4 hours after application
• Let solution dry before going to bed
Propecia

Why Propecia Works
Effects of Androgens on Scalp Hair in Men

Androgen-sensitive regions

Long, thick pigmented terminal hair

T DHT

5a-Reductase

→ Short, fine unpigmented miniaturized hair
Effect on Hair Count

Mean Change in Hair Count from Baseline (±1 SE)

- Finasteride 1 mg
- Placebo

Year

Baseline 1 2 3 4 5

Finasteride (n) 679 433 351 291 219
Placebo (n) 672 47 32 20

Δ=107  Δ=138  Δ=146  Δ=216  Δ=277

# Propecia® 5 Year Safety Profile

## Any sexual AE

<table>
<thead>
<tr>
<th>Condition</th>
<th>Propecia (N=323)</th>
<th>Placebo (n=23)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any sexual AE</td>
<td>2 (0.6)</td>
<td>0</td>
</tr>
<tr>
<td>Libido decreased</td>
<td>1 (0.3)</td>
<td>0</td>
</tr>
<tr>
<td>Erectile dysfunction</td>
<td>1 (0.3)</td>
<td>0</td>
</tr>
<tr>
<td>Ejaculation disorder</td>
<td>0</td>
<td>0</td>
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<tr>
<td>Sexually-related AE</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Resulting in discontinuation</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>
Post-Finasteride Syndrome

- **Sexual SEs** – decreased labido, erectile dysfunction, decrease in semen volume...
- **Physical SEs** – gynecomastia, fatigue, muscle atrophy, tinnitus
- **Mental SEs** – memory impairment, depression, suicidal ideation

http://www.pfsfoundation.org
Uncertainties in Adverse Event Reporting

- Significant amounts of data regarding SEs stems from meta-analyses which tend to be weak in assessing clinical harm in several clinical trials.
- Men may be unlikely to spontaneously report sexual issues especially if asked by a female provider, nurse, or assistant.
- Available finasteride toxicity data from clinical trials is often biased, of poor quality, and limited.
- Clinical trials have provided insufficient information to establish safety.
- The extent of persistence of sexual side effects is not clear due to the length of time patients are followed in a study.

FDA-Mandated Label Changes for Finasteride

“A revision to the (Finasteride) label to include libido disorders, ejaculation disorders, and orgasm disorders that continued after discontinuation of the drug…

A revision to include a description of reports of male infertility and/or poor semen quality that normalized or improved after drug discontinuation.”

http://www.pfsfoundation.org
Low Level Laser Therapy (LLLT)

• Low-power lasers or light-emitting diodes (LEDs) are applied to the surface of the skin to stimulate cell function for improved hair growth (this is known as Photobiomodulation (PBM))
• The wavelength is typically between 630-670nm in the red visible light spectrum

https://www.bosley.com
The following theories of the mechanisms of action of LLLT have been published.

- Anagen Induction
- Increased Vascularization
- Reduction of Inflammation
- Increase ATP Synthesis
- Regulation of Oxidative Stress
- Delay of Follicular Apoptosis
- Reverses Miniaturization
Clinical Studies on Efficacy and Safety

The efficacy and safety of the HairMax LaserComb® in the treatment of androgenetic alopecia (AGA) in males and females has been demonstrated in 7 clinical trials involving 460 subjects.
After approximately six months of treatment these clinical images provide evidence of (1) reverse miniaturization of vellus hair, (2) increased number of hairs per follicular unit and (3) revival of dormant hairs. These results are both medically and scientifically significant.
Hair Transplantation
The Procedure: Design

- Premium on maintenance of hair and preservation of donor hair
- Irregular pattern in hairline
The Procedure: Design

- Grafts very close together
The Procedure: Design

- No scarring
- Natural at every stage
- No evidence of transplants
The Procedure

- Ellipse
  - Advantage -- less transsection of hair
  - Efficient utilization of donor and greater availability of donor
The Procedure

Sutured donor area

Hair combed over suture at time of surgery
Follicular Unit Extraction (FUE)
Follicular Unit Extraction (FUE)

Immediate Post-Op
FUE Immediately After Donor Extraction
FUE

Before

4 days Post-Op

After
Manual FUE Instruments

Cylindrical Punch in Versi Handle

7mm

Follicular Unit Extraction (FUE)
Robotic-assisted FUE
FUE Complication

- Overharvesting/Donor Depletion
The Procedure: Graft by Section

- Dissection
  - Follicular Units
  - Microscopes/magnification
The Procedure: Graft by Section

- Follicular Bundle (1-3 hairs)
- grafts cut so naturally occurring bundles are maintained
The Procedure: Site Creation/Recipient

- Insertion
  - Minde
  - SP 90
  - Sharp Point
  - 18 gauge
Alopecia Areata

- 1-2% of population
- Non scarring alopecia with a lifetime risk of 1.7% M=F
- 60% of patients develop the first patch before 20 years of age
- Abrupt onset and asymptomatic
- Round-oval smooth bald patches
- “Exclamation point hairs” - broken hairs tapered proximally
- Majority of patients will regrow within 1 year
- 50-80% sporadic
Alopecia Areata (AA): Exclamation Point Hair

- Onset of alopecia areata
  - Growth stops, anagen arrests
  - Follicle breaks at skin level
  - Trichorrhexis nodosa type fracture
  - Hair pull is positive with dystrophic and telogen hairs
Alopecia Areata

- Autoimmune etiology
- Combination of environmental and genetic factors may initiate hairloss, but an exact cause is unknown
- High frequency positive family hx (10-42%), esp. in early onset patients
- Class II antigens have been associated with early and more extensive disease
- Filaggrin gene mutation – alopecia totalis or universalis
Alopecia Areata: Etiology / Autoimmune

• Peribulbar infiltrates
  – Composed mainly of T-cells
    • T-cells are oligocional / autoreactive
• Follicular components / autoantibodies
• AA linked with autoimmune thyroiditis
Alopecia Areata

• Treatment
  – Corticosteroids
    • Beneficial effect
  – Contact sensitizers
    • Also have a positive effect
  – Jak inhibitors
JAK Inhibitors

JAK-STAT Inhibitors inhibit janus-associated kinases (JAK) 1, 2, 3, leading to disruption of cytokine and growth factor signaling, thus interfering with the inflammatory cascade

Janus Kinases: An Ideal Target for the Treatment of Autoimmune Diseases
Author links open overlay panel, Massimo Gadina, Journal of Investigative Dermatology Symposium Proceedings Volume 16, Issue 1, December 2013, Pages S70-S72
Alopecia Areata

- Patterns:
  1) Oval/round - most common
  2) Reticular
  3) Ophiasis - parietal temporo-occipital
  4) Diffuse - entire scalp
- Alopecia totalis - 100% scalp
- Alopecia universalis - 100% scalp and body
Alopecia Areata

- Nail dystrophy may be present
  - 10-66%
  - nail pitting most common
  - other nail findings
    - Longitudinal ridging
    - Brittle
    - Onycholysis
    - Periungual erythema
    - Onychomadesis
- 7-10% patients develop severe chronic form
Alopecia Areata: Treatments

• Corticosteroids
  – Beneficial effect
• Contact sensitizers
  – Also have a positive effect
• JAK inhibitors
• Topical, IL, and Systemic Steroids
  Topical tacrolimus
  Topical syntisitizers- DNCB, Squaric Acid
  PUVA
  Cyclosporine
Disease Associations

- Atopy
- Autoimmune diseases
- Downs and Turner Syndrome
- Thyroid disease
Alopecia Areata
Exclamation Point Hairs
Chemical Alopecia

- Tints, bleaches, straighteners, permanents used too frequently or improperly

- Long-term chemical damage can cause scarring
Hair Care Questions

- How often do you shampoo
- List the products you use on your hair
- Do you bleach or color your hair
- Do you use strengtheners, pomades, relaxers, perms?
- Have you changed your hairstyle in last 6 months
- Do you wear extensions, fall or wig
Hair Care Questions

- Do you braid, plait, tease, wear a bun / ponytail
- Do you use rubber bands, hair pins, barrettes or other ornaments
- Do you use hot or sponge rollers?
- Do you use curling iron?
- Do you blow dry your hair
Traction Alopecia

- Hair styles that put excess tension or pull hair
  - Most common among African Americans
  - Hair breakage and/or patches of baldness
  - May see erythema, scaling, pustules
  - Most common in frontal or temporal areas
  - Persistence of short hairs along anterior margins
  - Long-term traction can cause scarring
Trichotillomania

- Obsessive-compulsive
- Irregular patches of alopecia with broken off hairs of irregular length

Treatment:
  - counseling
  - behavior change
  - SSRI’s
• Additional slides for education
• Will not be presented
Hair Shaft Abnormalities

- Increased Fragility
  - Monilethrix
    - Shaft has regular and internodes (resembles pearl necklace) with internodal fragility, leading to breakage and alopecia
    - Autosomal dominant, variable pinstruc
    - Mutation in hair keratin (hHb6) in cortex
    - Hair breaks almost flush with skin
Hair Shaft Abnormalities

- Pseudomonilethrix
- Trichorrhexis congenita
  - Most common
  - Congenital or acquired
  - May have normal hair at birth but within few months breaks, variable lengths, and alopecia
  - Associated with mental retardation and argininosuccinic aciduria
  - Due to in-born error in urea synthesis
  - Brush-like aspect at the extreme frontage of nodule
Hair Shaft Abnormalities

• Increased fragility
  – Pili torti
    • Flattened hair shaft twisted through 180 degrees on its own longitudinal arms
  • Two types
    – Early onset Ronchese type
    – Late type Beare type
• Associations
  – Menkes
  – Crandall or Björnsteds syndrome
  – Bazeks
Miscellaneous Hair Shaft

- Peripilar cast – pseudonits
  - Tubular masses of amorphous material of varied length and diameter
  - Seen with psoriasis, seborrhea
  - Distinguished from true nit by lack of mobility and because they uniformly surround hair
- Deposits lacquer, paint, glue
  - Extraneous material throughout scalp hair but localized to areas of contact
  - Unlike peripilar cast deposits cannot easily be moved
Tinea Capitis

• Fungal infection which predominantly affects children
  – Most common fungal infection in children 4 and 7 yrs
• Caused by 2 types of organisms:
  – Trichophyton, Microsporum
• More than 95% of tinea capitis cases are caused by T. tonsurans (anthropophilic dermatophyte) in the US
  – The remaining 5% are usually caused by M. canis (zoophilic organism).
• Favus
  – Usually caused by T. schoenleinii
  – Distinguished by hyphae in parallel arrangements to the hair
  – Air spaces can be seen within the shaft
  – Patches develop into scutula-cup shaped crust
Infections: Ecotothrix

- Caused by hyphae on the hair shaft that produce arthroconidia
  - Transmitted most often by infected animals
    - Most common is microsporum canis
      - Broken hairs with fine powdery dandruff
    - Results in destruction of cuticle
  - Using Woods lamp may show
    - Fluorescent – microsporum spp
    - Non-fluorescent – trichophyton rubron and some microsporum spp
Infections: Endothrix

- Spread person-to-person
- Arthroconidia found in hair shaft
- Does not fluoresce under Woods lamp
- Presents form black dot to patchy alopecia to kerion
- Black dot result of invasion in shaft which breaks off at surface
- Usually caused by T. tonsurans in USA and T. violaceum subtropical
- Small, chronic with only few hairs infected