Hair Loss 2009

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Patient is losing her hair and is very distraught over balding.
Diffuse Hair Loss

• What next???
  – 1. DO A SCALP BIOPSY
  – 2. PRESCRIBE MINOXIDIL
  – 3. DO A SCALP FUNGAL CULTURE
  – 4. DO A PULL TEST
Diagnosis Is ??????

- 1. Anagen alopecia
- 2. Catagen alopecia
- 3. Stress hair loss
- 4. Telogen effluvium
What Next ???

• 1. Prescribe minoxidil
• 2. Take a history
• 3. Place patient on Yasmin OCs
• 4. Do hormone workup with appropriate lab tests
• 5. Refer to dermatology
Of the following choices, which one(s) is/are most pertinent to telogen effluvium?

- a. Has a 3 week old baby
- b. Gained 30 pounds in the past 3 months
- c. History of thyroid disease
- d. A and C
- e. All the above
Next Patient On Your Schedule Is A Child With Unruly Hair
Your Working Diagnosis Is??

- a. Breakage from a hair shaft abnormality
- b. Alopecia areata
- c. Trichotillomania
- d. A and C
- e. None of the above
Correct diagnosis is

- Loose anagen hair loss
- Classic patient is a girl aged 2-5 with blonde hair
  - Can also see in boys, adults, and dark-haired individuals
- Children affected are healthy with normal growth and development
- Often misdiagnosed as alopecia areata or trichotillomania
New Patient

• Hair mount from a pull test shows the following under the microscope.
Your diagnosis is ??

- a. Monelithrix
- b. Trichorrhexsis invaginata
- c. Pediculosis capitis
- d. Trichorrhexsis nodosum
- e. None of the above
SCHEMATIC DRAWINGS AND MICROSCOPIC APPEARANCE OF HAIR SHAFT ABNORMALITIES

A. Trichorrhexis nodosa
B. Trichorrhexis invaginata
C. Monilethrix
D. Trichoschisis due to trichothiodystrophy (polarization)
E. Trichothiodystrophy (polarization; in comparison to normal hair shaft)
F. Bubble hair
G. Pili torti (scanning EM)
H. Pili annulati
I. Trichonodosis
J. Pili trianguli et canaliculi (scanning EM)
Trichorrhexis nodosa

• The most common structural hair shaft abnormality
• The affected hair shafts fracture easily at nodal sites
• The splitting into strands produces a microscopic appearance of a pair of brooms stuck together end to end by their bristles.
Causes of TN

- Most cases are directly related to environmental causes
  - Perming
  - Blow drying
  - Aggressive hair brushing
  - Excessive chemical exposure
  - Scalp pruritis from neurodermatitis, contact dermatitis, and atopic dermatitis
Other Causes of TN

- Underlying disorders such as Argininosuccinic aciduria, Menkes’ kinky hair syndrome, Netherton’s syndrome, Hypothyroidism, or Trichothiodystrophy
Treatment of TN

• **Medical Care**

• Regardless of the presence or the absence of an underlying defect of the hair shaft, trichorrhexis nodosa is ultimately the result of trauma. Therefore, treatment is aimed at minimizing physical or chemical trauma.

• Excessive brushing, hot combing, permanent waving, and other harsh hair treatments should be avoided.

• In acquired localized trichorrhexis nodosa, the underlying pruritic dermatosis should be treated to prevent trauma from scratching or rubbing.

• Underlying metabolic disorders are treated accordingly, usually through the implementation of a specifically tailored diet.
Most Likely Diagnosis Is
Diagnosis Most Likely Is

- a. Frontal fibrosing alopecia
- b. Alopecia areata
- c. Female patterned alopecia
- d. Traction alopecia
- e. None of the above
Traction Alopecia

• Traction alopecia is a common cause of hair loss due to pulling forces exerted on the scalp hair.
• This excessive tension leads to breakage in the outermost hairs.
• This condition is seen in children and adults, but it most commonly affects African American women.
Diagnosis of TA

• Diagnosis is via a thorough history taking and meticulous PE

• Hx of tight braids, “pull-back” hairstyle, or chemical straightening suggests dx of TA
Treatment of Traction Alopecia

• Change method of hair styling
History of balding occurring over many years
Diagnosis Is ???
May be more than one correct answer.

• A. Alopecia areata
• B. Heavy metal poisoning
• C. Common baldness
• D. Chemotherapy induced hair loss
• E. Andronizing syndrome
Male and Female Patterned Alopecia

• Synonyms:
  – Androgenetic alopecia
  – Pattern balding (male and female)
  – Common balding
  – Hereditary balding or thinning
Key Features

- Genetically determined sensitivity of scalp hair follicles to adult levels of androgens
- Miniaturization of hairs in a symmetric “pattern” on the crown, frontal and vertex regions
- Antiandrogen medications can be used for successful treatment
Introduction To MPHL AND FPHL

• Common balding is an androgen-dependent hereditary disorder
• More is known about AGA in men than in women
• Frequency increases with age
  – 80% of men by age of 70 show some signs of MPHL
  – Smaller % of women express the trait
Pathogenesis

• Interplay of genes and hormones
  – Inheritance is almost certainly polygenetic with a genetic input from both parents
    • Strong family history more common in men than women
• The androgen hormones testosterone and dihydrotestosterone (DHT) have selective roles at puberty
• Expression of AGA is particularly related to DHT
5α-REDUCTASE ISOZYME ACTIVITY IN ADULT HUMAN TISSUES

Type I 5α-reductase
- Scalp hair follicles
- Sebaceous glands

Type II 5α-reductase
- Scalp
- Beard
- Hair follicles
- Chest
- Liver
- Prostate
TREATMENT OF COMMON BALDING
TREATMENT MPHL

• Topical minoxidil solution or foam (2% and 5%)

• Finasteride (1mg/day)
  – Caveats: *5 mg /day associated with high grade prostate cancer in elderly men
    * 1 mg/day will decrease PSA by 40% in the 40-49 year age group and 50% in the 50-60 age group
Normal hair

Hair loss
Treatment of FPHL

• In the US, 2% minoxidil is approved for the management of FPHL (but 5% is oftentimes prescribed).

• FPHL may occur with hyperandrogenemia
  – May benefit from oral contraceptives to suppress ovary androgens, spironolactones, and if appropriate finasteride
MECHANISM OF ACTION FOR ANTIANDROGENS AND FINASTERIDE

Cytoplasm

Finasteride

5\(\alpha\) reductase

DHT

Antiandrogen*

Active AR

T

Active AR

Nucleus

DNA

Gene transcription

* e.g. spironolactone, cyproterone acetate, drospirenone, and flutamide

T = testosterone

DHT = dihydrotestosterone

AR = androgen receptor
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<th>Basic Data of Human Hair Follicles</th>
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<tr>
<td><strong>Total number</strong></td>
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<td><strong>Number of scalp hair follicles</strong></td>
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<td><strong>Average density (scalp) terminal + vellus</strong></td>
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<td><strong>Hair embryology</strong></td>
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<td><strong>Hair cycle distribution</strong></td>
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<tr>
<td>(terminal scalp hair)</td>
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<tr>
<td><strong>Duration of hair cycle phases</strong></td>
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This healthy 6-year-old boy has a history of increased hair loss since age 2. He was evaluated following an episode when his brother pulled a large clump of hair from the back of the scalp which quickly regrow. Microscopic examination of his hair revealed a baggy stocking sign at the base of each hair.
CAUSES OF TELOGEN EFFLUVIUM

- Shedding of the newborn (physiologic)
- Postpartum (physiologic)
- Chronic telogen effluvium\(^{29}\) (no attributable cause or illness)
- Postfebrile (extremely high fevers, e.g. malaria)
- Severe infection
- Severe chronic illness (e.g. HIV disease\(^{30}\), systemic lupus erythematosus)
- Severe, prolonged psychological stress
- Postsurgical (implies major surgical procedure)
- Hypothyroidism and other endocrinopathies (e.g. hyperparathyroidism)
- Crash or liquid protein diets; starvation
- Drugs:
  - retinoids (acitretin, isotretinoin)
  - anticoagulants (especially heparin)
  - antithyroid (propylthiouracil, methimazole)
  - anticonvulsants (e.g. phenytoin, valproic acid, carbamazepine)
  - heavy metals
  - \(\beta\)-blockers (e.g. propranolol)
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<th>Number of lifetime cycles</th>
<th>10–20</th>
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<td>Physiologic hair shedding rate (scalp)</td>
<td>~ 100–200/day (substantial interindividual and seasonal variations)</td>
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<td>Hair shaft production rate (scalp)</td>
<td>~ 0.35 mm/day, 1 cm/month</td>
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<td>Hair production is not influenced by cutting/shaving</td>
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<td>Estrogens reduce hair growth rate</td>
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<td>Androgens increase hair growth rate and hair diameter in androgen-dependent sites (e.g. beard)</td>
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<td>Hair shaft diameter and length</td>
<td>Vellus: &lt;0.03 mm; 1–2 mm</td>
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<td>Terminal: &gt; 0.06 mm; 1–50 cm</td>
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<td>Average diameter:</td>
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<td>Asian hair (circular): 120 μm</td>
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<td>Caucasian (elliptic): 50–90 μm</td>
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<td>Hair shaft structures</td>
<td>Cuticle (outside), cortex, medulla (center)</td>
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<td>Cuticle maintains hair fiber integrity</td>
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<td>Cortex contains bulk of hair keratins and keratin-associated proteins</td>
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<td>Hair fiber strength is largely due to disulfide bonding</td>
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<td>Medulla consists of loosely connected trichocytes with large intercellular air spaces; provides insulation (animals)</td>
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<td>Hair shaft pigmentation</td>
<td>Dark hair: predominance of eumelanin</td>
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<td>Blond/red hair: pheomelanin predominates</td>
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<td>Melanosomes of hair cortex larger than those of epidemis</td>
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<td>Hair graying (canities)</td>
<td>Generally commences in the third to fourth decade of life on the temples, spreading later to crown and occiput; by the age of 50 years, 50% of the population has at least 50% grey hair</td>
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<td>Hair patterns</td>
<td>Pubic hair: horizontal (90% of women, 20% of men), acuminate (10% of women, 50% of men)</td>
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<td>Diffuse chest hair: normally grows only in men, after puberty (until 6th decade)</td>
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<td>Axillary: appears about 2 years after first pubic hairs, more sparse in Mongoloids than Caucasians; frequently absent in older individuals</td>
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<td>Trichoglyphics: single, clockwise parietal whirl present in 95% of individuals</td>
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