Common Dermatologic Disorders:
Tips for Diagnosis and Management

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DISCLOSURE OF CONFLICTS OF INTEREST

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I have no relevant conflicts of interest to disclose.
Outline

- Approach to the itchy patient
- How to really treat eczema
- Acne in the adult
- Rosacea
- Perioral dermatitis
- Hair 101
- Skin cancer (melanoma)
- Sunscreens

Approach to the itchy patient
Question 1

- 57 F with 3 months of itch
- started on lower extremities
- No response to antifungal creams and OTC hydrocortisone cream
- Showers 2 x/day with hot water, uses an antibacterial soap, and does not moisturize

Question 1: The Best Diagnosis Is

1. Asteatotic dermatitis
2. Pruritus of renal failure
3. Nummular dermatitis
4. Tinea corporis
5. Neuropathic pruritus
Question 2
68M with ESRD complains of generalized itch

Question 2: The Best Diagnosis Is

1. Asteatotic dermatitis
2. Pruritus of renal failure
3. Nummular dermatitis
4. Tinea corporis
5. Neuropathic pruritus
Pruritus = the sensation of itch

- Itch can be divided into four categories:
  1. Pruritoceptive
     - Generated within the skin
     - Itchy rashes: scabies, eczema, bullous pemphigoid
  2. Neurogenic
     - Due to a systemic disease or circulating pruritogens
     - Itch “without a rash”
  3. Neuropathic
     - Due to anatomical lesion in the peripheral or central nervous system
     - Notalgia paresthetica, brachioradial pruritus
  4. Psychogenic itch

Pruritus- History

- Suggest cutaneous cause of itch:
  - Acute onset (days)
  - Related exposure or recent travel
  - Household members affected
  - Localized itch
- Itch is almost always worse at night
  - does not help identify cause of pruritus
- Aquagenic pruritus suggests polycythemia vera
- Dry skin itches
Pruritus- Physical Exam

Are there primary lesions present?

- yes
  - Pruritoceptive
- no
  - Neurogenic, Neuropathic, or Psychogenic

Question 1

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Nummular dermatitis
Case 2
68M with ESRD complains of generalized itch

Linear Erosions in “Butterfly” Distribution
Pruritus “Without Rash”

Causes of Neurogenic Pruritus
(Pruritus Without Rash)

• 40% will have an underlying cause:
  • Dry Skin
  • Liver diseases, especially cholestatic
  • Renal Failure
  • Iron Deficiency
  • Thyroid Disease
  • Low or High Calcium
  • HIV
  • Medications
  • Cancer, especially lymphoma (Hodgkin’s)
Linear erosions due to pruritus in patient with cholestatic liver disease

Workup of “Pruritus Without Rash”

- CBC with differential
- Serum iron level, ferritin, total iron binding capacity
- Thyroid stimulating hormone and free T4
- Renal function (blood urea nitrogen and creatinine)
- Calcium
- Liver function tests
  - total and direct bilirubin, AST, ALT, alkaline phosphatase, GGT, fasting total plasma bile acids
- HIV test
- Chest X-ray
- Age-appropriate malignancy screening, with more advanced testing as indicated by symptoms
Neuropathic Pruritus

• Notalgia paresthetica
• Brachioradial Pruritus
  – Localized and persistent area of pruritus, without associated primary skin lesions, usually on the back or forearms
• Workup= MRI!!
  – Cervical and/or thoracic spine disease in ~100% of patients with brachioradial pruritus and 60% of patients with notalgia paresthetica
• Treatment- capsaicin cream TID, neurontin
  – Surgical intervention when appropriate
Treatment of Pruritus

• Treat the underlying cause if there is one
• Dry skin care
  – Short, lukewarm showers with Dove or soap-free cleanser
  – Moisturize with a cream or ointment BID
    • Cetaphil, eucerin, vanicream, vaseline, aquaphor
• Sarna lotion (menthol/phenol)
• Topical corticosteroids to inflamed areas
  – Face- low potency (desonide ointment)
  – Body- mid to high potency (triamcinolone acetonide 0.1% oint)

Antihistamines for Pruritus

• Work best for histamine-induced pruritus, but may also be effective for other types of pruritus
• First generation H1 antihistamines
  – hydroxyzine 25 mg QHS, titrate up to QID if tolerated
• Second generation H1 antihistamines
  – longer duration of action, less somnolence
  – cetirizine, loratidine, desloratidine, fexofenadine
Systemic Treatments for Pruritus

- **Doxepin** - 10mg QHS, titrate up to 50 mg QHS
  - Tricyclic antidepressant with potent H1 and H2 antihistamine properties
  - Good for pruritus associated with anxiety or depression
  - Anticholinergic side effects
- **Paroxetine (SSRI)** - 25-50 mg QD
- **Mirtazapine** - 15-30 mg QHS
  - H1 antihistamine properties
  - Good for cholestatic pruritus, pruritus of renal failure
- **Gabapentin** - 300 mg QHS, increase as tolerated
  - Best for neuropathic pruritus, pruritus of renal failure

Eczemas
Eczema (=dermatitis)

- Group of disorders characterized by:
  1. Itching
  2. Intraepidermal vesicles (= spongiosis)
     - Macroscopic (you can see)
     - Microscopic (seen histologically on biopsy)
  3. Perturbations in the skin’s water barrier
  4. Response to steroids

Eczemas

- Atopic Dermatitis
- Hand and Foot Eczemas
- Asteatotic Dermatitis (Xerotic Eczema)
- Nummular Dermatitis
- Contact Dermatitis (allergic or irritant)
- Stasis Dermatitis
- Lichen Simplex Chronicus
Asteatotic Dermatitis

Nummular Dermatitis
Eczema
Good Skin Care Regimen

• Soap to armpits, groin, scalp only (no soap on the rash)
• Short cool showers or tub soak for 15-20 minutes
• Apply medications and moisturizer within 3 minutes of bathing or swimming

Eczema
Topical Therapy

• Choose agent by body site, age, type of lesion (weeping or not), surface area
• For Face:
  – Hydrocortisone 2.5% Ointment BID
  – If fails, aclometasone (Aclovate), desonide ointment
• For Body:
  – Triamcinolone acetonide 0.1% Ointment BID
  – If fails, fluocinonide ointment
• For weepy sites:
  – soak 15 min BID with dilute Burrow’s solution (aluminum acetate) (1:20) for 3 days
Eczema
Oral Antipruritics

• Suppress itching with nightly oral sedating antihistamine
• If it is not sedating it doesn’t help
  – i.e. Claritin, Allegra, Zyrtec not useful
• Diphenhydramine, Hydroxyzine 25-50mg, Doxepin 10-25mg

Eczema
Severe Cases

• Refer to dermatologist
• Do not give systemic steroids
• We might use phototherapy, hospitalization, immunotherapy

• Beware of making the diagnosis of atopic dermatitis in an adult- this can be cutaneous T cell lymphoma!
Question 3: The diagnosis is:

1. Acne
2. Rosacea
3. Seborrheic dermatitis
4. Perioral dermatitis
5. Contact dermatitis

Approach to the Adult Acne Patient
Acne Treatment Options- Topical

- Benzoyl peroxide
- Antibiotics - clindamycin, erythromycin, combination benzoyl peroxide and either of above
- Sulfur based preparations
- Azelaic acid
- Retinoids

Acne Treatment Options- Systemic

- Antibiotics
  - Doxycycline 100 mg po BID
  - Minocycline 50-100 mg po BID
  - Tetracycline 500 mg po BID
- Oral contraceptives
- Spironolactone
- Isotretinoin
### Pathogenesis and Clinical Features of Acne

- **Pathogenesis** (treatment targets)
  - Excess sebum
  - Abnormal follicular keratinization
  - Inflammation from *Propionibacterium acnes*

- **Clinical features**
  - Non-inflammatory open and closed comedones ("blackheads and whiteheads")
  - Inflammatory papules and pustules
  - Cystic nodules

### Acne Treatment

- Mild inflammatory acne- benzoyl peroxide + topical antibiotic (clindamycin, erythromycin)
- Moderate inflammatory acne- oral antibiotic (tetracyclines) (with or without topicals)
- Comedonal acne - topical retinoid
- Acne with hyperpigmentation - azelaic acid
- Acne/roacea overlap or if also has seboreheic dermatitis- sulfur based preparations
- Hormonal component- oral contraceptive, spironolactone
- Cystic, scarring- isotretinoin
  - Teratogenic, hypertriglyceridemia, transaminitis, chelitis, xerosis, alopecia (telogen effluvium)
Topical Retinoids

• Side effects
  – Irritating- redness, flaking/dryness
  – May flare acne early in course
  – Photosensitizing
  – Tazarotene is category X in pregnancy !!!

Topical Retinoids- How to Use Them

• Warn patients of side effects
• Start with a low dose: tretinoin 0.025% cream
• Wait 20-30 minutes after washing face to apply
• Use 1-2 pea-sized amount to cover the whole face
• Start BIW or TIW
• Moisturize 30 minutes after applying
• If using another topical acne therapy, use on alternate days
• Sunscreen daily
Acne in Adult Women

• Often related to excess androgen or excess androgen effect on hair follicles
• Other features of PCOD are often not present—irregular menses, etc.
• Serum testosterone can be normal
• Spironolactone 50 mg-100mg daily with or without OCP’s can be very effective, especially in women with lower facial acne

Rosacea
Rosacea

• Chronic inflammatory condition of the central face (nose, cheeks, chin)
• Caucasians with fair skin
• F>M
• Middle age (30-50)
• Many types:
  – Telangiectatic- redness and telangiectasias
  – Papulopustular- no comedones
  – Rhinophymatous

Rosacea- Triggers

• Alcohol
• Sunlight
• Hot beverages (heat)
• Hot, spicy food
• If it makes you flush it can flare rosacea
• Rosacea is NOT related to androgens!!
Rosacea- Treatment

- Medical treatment - papular/pustular rosacea
  - Topical agents
    - Metronidazole
    - Sulfur/sulfacetamide
  - Oral antibiotics (months to years to life)
    - Doxycycline 100 mg BID
    - Cefadroxil 500 mg BID
    - Amoxicillin 500 mg BID
  - SUNSCREEN
- Surgical (laser)
  - Telangiectatic, rhinophymatous

Steroid Rosacea

- Topical steroids may exacerbate or induce an acneiform eruption resembling rosacea
- Treatment
  - stop the topical steroids
  - oral tetracyclines (doxycycline)
- Rosacea may flare severely when the steroids are stopped
Perioroficial Dermatitis

- Women aged 25-35
- Extremely common
- Tiny papules, papulovesicles in same stage of development
  - Grouped and might come together to form plaques
- May sting, itch, burn
- Perioroficial- eyes, mouth, upper lip, peri-nares
  - Narrow spared area around the lips
- May be triggered by topical steroid use to the face
Perioral Dermatitis- Treatment

- Discontinue topical steroids
- Topical
  - Clindamycin
- Systemic (4-6 week course)
  - Doxycycline 100 mg BID
  - Tetracycline
  - Minocycline
  - Erythromycin
  - Azithromycin
- Usually does not recur

Hair 101

Too little hair
Too much hair
Hair Stats 101

• Anagen 90-95%
  – Growing hair, lasts 2-6 years
• Catagen
  – Programmed cessation of hair growth, a few weeks
• Telogen 5-10%
  – Shedding, 3-4 months
• Length of hair is determined by length of anagen phase
• Normal amount of shedding - 50-150 hairs/day

Alopecias

Non-Scarring

• Focal
  – Alopecia areata
  – Trichotillomania
• Diffuse
  – Telogen effluvium
  – Androgenetic alopecia

Scarring

• Neutrophilic predominant
  – Folliculitis decalvans
  – Dissecting cellulitis of the scalp
• Lymphocytic predominant
  – Lichen planopilaris
  – Pseudopelade
Telogen Effluvium

• Increased shedding of normal telogen hairs
  – Response to pathologic or normal physiologic change in health status
  – Chronic form with no identifiable cause exists
• Shedding begins 3-4 months after event
• Physical exam
  – Diffuse thinning
  – Hair pull (gentle tug) positive ≥ 2 telogen hairs
• Prognosis- full recovery
• Treatment- remove trigger, minoxidil

Telogen Effluvium- Causes

• Postpartum (physiologic)
• Postfebrile
• Severe infections
• Severe, chronic psychological stress
• Post major surgery
• Endocrinopathy (thyroid)
• Extreme diets
• Medications
Telogen Effluvium- Workup

- CBC
- Fe
- Ferritin
  - Replete if ≤ 40ng/dl
- TSH
- 25,OH Vitamin D
- ANA
- RPR
- May be component of androgenetic alopecia, so consider scalp biopsy if above normal and doesn’t improve

Androgenetic alopecia

- Genetically determined sensitivity of the hair follicles to androgens
- Occurs age puberty to age 60
- Symmetric pattern of miniaturization of hair follicles
  - In women, presents as a widened part or thinning of the vertex with retention of the anterior hairline
- Treatment (women)- antiandrogens (arrest progression)
  - minoxidil 5% foam qd
  - finasteride 1mg (non-childbearing)
  - spironolactone
Androgenetic Alopecia

- Pathogenesis: Conversion of testosterone to dihydrotestosterone (DHT) by the hair follicle enzyme 5 alpha-reductase, type 2
- With each cycle the affected hairs have a shorter anagen phase and a narrower diameter ("miniaturized" hairs)
Androgenetic alopecia

• Systemic workup if:
  – Hirsutism
  – Virilization
  – Severe, scarring cystic acne
  – Irregular menses
  – Infertility

• Check:
  – Free and total testosterone
  – DHEAS
  – Prolactin

Hypertrichosis and Hirsutism

• Hypertrichosis
  – Excess hair all on any part of the body

• Hirsutism
  – Applies to women only
  – Excess growth of terminal hairs in a male pattern (face, chest, areola, linea alba, lumbosacral...)
  – Due to overproduction of or increased end organ sensitivity to androgens
Hirsutism- Etiology

• Increase in androgens- ovary or adrenal gland
• Increased end-organ sensitivity to androgens

• Patterns:
  — Ovarian- lateral face, neck, areola
  — Adrenal- central (pubic triangle to the upper abdomen and presternal area to neck and chin)
**Clinical Guidelines**

**Screening for Skin Cancer: U.S. Preventive Services Task Force Recommendation Statement**

**U.S. Preventive Services Task Force**

**Description:** Update of the 2001 U.S. Preventive Services Task Force (USPSTF) recommendation statement on screening for skin cancer.

**Methods:** To update its recommendation, the USPSTF reviewed evidence published since 2001 on studies on screening effectiveness, the stage of detection by screening, and the accuracy of whole-body examination by primary care clinicians and self-examination by patients.

**Recommendation:** The USPSTF concludes that the current evidence is insufficient to assess the balance of benefits and harms of screening for skin cancer by primary care clinicians or by patient skin self-examination. *(I statement)*
• Applies to adults without history of malignancy or premalignant conditions
• Clinicians should remain alert for skin lesions with malignant features noted in the context of the physical exam performed for other purposes
  – LOOK! for ABCDs, rapidly changing lesions, do a biopsy when indicated

• Know who is at risk:
  – Fair skin patients >65yrs
  – Atypical nevi
  – > 50 nevi
  – Positive family history of skin cancer
  – History of significant sun exposure and sunburns
Malignant Melanoma

• Most frequent cause of death from skin cancer
• Frequently occurs in young adults
  – #1 cause of cancer death in women age 30-35
• Intermittent, intense sun exposure (sunburns)

Melanoma Diagnosis and Prognosis

85% are cured by early diagnosis

• The prognosis is DEPENDENT on the depth of lesion (Breslow’s classification) and lymph node status
• Melanoma of < 1mm in thickness is low risk
• Sentinel lymph node biopsy is recommended for melanoma > 1mm (controversial)
• If melanoma is on the differential, complete excision or full thickness incisional biopsy is indicated
Malignant Melanoma

- Asymmetry
- Border
- Color
- Diameter
- Evolution
Acral Melanoma

- Suspect in African American, Latino, Asian patients
Skin Cancers: What to Refer to Dermatology

• ANY suspicious pigmented lesion
• Any bleeding skin lesion
• Any red spot that doesn’t clear in 6-8 weeks
• Any non-healing erosion or ulceration
• Persons with greater than 50 moles, atypical moles, or family history of melanoma
• Fair-skinned organ transplant recipients with prior sun exposure

Sunscreens 101
Why Sunscreens?

• Prevention of skin cancer
• Prevention of photosensitivity (UVA)
  – Medications
  – Diseases: e.g. lupus erythematosus
• Prevention of skin aging

UV-B and UV-A

**UVB (290-320nm)**
- Burning rays of the sun
- Filtered by the ozone layer
- Most carcinogenic
- Primary target of sunscreens
- SPF refers only to UVB blockade

**UVA (320-400nm)**
- Tanning rays
- Aging rays
  – a complete UVA blocker = anti-aging cream
- Cause of medication related photosensitivity (e.g. HCTZ)
- Harder to block
New Sunscreen Labeling (Summer 2012)

- Broad spectrum = blocks UVA and UVB
- SPF= UVB blockade
- For sunscreen to say can prevent skin cancer AND sunburn, must
  1. be broad spectrum
  2. SPF ≥ 15
- Water resistant for 40 min or 80 min
  - No more “water proof”, “sweat proof”
  - Suggests that always need to re-apply every 2h

Chemical vs Physical Sunscreens

- Chemical sunscreens have UV absorbing chemicals
  - Benzophenone, Parsol 1789, Mexoryl, etc
  - Chemical UVA blockers are photo-unstable (degrade)
    - Stabilizers are now common (e.g. Helioplex)

- Physical sunscreens scatter or block UV rays
  - Zinc and titanium are physical blockers
  - More photostable
  - Block UVA well
  - Inelegant (white film)
How to Apply Sunscreen

• Put it on every morning before leaving the house
  — at least 20 min before sun exposure
• For heavy sun exposure: reapply 20 minutes after exposure begins
• Reapply every 2 hours or after swimming/sweating/towel-drying
• Apply liberally
  — 1oz application= shot glass = covers the body

What to Tell Your Patients

• Use sunscreen, SPF ≥ 30 EVERYDAY
• Avoid mid-day sun/Short Shadow Seek Shade
• Wear protective clothing (hats)
• Put sunscreen on your children
• Ask your doctor to check your skin lesions (most persons with melanoma have been seeing doctors regularly for years)
• Vitamin D Supplement for those at risk for osteoporosis who obey stringent sun-protections practices
  • E.g. organ transplant patients
• The American Academy of Dermatology recommends that an adequate amount of vitamin D should be obtained from a healthy diet that includes foods naturally rich in vitamin D, foods/beverages fortified with vitamin D, and/or vitamin D supplements. Vitamin D should not be obtained from unprotected exposure to ultraviolet (UV) radiation.

• Unprotected UV exposure to the sun or indoor tanning devices is a known risk factor for the development of skin cancer.

• There is no scientifically validated, safe threshold level of UV exposure from the sun or indoor tanning devices that allows for maximal vitamin D synthesis without increasing skin cancer risk.

• To protect against skin cancer, a comprehensive photoprotective regimen, including the regular use and proper use of a broad-spectrum sunscreen, is recommended.

Taken from: American Academy of Dermatology website, 1/25/11