

Skin of Color Case 1, 2, and 3

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Ringworm on the Face?



- 12 year old Indian female
- Presents with expanding circinate rashes
- Wash KOH+ at another facility
- Failed 2 topical AF and griseo
- + Hashimoto's Thyroiditis

Cutaneous Lupus Erythematosus

- Diagnosis: Discoid Lupus
- Concern for early systemic disease associated
- Placed on plaquenil
- Limited improvement
- Lab screening→ JAK2 inhibitor
- ANA + 1: 1640
- Smith >8
- Chromatin 2.8
- RNP >8
- CBC WBC 2.8
- G6PG adequate
- Interface dermatitis c/w DLE
- PAS stain fails to reveal fungus

Psoriasis on the Face?



- Circinate plaque on the left ear
- Applied topical corticosteroids to the lesions
- The lesions spread but are less itchy

Tinea Incognito in Association with Undiagnosed Tinea Capitis

- Culture of the head and ear were + for *T. tonsurans*
- AF shampoo and oral griseofulvin
- Avoid sharing combs, brushes, pillowcases, hoodies
- AF shampoo for the household

Hair Loss Three Months

- This child has had worsening hairloss three months despite topical antifungals, then class 1 corticosteroids



Tinea capitis

- Only hair loss < 25% chance tinea
- Hair loss and scalp 2/3 tinea
- Hair loss, scale, LN neck >80% chance
- This child also has black dots

SOC Round Table Cases

Mercedes E. Gonzalez MD, FAAD

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Pediatric Skin Research

Assistant Professor, University of Miami The Phillip Frost Department of Dermatology & Herbert Wertheim College of
Medicine Florida International University

Friday 9th, 2024 1:15 pm

Disclosures

Speaker & Investigator

Abbvie
Ely Lilly
Krystal Biotech
Pfizer
Regeneron Pharmaceuticals
Sanofi Genzyme
Verrica Pharmaceuticals

Investigator

Amgen, Anterogen, Arcutics
Dermavant
Incyte
Neilsen Biosciences, Novartis

Consultant & Advisory board

Abeona, Alphyn, Amyrt, Arcutis
Cerave, Dermavant
Incyte, Noblepharma
Regeneron, Sanofi, Unilever, Inc
Verrica Pharmaceuticals

Off label uses of medications will be discussed

Case



- 13-year-old girl
- History of atopic dermatitis
- New complaint of persistent hyperpigmented streak on forehead
- Denies injury or preceding flare of eczema
- Concerned about the persistence of the dark spot

Examination

- Atrophy on palpation
- Lesion extended into scalp
- Biopsy: dermal fibrosis, patchy lymphocytic infiltrate and zones in which the eccrine coils appear encased in sclerotic collagen
- Diagnosis: Morphea
- Work-up:
- Brain MRI: Normal
- Baseline bloodwork: CBC, Chemistry



Management

- Oral prednisone ~1mg/kg, tapered over 3.5 months
- Methotrexate 0.3 - 0.5 mg/kg/week for ~2.5 years
- Daily folic acid
- Ongoing lab monitoring
- Visits q 3 months



Follow up:

- No atrophy and minimal hyperpigmentation
- Weaned of methotrexate
- Continued treatment of hyperpigmentation with Specific Beauty® Dark spot corrector pads (Vitamin C, Bearberry extract, Emblica fruit, Licorice root extract) & Sun protection daily
- 4 months s/p Methotrexate



Morphea in SOC

- Diagnosis often delayed
- Lacks early erythema
- DDx: post inflammatory pigmentary change, port wine stain
- Requires prompt systemic treatment to prevent permanent atrophy
- Prolonged post-inflammatory hyperpigmentation can be distressing
 - Requires treatment



Hyperpigmentation and QOL

Table II. Factors associated with Dermatology Life Quality Index scores among patients with a disorder of hyperpigmentation/hyperchromia (N = 419)

Dependent variables	n, (%) ^a	Unadjusted mean DLQI score (SD)	Adjusted coefficient [†]	P value	Confidence interval	
Clinical diagnosis						
Melasma (reference)	161 (38.98)	7.05 (4.93)	—	—	—	—
PIH	138 (33.41)	8.57 (6.14)	0.56	.001	0.23	0.87
Other	114 (27.60)	4.42 (4.521)	−0.40	.017	−0.73	−0.07
Race						
White (reference)	58 (14.18)	5.01 (4.57)	—	—	—	—
African American	118 (28.85)	6.89 (6.46)	0.10	.598	−0.28	0.48
Asian	205 (50.12)	6.15 (5.28)	0.27	.318	−0.25	0.79
Hispanic	28 (6.85)	7.42 (5.03)	0.37	.050	0.0005	0.73
Education						

PIH: DLQI score showed severe to very severe effect in 23.8% and mild to moderate effect in 57.1% on QoL

No data on impact of dyspigmentation on children & adolescents

Hyperpigmentation: treatment

Table. Commonly Used Hydroquinone Alternatives

Active ingredient	Mechanism of action	Special considerations and/or common uses
Arbutin	Inhibits tyrosinase	Can be used in sensitive skin (acne, rosacea), α -arbutin is more stable and stronger than β -arbutin, risk of contact dermatitis
Ascorbic acid	Antioxidant/inhibits tyrosinase	Limited stability, irritant dermatitis, antioxidant
Azelaic acid	Inhibits tyrosinase	Similar efficacy to 2% hydroquinone, 15% or 20% formulations, safe in pregnancy, preferred in acne/acne-induced PIH
Cysteamine	Inhibits melanogenesis at high concentrations by multiple mechanisms	5% Concentration, unpleasant sulfur odor, not widely available in US
Kojic acid	Inhibits tyrosinase	Risk of irritant dermatitis in higher percentages
Niacinamide	Inhibits melanosome transfer	Used in combination therapy, preferred in acne with PIH, used in cosmeceuticals
Tranexamic acid	Multimodal including inhibition of tyrosinase activity and reduction of levels of melanocyte-stimulating hormone	Topical, microinjection, and oral forms available, most effective in melasma, can be used in acne-induced PIH, less risk of halo hypopigmentation
Tretinoin	Melanocyte differentiation and increased turnover	Used in combination therapy, risk of irritant dermatitis

Abbreviation: PIH, postinflammatory hyperpigmentation.

TABLE 1 Mechanism of action of cosmeceuticals

Cosmeceutical	Mechanism of action	References
Thiamidol	Human tyrosinase competitive inhibitor	4-6
Kojic Acid	Inhibits tyrosinase genesis with antioxidant effects	7-11
Vitamin C	Tyrosinase inhibition through interaction with copper ions at tyrosinase active sites, decreasing melanogenesis Antioxidant effects	12-18
Arbutin	Reduces tyrosinase activity and inhibits melanocyte maturation	19-22
Retinol	Inhibition of matrix metalloproteinase activation inhibition of oxidative stress decreased melanosome transfer. Through regeneration of the extracellular matrix and regulation of keratinocyte differentiation, accelerates exfoliation and reduces epidermal melanin	23-28
Nicotinamide	Antioxidant through polyadenosine diphosphate-ribose polymerase inhibition. Melanosome transfer inhibition	29-30
Ferulic acid	Antioxidant properties	31-34
Resorcinol	Tyrosinase inhibition	36-42
Licorice root extract	Glabridin inhibits tyrosinase. Liquiritin, reduces UV-induced erythema, is anti-inflammatory and has inhibitory effect on melanosomes	43-47
Soybean	Contains isoflavanes and serine proteases which have anti-senescent, antioxidant, pigment attenuating, photoprotecting and melanosome transfer inhibiting properties	48-51

Limited data on treatments for hyperpigmentation in children/adolescents

Additional cases of inflammatory skin disease with subdued erythema in SOC



Psoriasis
Biopsy +



Psoriasis

Progressive Macular hypomelanosis



- Acquired hypopigmentation
- More common: darker skin types and tropical environments
- Wood's lamp punctiform orange-red fluorescence
- Association with *Cutibacterium acnes* phylogenetic type III has been suggested

Progressive Macular hypomelanosis

- A recent review of 108 patients

Treatment done, no (%)	
No treatment	40 (37.0%)
Topical antimicrobials	36 (33.3%)
NBUVB	32 (29.6%)

Recovery, no (%)	
No treatment (spontaneous)	7 (23%)
Topical antimicrobials	10 (38.4%)
NBUVB	29 (90.6%)

SD: Standard deviation. NBUVB: Narrow band ultraviolet B.

- Treatment regimens – benzoyl peroxide + topical antibiotic + natural sunlight (pm sun, 3 times/week)
- Oral minocycline
- nbUVB is often not practical
- Repigmentation is slow

Mitchell KN, et al. *Pediatric Dermatology*. 2021;38(Suppl. 2):20–29.
Kim MB, et al. *J Am Acad Dermatol* 2012;66:598-605.

Progressive Macular hypomelanosis



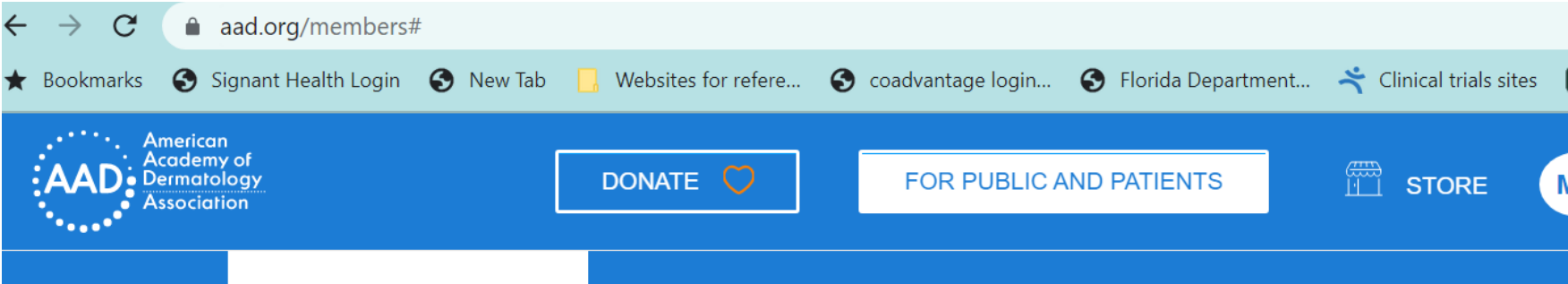
Figure 1: (a) Patient with progressive macular hypomelanosis before treatment. (b) Patient with progressive macular hypomelanosis after 2 months of narrow-band ultraviolet B

The first response was within 4–8 sessions of nbUVB

Time for maximal repigmentation was about 22 sessions

If patients do not respond within 12 sessions, may not respond

AAD Skin of Color Curriculum



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Case SOC

DR PEARL KWONG MD PHD
PEDIATRIC DERMATOLOGIST
JACKSONVILLE FL

Case 1

- 5 yo autistic Egyptian boy with 3 year history of chronic relapsing course of eczema normally affecting only his folds of the extremities
- Comes in with mom dad a couple of other siblings (huge crowd)
- Over last 6 months worsening , more diffuse , more itching, more irritable, visits to pediatrician, ER , and allergist . NB: Why don't they go to a derm for a derm problem?
- Scratching a lot affecting sleep and scratches until he bleeds , bloody sheets
- Meds tried : Tylenol for pain, clindamycin oral , prednisone , fluocinolone oil, Zyrtec, hydrocortisone , topical mupirocin and lotrimin
- Meds that worked transiently: clindamycin oral, prednisone
- Mom had some left over clinda from a few months ago and restarted his clindamycin
- Request: please fix him as we are going overseas to Egypt in one week and we don't want him itching and bleeding in the long plane ride and having an emotional outburst

More history?

- History initially from dad and he knows eczema as he himself suffers from eczema and asthma
- Dad's history is a bit shaky and mom seems to be more on point
- Who is the better historian in the background of the activity of the kids and the noise from the patient and the fact that both parents are talking at the same time
- How can we be efficient and be good clinicians and be sensitive at the same time ?

Case 1



Case 1



Case 1



Case 1



What would you do?

More history

- I asked the more knowledgeable historian (MOM)
- History: get accurate hx of what is being applied to the skin
- Open areas and itching: Triple antibiotics

Learning points

- Get the right history from the right person
- Meds: otc and other topicals and meds ; Med sharing/self treatment
- Not all rashes is due to AD
- Rule out Allergic Contact Dermatitis
- Rule out secondary impetiginization
- ACD treatment : slow wean of prednisone
- Follow up follow up follow up