This continuing medical education activity is jointly provided by the North Carolina Dermatology Association and Southern Regional Area Health Education Center.

FRIDAY PRESENTATIONS
Medical and Diagnostic Pearls
Mark Lebwohl, MD
Sol and Clara Kest Professor
And Chairman
Department of Dermatology
The Mount Sinai School of Medicine
Mount Sinai gets dollars from:

- Abbvie
- Amgen
- Boehringer Ingelheim
- Celgene
- Eli Lilly
- Janssen / Johnson & Johnson
- Kadmon
- Medimmune/Astra Zeneca
- Novartis
- Pfizer
- ViDac.

Consultant

- Allergan
- Dr. Reddy
• Pruritus in the elderly
• Lichen planus
• Ostomies
• Raynaud’s phenomenon
• Optimal phraseology for patients
• Actinic Keratoses
• Local anesthesia alternatives
• Tool tips
• Management of bleeding
• Patient with hyperverbia profundia
• Ocular rosacea
• Gingival hyperplasia
• Drug sampling
• Lyme disease
• Defibrillators
• Nickel allergy
• Atopic dermatitis
Chronic eczematous eruptions of the elderly are associated with chronic exposure to calcium channel blockers: results from a case-control study.

[Lisinopril-induced erythroderma]
Schmutz JL, Barbaud A, Tréchot P. 
Epub 2009 Apr 3. French
Angiotensin-converting enzyme inhibitors as inducers of adverse cutaneous reactions.
Steckelings UM, Artuc M, Wollschläger T, Wiehstutz S, Henz BM.

Enalapril and vulvovaginal pruritus.
Heckerling PS.
Rash, eosinophilia, and hyperkalaemia associated with enalapril.
Barnes JN et al.
[Captopril-induced eruptions: occurrence over a 3-year period]
Lichen planus

Mark Glsb

**Management**

The management of lichen planus depends on the extent and severity of the condition. It may be managed with topical corticosteroids, topical calcineurin inhibitors, or oral medications such as azathioprine or mycophenolate mofetil. In refractory cases, oral or intralesional corticosteroids may be used. For lesions on the tongue, topical triamcinolone acetonide or 5-fluorouracil may be effective.

**Spectacle 7111S**

Barclay for hepatitis B and C

Liver function tests

Drug history


Six or more panants with Mrx may play a role in the development of lichen planus. In cases of severe disease, systemic corticosteroids may be necessary.

Drug recommendations should be individualized based on the patient's specific needs and response to treatment.
FIRST-LINE THERAPIES

- Topical corticosteroids
- Intralional corticosteroids
- Antihistamines
SECOND-LINE THERAPIES

Metronidazole
Systemic corticosteroids
Isotretinoin, acitretin  Narrowband
or broad band UVB  PUVA
Oral metronidazole treatment of lichen planus.
Büyük AY, Kavala M.

• Metronidazole 500 mg bid x 20-60 d.
• 15/19 (79%) improved
• 13/15 → complete clearing

- sulfasalazine up to 2.5g/d vs. placebo x 6w
- lesion improvement 82.6% vs. 9.6%
- pruritus improvement 91.3% vs. 14.3%
- side effects 30.7% - GI and HA
“Small fistula tracks … from which pus could be obtained on pressure.”

Brunsting LA, Goeckerman WH, O'Leary PA
Arch Dermatol Syph. 1930; 22:655
If you’re confident about a patient’s diagnosis and treatment, let them know you see a lot of this condition and know exactly how to deal with it.

• Mycosis fungoides/CTCL
• Perioral dermatitis

- Both work
- Diclofenac less irritating
Imiquimod 5% cream for the treatment of actinic keratosis: results from two phase III, randomized, double-blind, parallel group, vehicle-controlled trials.


Ingenol mebutate gel for actinic keratosis.

Mean composite LSR scores peaked at day 4 and returned to baseline levels by day 15.

The composite LSR score represents the sum of the scores for the 6 specific types of LSRs graded from 0 to 4, with a maximum score of 24 at each study visit.
Long-term follow-up of photodynamic therapy with a self-adhesive 5-aminolaevulinic acid patch: 12 months data.
Szeimies RM, et al

- PDT – 1 rx: 63% and 79% efficacy at 1 yr
- Placebo PDT: 9% and 25%
- Cryosurgery: 63%
A randomised study of topical 5% imiquimod vs. topical 5-fluorouracil vs. cryosurgery in immunocompetent patients with actinic keratoses: a comparison of clinical and histological outcomes including 1-year follow-up

Krawtchenko N, Roewert-Huber J, Ulrich M, Mann I, Sterry W, Stockfleth E.

*British Journal of Dermatology.*
• Cryo 20-40 sec per lesion x 1-2 sessions
• 5FU bid x 4w.
• Imiquimod tiw x 4 w. x 1-2 courses
Clinical Evaluation:
Comparison of All Treatment Groups

Histological Confirmation: Comparison of All Treatment Groups

Biopsies are checked by 2 independent histopathologists

Sustained Clearance of Initially Cleared Lesions in All Patients

Twelve months after end of treatment

Out of all treated patients (including in the denominator also those not cleared at end of therapy)

Severe refractory fingertip ulcerations in a patient with scleroderma: successful treatment with sildenafil.


- Atorvastatin 40/d vs placebo x 4 mos
- new ulcers: 1.6 vs 2.5
- ↓ RP, ↓ pain and severity of ulcers, ↓ endothelial damage markers

- 100 unit botulinum toxin vial diluted in 2cc preservative-free saline
- 50-100 U of toxin injected into palm around neuromuscular bundles at MCP
• pain relief was immediate
• ulcers healed within 2 months
• Doppler showed increased blood flow within 30 minutes
• pain relief persisted in 12/19 at 13-59 months
Management of vasospastic disorders with botulinum toxin A.
Van Beek AL et al.

- 11 patients, painful Raynaud’s, digital ulcerations.
- Failed vasodilators, anti-platelet agents, and IV prostacyclin.
Botox 100 U at 8-10 sites, perivascular digital and palmar.

- Temporary hand weakness in 3 patients.
- All patients improved:
  - Less frequent and less severe vasospasm and cyanosis within 48 hours.

  PreRx scores: 9-10

  PostRx: 0-2

Van Beek AL et al.  
Increased sensitivity to thermal pain and reduced subcutaneous lidocaine efficacy in redheads.
Liem EB et al.

Anesthetic requirement is increased in redheads.
Liem EB et al.
Anesthesiology. 2004;101:279-83.
Alternative Local Anesthetics

• Diphenhydramine
  – 50mg/mL (5%) Dilute 1:5 (1%)  
  – Lasts ~20 minutes  
  – Risk of necrosis and delayed sedation

• Bacteriostatic saline w/0.9% benzyl alcohol
  – Sufficient volume and pressure  
  – Lasts ~2 minutes
Injectable sodium chloride as a local anesthetic for skin surgery.
Weiner SG
Cutis. 1979; :342-3.

“parallel scalpel technique, razor technique, or curettage...punch biopsies and electrocautery techniques”
Diphenhydramine versus lidocaine as a local anesthetic.
Dire DJ, Hogan DE.

- No significant differences between 1% lidocaine and 1% diphenhydramine injections for local anesthesia.
Lidocaine versus diphenhydramine for anesthesia in the repair of minor lacerations.
Ernst AA, et al.

• 1% diphenhydramine more painful than 1% lidocaine, but anesthesia is equivalent
Reasons to Become a Registry Investigator

• Contribute to education/clinical knowledge of the psoriasis community
• Opportunity to establish a database of your patient population
• Academic recognition and publication opportunities
• Supplement existing insurance fee schedules
  – Site compensation is $400 (including $20 for patient) per Enrollment visit and $300 (including $20 for patient) per biannual Follow Up visit
If you are interested in participating in the Psoriasis Registry as a research investigator, please email psoriasis@corrona.org or visit www.corrona.org or call 508.408.5432
Breakthrough Drugs in Dermatology

Mark Lebwohl, MD
Sol and Clara Kest Professor
And Chairman
Kimberly and Eric J. Waldman
Department of Dermatology
Icahn School of Medicine at Mount Sinai
LIFE CHANGING MEDICATIONS

• New psoriasis therapies
  • Dupilumab
  • Omalizumab
  • Vismodegib/Sonidegib
• Penetration enhancers that improve topical therapy
  • JAK inhibitors
• New vitiligo therapies
Drugs for Psoriasis and Psoriatic Arthritis

- ETANERCEPT
- ADAJIMUMAB
- INFLIXIMAB
- CERTOLIZUMAB
- GOLIMUMAB
- USTEKINUMAB
- SECUKINUMAB
- IXEKIZUMAB
- APREMILAST
- METHOTREXATE
- CYCLOSPORINE
- ACITRETIN

- BIODALUMAB
- GUSELKUMAB
- TILDRAKIZUMAB
- RISANKIZUMAB
- LY3074828
Drugs for Psoriasis and Psoriatic Arthritis-ORAL

• ETANERCEPT
• ADALIMUMAB
• INFliximab
• CERTOLIZUMAB
• GOLUMUMAB
• USTEKINUMAB
• SECUKINUMAB
• IXEKIZUMAB

• APREMILAST
• METHOTREXATE
• CYCLOSPORINE
• ACITRETIN

• BRODALUMAB
• GUSELKUMAB
• TILDRAKIZUMAB
• RISANKIZUMAB
• LY3074828
• ACITRETIN
Drugs for Psoriasis and Psoriatic Arthritis - FEW INJECTIONS

- ETANERCEPT
- ADALIMUMAB
- INFLIXIMAB
- CERTOLIZUMAB
- GOLIMUMAB
- USTEKINUMAB
- Secukinumab
- Ilekizumab
- Apremilast
- Methotrexate
- Cyclosporine
- Acitretin

- Brodalumab
- Guselkumab
- Tildrakizumab
- Risankizumab
- LY3074828
Drugs for Psoriasis and Psoriatic Arthritis – LONG Hx & ↓ CARDIAC DISEASE

- ETANERCEPT
- ADALIMUMAB
- INFILIXIMAB
- CERTOLIZUMAB
- GOLIMUMAB
  - USTEKINUMAB
  - SECUKINUMAB
  - IXEKIZUMAB
  - APREMILAST
  - METHOTREXATE
  - CYCLOSPORINE
  - ACITRETIN
- BRODALUMAB
- GUSELKUMAB
- TILDRAKIZUMAB
- RISANKIZUMAB
- LY3074828
Drugs for Psoriasis and Psoriatic Arthritis – OBESITY: ADJUST FOR WEIGHT

- ETANERCEPT
- ADAлимУMAB

**INFLIXIMAB**
- CERTOLIZУMAB
- GОLIMУMAB

**USTEKINUMAB**
- SECUKINУMAB
- IXEKIZУMAB
- APREMILАST
- METHОTREXАTE
- CYCLOSPORINE
- ACITERETIN

- BRODALUMAB
- GУSELКUMAB
- TILDRAKIZУMAB
- RИSANKIZУMAB
- LY3074828
Drugs for Psoriasis and Psoriatic Arthritis – OBESITY: ADJUST FOR WEIGHT

- ETANERCEPT
- ADAHIMAB
- INFLIXIMAB
- CERTOLIZUMAB
- GOLIMUMAB
- USTEKINUMAB
- SECUKINUMAB
- IXEKIZUMAB
- APREMILAST
- METHOTREXATE
- CYCLOSPORINE

- BRODALUMAB
- GUSELKUMAB
- TILDRAKIZUMAB
- RISANKIZUMAB
- LY3074828
Drugs for Psoriasis and Psoriatic Arthritis – OBESITY

- ETANERCEPT
- ADAлимумаб
- ИНФАКИЗУМАБ
- CERTОЛИЗУМАБ
- GОЛИМУМАБ
- УСТЕКИНУМАБ
- SECУКИЗУМАБ
- ИКЕКИЗУМАБ
- АПРЕМИЛАСТ
- METHОТРЕКСАТ
- CYCLOSPORINE
- BROДАЛУМАБ
- GUSELKУМАБ
- TILДРАКИЗУМАБ
- RИСАНКИЗУМАБ
- LY3074828
Drugs for Psoriasis and Psoriatic Arthritis-PSA

- ETANERCEPT
- ADALIMUMAB
- INFliximab
- CERTOLIZUMAB
- GOLUMUMAB
- USTEKINUMAB
- SECUKINUMAB
- IXEKIZUMAB
- APREMILAST
- METHOTREXATE
- CYCLOSPORINE
- ACITRETIN

- BRODALUMAB
- GUSELKUMAB
- TILDRAKIZUMAB
- RISANKIZUMAB
- LY3074828
Drugs for Psoriasis and Psoriatic Arthritis (PSA)

- ETANERCEPT
- ADALIMUMAB
- INFLIXIMAB
- CERTOLIZUMAB
- GOLIMUMAB
- USTEKINUMAB
- SECUKINUMAB
- IXEKIZUMAB
- APREMILAST
- METHOTREXATE
- CYCLOSPORINE
- ACITRETIN

- BRODALUMAB
  - GUSELKUMAB
  - TILDRAKIZUMAB
  - RISANKIZUMAB
  - LY3074828
Drugs for Psoriasis and Psoriatic Arthritis - FAST

- ETANERCEPT
- ADA LIMUMAB
- INF LI XIMAB
- CERTOLIZUMAB
- GOLIMUMAB
- USTEKINUMAB
- SECUKINUMAB
- IXEKIZUMAB
- APREMILAST
- METHOTREXATE
- CYCLOSPORINE
- ACITRETIN

- BRODALUMAB
- GUSELKUMAB
- TILDRAKIZUMAB
- RISANKIZUMAB
- LY3074828
Dupilumab Phase 2b Study: Proportion of Patients Achieving ≥75% Improvement From Baseline in EASI (EASI-75†) Over 16 Weeks

†Observed value with censoring after rescue medication; missing treated as non-responder. EASI: Eczema and Severity Index; q2w: every 2 weeks; q4w: every 4 weeks.

Dupilumab SOLO 1 & 2: Proportion (%) of Patients with IGA 0 or 1 and ≥ 2-point Reduction From Baseline at week 16

SOLO 1

SOLO 2

Percent of patients achieving IGA ≤1

* P<0.0001.
IGA: investigator’s global assessment; qw: weekly; q2w: every 2 weeks.
Dupilumab SOLO 1 & 2: Proportion (%) of Patients Achieving EASI-75 at Week 16

SOLO 1
- Placebo: 15%
- Dupilumab 300 mg q2w: 51%
- Dupilumab 300 mg qw: 52.5%

SOLO 2
- Placebo: 12%
- Dupilumab 300 mg q2w: 44%
- Dupilumab 300 mg qw: 48%

* P<0.0001.
EASI: Eczema Area and Severity Index; EASI-75: 75% improvement in EASI; q2w: every 2 weeks; qw: weekly.
Dupilumab SOLO 1 & 2: Percent (%) Change From Baseline to Week 16 in EASI Score

SOLO 1

<table>
<thead>
<tr>
<th></th>
<th>Placebo</th>
<th>Dupilumab 300 mg q2w</th>
<th>Dupilumab 300 mg qw</th>
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</thead>
<tbody>
<tr>
<td>EASI Score</td>
<td>38</td>
<td>72*</td>
<td>72*</td>
</tr>
</tbody>
</table>

SOLO 2

<table>
<thead>
<tr>
<th></th>
<th>Placebo</th>
<th>Dupilumab 300 mg q2w</th>
<th>Dupilumab 300 mg qw</th>
</tr>
</thead>
<tbody>
<tr>
<td>EASI Score</td>
<td>31</td>
<td>67*</td>
<td>69*</td>
</tr>
</tbody>
</table>

* P<0.0001.

EASI: Eczema Area and Severity Index; qw: weekly; q2w: every 2 weeks.
Dupilumab CHRONOS: Proportion (%) of Patients with IGA 0 or 1 and ≥ 2-point Reduction From Baseline at Week 52

Patients achieving IGA ≤ 1 (%)

- Placebo + TCS: 12.5%
- Dupilumab 300 mg q2w + TCS: 36% (p<0.0001)
- Dupilumab 300 mg qw + TCS: 40% (p<0.0001)

IGA: investigator's global assessment; q2w: every 2 weeks; qw: weekly; TCS: topical corticosteroid.

Source: Data on file
Dupilumab CHRONOS: Proportion (%) of Patients Achieving EASI-75 at Week 52

- Placebo + TCS: 22%
- Dupilumab 300 mg q2w + TCS: 65% (*p<0.0001)
- Dupilumab 300 mg qw + TCS: 64%

EASI: eczema area and severity index; q2w: every 2 weeks; qw: weekly; TCS: topical corticosteroid.

Source: Data on file
Omalizumab treatment reduced mean weekly Itch Severity Score by Week 1

- Rapid-onset, dose-response, sustained efficacy at Wk 24 compared with Wk 12

Maurer M, et al. EADV 2013: FC09.1. Sponsored by Genentech, Inc. and Novartis
All doses of omalizumab significantly reduced mean weekly ISS vs placebo (primary endpoint)

Mean change from baseline in weekly ISS at Wk 12

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Change in ISS</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>80</td>
<td>-3.63</td>
<td></td>
</tr>
<tr>
<td>Omalizumab 75 mg</td>
<td>77</td>
<td>-6.46</td>
<td>0.0010</td>
</tr>
<tr>
<td>Omalizumab 150 mg</td>
<td>80</td>
<td>-6.66</td>
<td>0.0012</td>
</tr>
<tr>
<td>Omalizumab 300 mg</td>
<td>81</td>
<td>-9.40</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

p values derived from t-test of least squares means of the differences between each of the omalizumab groups and placebo group using ANCOVA controlling for baseline weekly ISS (<13 vs ≥13) and baseline weight (<80 kg vs ≥80 kg). Baseline observation carried forward imputation was used for missing values.

Maurer M, et al. EADV 2013: FC09.1. Sponsored by Genentech, Inc. and Novartis
Figure. Time to Urticaria Relapse After Omalizumab Treatment

Each patient is represented by a square, with colors indicating the type of urticaria disease. Two colors within a single square indicate comorbidity of 2 urticaria diseases.
Inhibition of the hedgehog pathway in advanced basal-cell carcinoma.
Von Hoff DD et al.

- 33 patients – metastatic or advanced BCC
- GDC – 044a  16 partial and 2 complete responses
- fatigue, hyponatremia, muscle spasm, afib
Randomized, double-blind study of sonidegib (LDE225) in patients with locally advanced or metastatic basal-cell carcinoma

J Clin Oncol 32:5s, 2014 (suppl; abstr 9009a^)
MR Migden, etal

SONIDEGIB
Absorption Azelaic acid

Azelaic acid is most commonly formulated as a 20% cream, as a 15% gel and a 15% foam. There is some published data absorption.

- Percutaneous absorption of azelaic acid into human skin from the 20% cream formulation is 3.6% of the dermally applied dose.\[1\]

- The 15% gel formulation probably delivers higher amounts of azelaic acid to the skin, as studies on mice showed an 8-fold higher delivery (25.3% versus 3.4%) into viable skin for the gel than the cream With both formulations the majority of the applied azelaic acid

Topical corticosteroid compounding: effects on physicochemical stability and skin penetration rate.
Krochmal L, Wang JC, Patel B, Rodgers J. 
**Fig. 2.** Amount of desoximetasone penetrated through $6.36 \times 10^{-1}$ cm$^2$ of human skin at $34^\circ \pm 0.1^\circ$ C. *Bars* indicate standard error of the mean ($n = 4$). ○, 0.25% Desoximetasone + 2% salicylic acid; △, 0.25% desoximetasone + 0.25% camphor, 0.25% menthol, 0.25% phenol; X, 0.25% desoximetasone + 5% LCD; ○, 0.25% desoximetasone; □, 0.25% desoximetasone + 10% urea.
Fig. 1. Amount of hydrocortisone 17-valerate penetrated through $6.36 \times 10^{-1}$ cm$^2$ of human skin at $34^\circ \pm 0.1^\circ$ C. Bars indicate standard error of the mean ($n = 4$). ○, 0.2% Hydrocortisone 17-valerate + 2% salicylic acid; △, 0.2% hydrocortisone 17-valerate + 0.25% camphor, 0.25% menthol, 0.25% phenol; ○, 0.2% hydrocortisone 17-valerate; X, 0.2% hydrocortisone 17-valerate + 5% LCD; □, 0.2% hydrocortisone 17-valerate + 10% urea.
Dependence of corticosteroid penetration on the vehicle.
Polano MK, Ponec M.
Fig 2.—Penetration of triamcinolone acetonide from 60% ethanolic solution with and without addition of 10% salicylic acid.

- X—X: 0.1% Triamcinolone Acetonide + 10% Salicylic Acid
- •••: 0.1% Triamcinolone Acetonide
- O—O: 10% Salicylic Acid

Amount Penetrated

Salicylic Acid, pg

150 100 50 0

Triamcinolone Acetonide, ng

8 16 24 32 40 48 56

Hours
Topical steroid formulation selected to balance skin penetration and retention, while minimizing percutaneous absorption

F-C = oleyl alcohol

- Test formulation F-10 demonstrated optimal penetration – permeation balance: High epidermal and dermal concentrations of betamethasone dipropionate with minimal receptor fluid levels
- Sernivo selected for further clinical development
DFD-01 HAD HIGHEST PENETRATION OF TOTAL BETAMETHASONES INTO THE EPIDERMIS AFTER 24 HOURS VERSUS OTHER COMMERCIAL FORMULATIONS

REDUCTION IN TSS WITH DFD-01 WAS SIGNIFICANTLY GREATER THAN AUGBD AT DAY 4 AND VEHICLE AT ALL TIME POINTS

ITT population. Total sign score is defined as the sum of erythema, scaling, and plaque elevation scores.

- Rapid evaporation of solvents leaves the calcipotriene and BD in a supersaturated state
- Crystals form with other formulations of Cal/BD (ointment, topical suspension) but do not form after dispensing of the foam (both Cal and BD, fully dissolved in the DME and butane solvents, do not form crystals
In vitro skin penetration data for BDP

Penetration of BDP (in CBD ointment and LEO 90100) into skin at different time points

- CBD ointment, 2h
- CBD ointment, 6h
- CBD ointment, 21h
- LEO 90100, 2h
- LEO 90100, 6h
- LEO 90100, 21h
In vitro skin penetration data for calcipotriol

Penetration of calcipotriol (in CBD ointment and LEO 90100) into skin at different time points

- CBD ointment, 2h
- CBD ointment, 6h
- CBD ointment, 21h
- LEO 90100, 2h
- LEO 90100, 6h
- LEO 90100, 21h
Primary Response Criterion

Subjects (%) with Controlled Disease by the IGA at Week 4

Full analysis set (LOCF)

Note: Graph illustrates observed values at each visit
Crisaborole Topical Ointment, 2%: A Nonsteroidal, Topical, Anti-Inflammatory Phosphodiesterase 4 Inhibitor in Clinical Development for the Treatment of Atopic Dermatitis.

Primary Efficacy Endpoint: Percentage of Patients Achieving Success in ISGA (Clear [0] or Almost Clear [1] with ≥2-Grade Improvement From Baseline)

![Graph showing percentage of patients achieving success over study days for Crisaborole (AD-301; n = 503) and Vehicle (AD-301; n = 256) compared to Crisaborole (AD-302; n = 513) and Vehicle (AD-302; n = 250). Primary endpoints at Day 29: 301, p=0.038; 302, p<0.001]
Comparison of **tofacitinib** vs ETN or PBO in moderate to severe chronic plaque psoriasis: Phase 3 RCT


Preliminary clinical activity of a topical JAK1/2 inhibitor in the treatment of psoriasis.

Punwani N, et al.

The efficacy of afamelanotide and narrowband UV-B phototherapy for repigmentation of vitiligo.

Grimes PE, Hamzavi I, Lebwohl M, Ortonne JP, Lim HW.

AAD Practice Management Center
Office of Access to Care and Treatment
Rachna Chaudhari
www.aad.org/piorauth
Prior Authorization Assistance Center

PRIOR AUTHORIZATION ASSISTANCE

We’ve combined time-saving tools and personalized service to help you navigate medication denials, prior authorizations, and step therapy challenges. Reduce administrative burden and stress on you and your staff, and get patients the medications they need.

Reduce burden and improve access

The Academy has created several resources to reduce administrative burden and help your patients gain access to medications, including:

- Easily create prior-authorization letters to help your patients get the medication approvals they need from insurers.
- Practical tips to help you and your staff navigate prior authorization issues.
- COMING SOON! A help hotline for members. One-on-one help provided by the Academy’s expert staff — complimentary for a limited time!

Prior authorization requires providers to obtain advance approval before performing a service to qualify for payment coverage. Prior authorization for medications usually involve brand-name products for which there is no generic equivalent, or a drug that a patient has taken for years but for which the insurance carrier now requires annual re-authorization.

Most physicians consider prior authorization to be an expensive and time-consuming process that questions their clinical judgment and siphons resources away from patient care. Even more concerning are the treatment delays and negative patient health outcomes that can be caused by prior authorization.

Common prior authorization drugs

In early 2016 the AAD sent a survey to a total of 208 AAD members and 300 Association of Dermatology Administrators & Managers (ADAM) members. Survey recipients were requested to forward the survey to those responsible for completing prior authorizations in their practices. A total of 72 AAD members and another 106 members of ADAM...
Prior Authorization

Drug Denial Letter Template

Complete the following steps to create an individualized letter appealing a denial for a prescribed treatment for your patient.
Prior Authorization Letter Tool

TREATMENT INFORMATION

Select the disease for which your patient is being treated and the prescribed drug below.

Dermatologic disease with ICD-10 diagnosis code
Psoriasis Vulgaris (L40.0)

Name of drug
Enbrel

Alternative Treatment Drug Options
For step therapy protocols, select drug/treatment the insurance company is requiring you to prescribe your patient as an alternative treatment (skip if not applicable)
- Acitretin
- Cosentyx
- Cyclosporine
- Humira
- Methotrexate
- Phototherapy
- Remicade
- Siliq
- Stelara
- Taliz
Prior Authorization Letter Tool

**PATIENT INFORMATION**

Patient name

Patient health insurance identification number

Patient date of birth

Date of prior authorization

I have previously prescribed this patient the following therapies

Name of medication

Start date

End date

List reason for stopping medication

ADD ITEM

PREVIOUS

NEXT
Prior Authorization Letter Tool

TEMPLATE COMPLETE

Click the button below to download your prior-authorization letter template.

DOWNLOAD DOCUMENT

START OVER  PREVIOUS  NEXT
2/16/2017

Mark G. Lelwuchi, MD, FAAD
5 E 88th St
New York, NY 10028

Dr. Jon Doe
National Medical Director
BCBS
1234 Anywhere Lane
Chicago, IL 60607

Re: Dental prior authorization for Maggie Simpson 1234 02/12/2016

Dear Dr. Jon Doe,

I am contacting you as a board-certified dermatologist caring for Maggie Simpson with regard to the patient’s diagnosis of Psoriasis Vulgaris (L40.0).

I recently prescribed this patient Enbrel, which required a prior authorization that was filed on 02/06/2017. The prior authorization was denied and the patient was unable to fill their prescription. I have reviewed the patient’s diagnosis, case plan and clinical guidelines for treatment and request a formal appeal of your denial for Enbrel.

When treating a patient with Psoriasis Vulgaris (L40.0), it is necessary to have access to the full spectrum of accepted treatments as patients may not be able to use one particular treatment due to lack of response, the potential for side effects or even an allergic reaction. It can become a serious safety issue for the patient if I am not able to prescribe a wide variety of treatments for this condition.

I have previously prescribed this patient the following therapies:

- Humira from 02/03/2017 to 02/01/2017. The patient had an adverse reaction to this medication, which included adverse reaction.

I strongly believe Maggie Simpson needs access to Enbrel. Enbrel is not only an approved and effective treatment for psoriasis and psoriatic arthritis, but it has been shown to reduce inflammation and slow the progression of psoriasis.

My patient is not a good candidate for your suggested alternatives for the following reasons:

- Humira has been associated with an increase in anti-nuclear antibodies and can cause drug-induced lupus.

- Remicade is only available by intravenous infusion and is not a practical treatment for this patient because it requires visits to an infusion center that is not easily accessible to the patient. Remicade has been associated with an increase in malignancies, particularly skin cancers, and is therefore not an ideal therapy in patients with a history of malignancy, especially skin cancers. It has also been associated with respiratory tract cancers in smokers. Remicade has also been associated with an increase in anti-nuclear antibodies and can cause drug-induced lupus.

Additionally, I request that you review the following evidence showing how this medication can be effectively utilized to treat Psoriasis Vulgaris (L40.0):


On behalf of Maggie Simpson, I would appreciate your prompt reconsideration of this denial. Please feel free to contact me at 1-847-245-1854 for any additional information you may require. I look forward to receiving your response and approval of coverage for this medication.

Sincerely,

Mark G. Lelwuchi, MD, FAAD
Pediatric Dermatology Pearls

Craig Burkhart, MD, MS
Pediatric Dermatology
The University of North Carolina at Chapel Hill
Menkes Disease Mimicking Child Abuse

Abstract: Although Menkes disease has well-recognized neurologic, developmental, and cutaneous features, the initial presentation may resemble child abuse. We describe a 5-month-old boy with multiple fractures indicative of nonaccidental trauma who was ultimately diagnosed with Menkes disease. Copper deficiency leads to connective tissue abnormalities and may result in subdural hematomas, wormian bones, cervical spine defects, rib fractures, and spurring of the long bone metaphyses. Several of these findings, including fractures and subdural hematomas, may be misinterpreted as child abuse.
Male infant with coarse white hair and dark tips

Saggy, pudgy cheeks

Saggy, wrinkly skin

Pili torti
Menkes Disease

• X-linked recessive neurodegenerative disorder
• Copper transporter mutation

• Copper depletion occurs after delivery
• Disease manifestations develop after 2-3 months of age
• Hair reflects copper depletion over time
Hair that is distally darker
Saggy skin and floppy baby

http://escholarship.org/uc/item/85x9m8m1
Menkies Pearls

• Why the down face?
• History of copper dilution reflected in hair
An 8-day-old girl presented to the hospital for acute-onset rash of 1-day duration that had started on the face and spread to the trunk and extremities. She was born at 35 weeks and 4 days gestation by spontaneous vaginal delivery with preterm premature rupture of membranes and Apgar scores of 8 and 9. The pregnancy was complicated by heparin-induced thrombocytopenia, anemia, and lupus anticoagulant disorder. Family history was remarkable for systemic lupus in maternal and paternal first-degree relatives. Physical examination revealed normal vital signs and an infant who was feeding and urinating regularly. There were serpiginous and annular erythematous patches on the cheeks and
Annular erythema in an 8 day old

Spongiosis, perivascular inflammation, no interface component: annular erythema of infancy (AEI)
Annular Erythema of Infancy
Annular erythema of infancy

**Scaly**
- Neonatal lupus until proven otherwise
  - ENA
  - Cardiology evaluation

**Not-scaly**
- Consider autoinflammatory disease (NOMID)
  - Articular disease
  - Neurologic disease
  - Fever
  - ESR, CRP, CBC
  - Ophthalmology evaluation
  - Immunology evaluation
Neonatal lupus

Face
Telangiectasias
Atrophy
NOMID/CINCA Syndrome
<table>
<thead>
<tr>
<th>Acronym</th>
<th>Name</th>
<th>SAID group</th>
<th>Gene</th>
<th>Inheritance</th>
<th>Ethnicity</th>
<th>Frequency</th>
<th>Timing of symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>FCAS</td>
<td>Familial Cold Autoinflammatory Syndrome</td>
<td>Cryopyrin Associated Periodic Syndromes (CAPS)</td>
<td>NLRP3</td>
<td>Autosomal dominant. Many large family groups spanning generations. Some patients with spontaneous mutations. [1]</td>
<td>Affects all races, but many are of European descent. [1]</td>
<td>1:1 million, or more. In USA 300+ diagnosed – most cases are from large family groups. [2], [5] Frequency of CAPS in France is 1:360,000. [55]</td>
<td>12-24 hours, or longer. Onset of fever and flares is often 1-3 hours after exposure to cold or cooling temperatures. [1]</td>
</tr>
<tr>
<td>MWS</td>
<td>Muckle-Wells Syndrome</td>
<td>Cryopyrin Associated Periodic Syndromes (CAPS)</td>
<td>NLRP3</td>
<td>Autosomal dominant. Spontaneous mutations, and some family groups with MWS spanning generations. [1]</td>
<td>Affects all races, but many are of European descent. [1]</td>
<td>1:1 million, but it may be more frequent. Some large family groups. [5] Frequency of CAPS in France is 1:360,000. [55]</td>
<td>Often lasts 2-3 days. Random onset-flares of fever and symptoms are often triggered by cold or cooling temperature. [1]</td>
</tr>
</tbody>
</table>

www.AutoInflammatory-Search.org
Neonatal Onset Multisystem Autoinflammatory Disease - aka Chronic Infantile Neurological Cutaneous Articular Syndrome

Acronym:
NOMID/CINCA

SAID group:
Cryopyrin Associated Periodic Syndromes (CAPS)

Gene:
NLRP3

Inheritance:
Autosomal dominant. Most cases are due to spontaneous mutations. Very few familial cases. [1]

Ethnicity:
Any, present in all races. [1]

Frequency:
Estimated frequency 1:1 million, mostly due to spontaneous genetic mutations. [3]

Photo credit:
Autinflammatory Alliance Image collection. Voluntaryy supplied by patients. Image use restricted - contact karen@autoinflammatory.org

Resources:
Download or view our CAPS Guidebook in English, or choose "ES" at the top of the page to access the Spanish version.
### Results: 1 to 20 of 84 (representing 31 labs)

<table>
<thead>
<tr>
<th>Tests names and labs</th>
<th>Conditions</th>
<th>Genes and analytes</th>
<th>Methods</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>NLRP3</strong></td>
<td>2</td>
<td>1</td>
<td>S</td>
</tr>
<tr>
<td>Division Human Genetics Medical University Innsbruck, Austria</td>
<td></td>
<td></td>
<td>Mutation scanning of the entire coding region</td>
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<tr>
<td><strong>NLRP3</strong></td>
<td>3</td>
<td>1</td>
<td>D</td>
</tr>
<tr>
<td>Fulgent Genetics, United States</td>
<td></td>
<td></td>
<td>Deletion/duplication analysis</td>
</tr>
<tr>
<td><strong>NLRP3</strong></td>
<td>2</td>
<td>1</td>
<td>C</td>
</tr>
<tr>
<td>Genome Diagnostics Laboratory University Medical Center, Utrecht, Netherlands</td>
<td></td>
<td></td>
<td>Sequence analysis of the entire coding region</td>
</tr>
<tr>
<td><strong>Cryopyrin-Associated Periodic Syndromes via the NLRP3 Gene</strong></td>
<td>3</td>
<td>1</td>
<td>D</td>
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<tr>
<td>PreventionGenica, United States</td>
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<td></td>
<td>Deletion/duplication analysis</td>
</tr>
<tr>
<td><strong>NLRP3 Gene Sequencing</strong></td>
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<tr>
<td>TDC Clinic Molecular Diagnostics Laboratory, TDC Clinic, Center for Special Needs Children, United States</td>
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<td></td>
<td>Sequence analysis of the entire coding region</td>
</tr>
<tr>
<td><strong>NLRP3, Complete sequencing</strong></td>
<td>1</td>
<td>1</td>
<td>C</td>
</tr>
<tr>
<td>Instituto de Medicina Genomica</td>
<td></td>
<td></td>
<td>Sequence analysis of the entire coding region</td>
</tr>
</tbody>
</table>
Annular Erythema in Neonates Pearls

• Always consider neonatal lupus and autoinflammatory disease

• Refer to cardiology if you have any suspicion for neonatal lupus
  • Even if biopsy and serologies are negative

• www.autoinflammatory-search.org
Congenital Milium of the Nipple

Abstract: A 12-month-old girl presented with an asymptomatic, pearly nodule on the left nipple that had been present from birth and was currently 3 mm in diameter and growing. Assuming the diagnosis of congenital primary milium of the nipple, we took a “wait and see” approach. After 3 months, the pearl disappeared without any scarring.
Pearly white lesion on the left nipple
Nipple Bump Differential

**Children**
- Milia
- Milia-like Syringoma
- Trichoepithelioma
- Fibroma
- Milia-like Idiopathic Calcinosis Cutis
- Neonatal Fibroadnexal Polyp

**Neonate**
- Milia
- Neonatal Fibroadnexal Polyp
Neonatal Nipple Pearl

• Isolated pearly bumps on neonates will self-resolve within a year
A previously well 9-year-old boy presented to our department with a 10-month history of brownish nipple discharge of the left breast. There was no associated pain or history of trauma. The parents related no previous breast manipulation or stimulation. No hormonal treatments or infection were reported. The patient was otherwise healthy and was developing normally. On physical examination, the patient had a small palpable soft mass underneath the left nipple (≤1 cm in diameter), with no signs of infection. During the physical examination, bloodstained discharge of the left nipple was noticed (Fig. 1). The remainder of the
Bloody discharge in a 9-year-old boy

Worrisome signs:
- Unilateral
- Persists longer than 9 months
- Spontaneous discharge

Reassuring signs:
- Centered on the nipple
- Preadolescent
Biopsied after 10 months persistence

Mammary duct ectasia
- brown-colored mass with bloodstained fluid
Work-up of discharge in infants and young children

- Gram-stain and culture of discharge
- Serum prolactin, estradiol, and thyrotropin
- Ultrasound of the affected breast

- If the above is normal and ultrasound consistent with mammary duct ectasia, consider a biopsy if the lesion lasts longer than 9 months
Pediatric Nipple Discharge Pearls

• Work-up of masses that last longer than 9 months
  • Culture
  • Ultrasound
  • Pituitary work-up (prolactin, TSH, estrodiol)

• Biopsy males if really needed
Plaque-Like Myofibroblastic Tumor of Infancy: A New Case Report and Literature Review

Francesco Alesini, M.D.,* Giuseppe Soda, M.D.,* Francesca Gianno, M.D.,* Alessandro Boscarelli, M.D.,† Denis A. Cozzi, M.D.,† and Sandro Bosco, M.D.*

*Department of Molecular Medicine and †Pediatric Surgery Unit, Sapienza University of Rome, Azienda Policlinico Umberto I, Rome, Italy
18 month old with a slow growing plaque

Pathology c/w dermatofibroma
Plaque-like myofibroblastic tumor of infancy

- Appear in infancy and early childhood
- Lower back and hip
- Large plaque
- Histology similar to dermatofibroma
- Positive for factor XIIIa and SMA, negative for S-100 and CD34
Plaque-Like Myofibroblastic Tumor: Report of Three Cases

Ann L. Marqueling, M.D.,* David Dasher, M.D.,† Sheila F. Friedlander, M.D.,† Timothy H. McCalmont, M.D.,* and Ilona J. Frieden, M.D.* †‡

*Department of Dermatology, University of California at San Francisco, San Francisco, California, †Department of Dermatology, University of California at San Diego, San Diego, California, ‡Department of Pediatrics, University of California at San Francisco, San Francisco, California

2 of 3 initially diagnosed as dermatofibromas by a great dermatopathologist
<table>
<thead>
<tr>
<th>Condition</th>
<th>Age at onset</th>
<th>Location</th>
<th>Clinical</th>
<th>Histopathology</th>
<th>Immunohistochemistry</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plaque-like myofibroblastic tumor</td>
<td>Infancy and early childhood</td>
<td>Lower back, hip; less commonly the upper back</td>
<td>Large indurated plaque (2–9 cm reported), may present with ulceration</td>
<td>Nodular proliferation of spindle cells in short fascicles and disorganized in the dermis and superficial subcutis with thickened collagen bundles and periphery and overlying epidermal acanthosis and basilar hyperpigmentation</td>
<td>Positive for factor XIIIa and SMA; negative for S-100 and CD34</td>
</tr>
<tr>
<td>Dermatofibroma</td>
<td>Young adults, rare in first year of life</td>
<td>Lower extremities; less commonly the upper extremities</td>
<td>Less than 1- to 2-cm flesh-colored to slightly hyperpigmented firm papules or nodules</td>
<td>Nodular proliferation of spindle cells in fascicles and haphazard in the dermis with peripheral collagen balls</td>
<td>Positive for factor XIIIa; negative for CD34; variable for SMA</td>
</tr>
<tr>
<td>Dermatomyofibroma</td>
<td>Adolescents and young adults, predominantly female</td>
<td>Neck, arms, upper trunk</td>
<td>1- to 2-cm firm red-brown plaques or nodules</td>
<td>Myofibroblastic fascicles running parallel to epidermis with collagen bundles thinner than surrounding dermis</td>
<td>Positive for vimentin; variable for SMA; negative for desmin, CD34, and S-100</td>
</tr>
<tr>
<td>Infantile myofibroma or myofibromatosis</td>
<td>60% at birth, 80% in first 2 yrs of life</td>
<td>Head, neck, trunk, upper extremities</td>
<td>0.5- to 7-cm skin-colored to vascular-appearing rubbery to hard nodules</td>
<td>Interlacing fascicles of spindled fibroblasts with minimal cytologic atypia in a pale collagenous background and foci of hemangiopericytoma-like vascular pattern; may see focal necrosis and calcification</td>
<td>Positive for vimentin and actin</td>
</tr>
<tr>
<td>Fibrous hamartoma of infancy</td>
<td>First 2 yrs of life, up to 20% at birth</td>
<td>Axilla, shoulders, upper chest wall</td>
<td>Solitary lumpy 2- to 5-cm nodule</td>
<td>Well-defined fascicles of fibroblasts in collagenous stroma, mature adipose tissue and mixed mesenchymal tissue in a basophilic matrix within subcutis and musculoaponeurotic tissues</td>
<td></td>
</tr>
</tbody>
</table>
Plaque-like Myofibroblastic Tumor Pearls

• Pathologist may diagnose as a dermatofibroma if you don’t provide with enough information

• Does not recur after excision

• Reassess diagnosis if it recurs after excision
Contact Burn with Blister Formation in Children Treated with Sennosides

Kimberly Cogley, M.S.N., M.B.A., Andrea Echevarria, M.D., Catalina Correa, M.D., and Luis De la Torre-Mondragón, M.D., F.A.A.P., F.A.P.S.A.

Colorectal Center for Children, Children's Hospital of Pittsburgh, Pittsburgh, Pennsylvania
- 8 Patients
- 6 days to 18 months after initiation of sennosides
- All bowel movements occurred overnight
- Chemical burn noticed during diaper change
Laxative-associated contact dermatitis

- Only in non-toilet-trained children
- Prevent by giving Senna products at a time of day the allows for bowel movements to occur during the day (and not overnight)
  - 6-10 hours after injection
Constipation Action Plan

☐ Green Zone
  - 1-2 poops every day
  - No strain, no pain
  - Poops are soft - like mashed potatoes

To help your child STAY in the Green Zone use:
Miralax: ___ capful(s)
  in ___ ounces of water, juice or Gatorade
  ___ time(s) every day

If child is having diarrhea: REDUCE dose by ½ capful each day until diarrhea stops.

Child should try to poop even if they say they don’t need to. Here’s what they should do:
  • Sit on toilet for 5-10 minutes after meals
  • Feet should touch the floor (may use step stool)
  • Read or look at a book
  • Blow on hand or at a pinwheel. This helps use the muscles needed to poop.

☐ Yellow Zone
  - No poops for 2-5 days
  - Has pain or strains
  - Hard poops

To help your child MOVE OUT of the Yellow Zone use:
Miralax: ___ capful(s)
  in ___ ounces of water, juice or Gatorade
  ___ time(s) for 3 days

After 3 days, if child is still having trouble pooping:
Add Chocolate Ex-Lax, ___ square at night until child has
1-2 poops every day.
Now your child is back in the Green Zone.

☐ Red Zone
  - No poops for 6 days
  - Bad pain
  - Vomiting or bloating

To help your child MOVE OUT of the Red Zone do:
Cleaning Out the Poop on the other side of this paper.

After Cleaning Out the Poop, if your child is still having trouble pooping, call 919-966-6669 to make an appointment with a doctor.
Irritant diaper dermatitis pearls

• Can clean feces with Vaseline soaked gauze instead of wipes

• Quantity of barrier cream (one-half golf ball per diaper change) is more important than type of barrier cream

• Add an antifungal for any diaper dermatitis that lasts longer than 72 hours
Eosinophilic Pustular Folliculitis in Children after Stem Cell Transplantation: An Eruption Distinct from Graft-Versus-Host Disease

Martin Theiler, M.D., *,† Vikash S. Oza, M.D., ‡ Erin F. Mathes, M.D., ¶, #
Christopher C. Dvorak, M.D., ¶ Timothy H. McCalmont, M.D., #, ** Iwei Yeh, M.D, Ph.D., #, **
Robert Sidbury, M.D., M.P.H., †† and Kelly M. Cordoro, M.D. ¶, #

*Department of Pediatric Dermatology, University Children's Hospital Zurich, Zurich, Switzerland, †Department of Dermatology, University Hospital Zurich, Zurich, Switzerland, ‡Ronald O. Perelman Department of Dermatology, School of Medicine, New York University, New York, New York, Departments of ¶Pediatrics #Dermatology, and **Pathology, University of California, San Francisco, California, ††Department of Pediatrics, Division of Dermatology, Seattle Children's Hospital and School of Medicine, University of Washington, Seattle, Washington
- 7 month old with this eruption 3 months ago
- Started upon tapering tacrolimus
- Biopsy of neck revealed EPF
- Cleared with triamcinolone 0.1% ointment
- 8 yo boy with pruritic macules and papules 2 months after transplantation
- Biopsy c/w EPF
- Cleared with 5 months of betamethasone dipropionate ointment
Eosinophilic pustular folliculitis

• Recurrent crops/clusters of erythematous papules and pustules with an eosinophilic infiltrate on the biopsy

• Four types
  • Classic EPF
  • Immunosuppression/HIV-associated EPF
  • Infantile EPF
  • HSCT EPF
EPF-HSCT

• Pruritic, follicular, erythematous papules and pustules
• Head, upper extremities, and trunk
• 2-3 months after HSCT
• Resolves in several months
Infantile EPF

Hyper-IgE Syndrome?
Eosinophilic pustular folliculitis of infancy: A series of 15 cases and review of the literature

Ángela Hernández-Martín, MD, Almudena Nuño-González, MD, Isabel Colmenero, MD, and Antonio Torrelo, MD
Madrid, Spain

Background: Eosinophilic pustular folliculitis (EPF) of infancy is characterized by the presence of pustular lesions containing eosinophils. It is the least well-characterized of the EPF diseases.

Objectives: We sought to define the clinical and histopathologic features of the condition.

Methods: We conducted a retrospective review of the clinical data and histologic findings of 15 patients given the diagnosis of EPF of infancy at the Hospital Niño Jesús, Madrid, Spain, from 1995 to 2011, and patient data published in MEDLINE with such a diagnosis from the disease description (1984-2011).

Results: A total of 61 cases were collected. The disease was more common in males than females (ratio 4:1 and presented before 14 months of life in 95% of cases (mean 6.1 months; median 5 months). All patients had recurrent outbreaks and scalp involvement, and 65% had lesions on areas of the body other than the scalp. Tissue eosinophilia was present in all cases; however, true follicular involvement was observed only in 62% of cases in which histologic study was available. More than 80% of the patients were cured by 3 years of age (mean 25.3 months; median 18 months). Topical steroids were effective in 90% of cases.

Limitations: This was a retrospective study.

Conclusions: EPF of infancy presents most often in the first 14 months of life and usually resolves by 3 years of age. All patients showed scalp involvement, tissue eosinophilia, and recurrent outbreaks. The condition does not require aggressive treatment, as it is benign and self-limiting. (J Am Acad Dermatol 2013;68:150-5.)
Cutaneous manifestations of hyper-IgE syndrome in infants and children

Sarah L. Chamlin, MD, Timothy H. McCalmon, MD, Bari B. Cunningham, MD, Nancy B. Esterly, MD, Chan-Ho Lai, MD, Susan Bayliss Mallory, MD, Anthony J. Mancini, MD, Joan Tamburro, DO, and Ilona J. Frieden, MD

We describe 8 children with hyper-IgE syndrome who had papulopustular eruption on the face and scalp in the first year of life. Seven of the 8 patients had persistent peripheral eosinophilia and 3 had leukocytosis noted before diagnosis. Skin biopsy specimens in 6 patients revealed spongiosis and perivas- sentation in infancy or childhood as a papulopustular or vesicular eruption.7,8 We describe a distinctive papulopustular eruption in 8 patients with hyper-IgE syndrome as the initial manifestation of disease, with an eczematous

**Burkhart Pearl:** Bad red rashes in the first month of life are either:
1. Ichthyosis
2. Immunodeficiency
3. Infection (candida or syphilis)

Consider an immunology workup for eczematous rashes that start in the 1st month of life. Don’t worry about infantile EPF.
Pediatric Eosinophilic Pustular Folliculitis Pearls

• Treat symptomatically with steroids

• HSCT EPF resolves within months
• Infantile EPF resolves within years

• Base hyper-IgE workup on timing of eruption and health of infant rather than EPF
DERMOSCOPY FOR THE NON-DERMOSCOPIST

Kelly Nelson, MD FAAD
Associate Professor
Department of Dermatology
The University of Texas
MD Anderson Cancer Center
Start Slow

Phase 1: Dermatoscope as a magnifying glass
- Get a dermatoscope
- Keep it in your pocket
- Look at everything before you biopsy it

Phase 2: Test tumor identification accuracy
- Commit on your pathology form
  - BCC vs SCC
  - Melanocytic vs non-melanocytic
- Do the introduction to dermoscopy course next year
Start Slow

Phase 3: Iterative learning

- Attend the advanced dermoscopy course
- Photograph everything
- Review your clinical impression, photographs and images
Data organization

- Organized folders
  - Year/Month/Clinic date
- Diagnosis spreadsheet
- Patient privacy/security
Dermoscopy Overview

- Melanocytic: benign vs malignant
- Non-melanocytic
  - AK, SCC
  - BCC
  - SK
- Special sites
  - Facial
  - Acral
- Unknowns
Practical Use of Dermoscopy

- Ugly Duckling (clinical)
- Ugly Duckling (dermoscopic)
- Individual lesion (dermoscopic)
  - Pattern recognition
3 point check list

• Asymmetry
• Atypical Network
• Blue-white structures
### 3 point checklist

<table>
<thead>
<tr>
<th></th>
<th>Average</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sensitivity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall</td>
<td>69.7</td>
<td>61.5 – 76.8</td>
</tr>
<tr>
<td>3-point checklist</td>
<td>96.3</td>
<td>93.3 – 98.0</td>
</tr>
<tr>
<td><strong>Specificity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall</td>
<td>82.8</td>
<td>77.0 – 87.4</td>
</tr>
<tr>
<td>3-point checklist</td>
<td>32.8</td>
<td>20.7 – 47.7</td>
</tr>
</tbody>
</table>

**Sensitivity**: true positive rate; if the test is highly sensitive and it is negative, you can be nearly certain that they *don’t* have the condition; Sn(out)

**Specificity**: true negative rate; if the test is highly specific and it is positive, you can be nearly certain that they *do* have the condition; Sp(in)
### 3 point checklist

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</table>

The three point check list helps you **rule out** melanoma

If the three point check list is positive, it doesn’t mean that it certainly is melanoma; your clinical intuition is more reliable.
Pattern Recognition

- Reticular
- Globular
- Homogenous
- Organized
- Disorganized
Special Sites

- Facial
  - Asymmetrical pigmented openings
  - Slate grey structures

- Acral
  - Furrows are fine
  - Ridges are wrong
Ink test: furrows are fine, ridges are wrong
Acral structure

- Stratum corneum
- Epidermis
- Dermis

- Christa intermedia
- Eccrine duct
- Christa limitans
- Christa intermedia
Acral algorithm

Koga H, Saida T. Revised 3 step dermoscopic algorithm for the management of acral lesions. JAMA Derm 2011;147(6):741