ALL THAT RASHES

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DISCLOSURES

- Financial – none
- Educationally - a large amount of gratitude to the nurses and the dermatology and pediatric residents I have the opportunity to work beside
OBJECTIVES

• Review common pediatric skin disorders that can be mistaken for Atopic Dermatitis

• Review clinical findings associated with Atopic Dermatitis that complicate the clinical presentation

• Explore recent literature separating Atopic Dermatitis from Psoriasis
COMMON PEDIATRIC SKIN DISORDERS: MISTAKEN FOR ATOPIC DERMATITIS

- Infectious
- Exanthems
- Other cutaneous inflammatory disorders
- Genetic skin disorders
WHAT IS THE DIAGNOSIS

• Histiocytosis
• Scabies
• Linear IgA
• Dermatitis herpetiformis
Scabies

- Very pruritic
- Typically diffuse lesion distribution, but the depends on length of infestation
- Genitalia commonly involved
- Does not follow typical pattern as in adults
  - may have facial lesions
  - Palms and soles commonly involved
  - May have nodules
- Usually polymorphous
  - Burrows
  - Vesicles
  - Erythematous papules
  - nodules
SCABIES

• If family members have scabies there is a very high likelihood infant will contract it due to the close skin contact (holding)
• Can occur as young as 4 weeks of life
• Treatment
  • Elimite cream – apply to skin surface except face and caution to protect infant from putting hands, feet in mouth after application. Leave on eight hours and reapply in one week
WHAT IS THE DIAGNOSIS

- Bed bug
- Pityrosporum Folliculitis
- Scabies
- Swimmer’s Itch
SWIMMER’S ITCH – CERCARIAL DERMATITIS

- Penetration of human skin by nonhuman schistosomes parasites occurs while swimming in fresh water lakes in mid and south west US
- Trichobilharzia is the organism and the life cycle will travel from bird to snail with the human becoming the accidental victim at the cercarial stage
- Spares covered skin
- Treatment is supportive for symptoms mostly itch and is self resolved within 1 week
WHAT IS THE DIAGNOSIS

- Atopic Dermatitis
- Milia
- Vellus Hair Cysts
- Molluscum Dermatitis
MOLLUSCUM DERMATITIS

• Common cutaneous infection in children
• Asymmetric “eczema” or wrong distribution for eczema
• Very pruritic and then may have autoinoculation
• Will look like localized dermatitis, but will closer evaluation you can see molluscum lesions within the dermatitis
• Commonly seen in patients with atopic dermatitis or patients who have family history of atopy
MOLLUSCUM DERMATITIS

- Treatment may include treating dermatitis before treating molluscum
- Class 4, 5 or 6 topical steroid for one to two weeks
- Treatment for molluscum includes:
  - Curetting, LN2, and many topicals (retinoids, imiquimod)
  - Observation if not too symptomatic
WHAT IS THE DIAGNOSIS

- Impetigo
- Eczema herpeticum
- Dermatitis Herpetiformis
- Stevens - Johnson
ECZEMA HERPETICUM

- Disseminated herpes simplex infection seen in AD patients
- Abrupt onset with fever, malaise and diffuse herpetic lesions focusing at previous AD skin sites
- Complications include keratoconjunctivitis, secondary bacterial infections, fluid loss and viremia
- Greatest concern in neonates and immunodeficient or immunosuppressed patients
- Systemic antivirals and urgent ophthalmologic evaluation with facial lesions
WHAT IS THE DIAGNOSIS

- Linear IgA
- Bullous Impetigo
- Ecthyma
- Bullous Pemphigoid
BULLOUS IMPETIGO

- Most likely cause S Aureus with the associated toxin causing the epidermal split
- Potential complications: sepsis, osteomyelitis, septic arthritis, lymphadenitis and pneumonia
- Topical or systemic antibiotics hopefully chosen from culture sensitivity report
WHAT IS THE DIAGNOSIS

- Steven Johnson
- Kawasaki Disease
- DRESS - Hypersensitivity Disorder
- Staphylococcal Scalded Skin Syndrome
STAPHYLOCOCCAL SCALDED SKIN SYNDROME

- Diffuse blistering disorder caused by the epidermolytic toxin producing S. Aureus
- Diffuse erythema and skin tenderness with subsequent blistering and skin fragility
- Culture sites can be conjunctivae, perioral, perineum and umbilical (look for nidus on infection)
- Occurs in neonates and children at higher rates due to lack of antitoxin antibodies and decreased renal excretion of the toxin
- Treatment is to eradicate the toxin producing S. Aureus and culture sensitivity should be used (MRSA)
- Neutralizing antibodies to desmoglienn 1 are under investigation
WHAT IS THE DIAGNOSIS

- Granuloma Annulare
- Tinea manuum
- Dyshidrotic Eczema
- Psoriasis
TINEA MANUUM

- Less common in children and can be seen with tinea pedis
- T. rubrum, T. mentagrophytes and E. fluccosum are most likely fungi
- Children with diffuse or recalcitrant cutaneous fungal infections should be evaluated for immunodeficiencies
- Topical or systemic antifungal therapy
WHAT IS THE DIAGNOSIS

- Polymorphous Light Eruption
- Impetigo
- Infantile Acne
- Autoeczematization
TINEA CAPITIS WITH AUTOECZEMITIZATION
Apple cider vinegar soaked cotton ball taped under occlusion to “ringworm” over night.
WHAT IS THE DIAGNOSIS

- Unilateral Laterothoracic Exanthem
- Molluscum Dermatitis
- Allergic Contact Dermatitis
- Pityriasis Lichenoides
UNILATERAL LATEROTHORACIC EXANTHEM

• First recently described in 1992, but some reports back to 1962
• Most common in 1 – 5 year olds
• Starts at lateral trunk and less often inguinal or arm and then spreads centrifugal
• Remains unilateral during spread but will become bilateral
• Associated low grade fever, resp or GI symptoms, lymphadenopathy and malaise
• Unclear infectious etiology
• Eruption resolves in 3 – 4 weeks, but can be up to 8 weeks
• Treatment is supportive
ERYTHEMA INFECTIOSUM

- **Incubation**: 6-14 days
- **Age**: 3-12 yrs
- **Prodrome**: none
- **Fever**: low-grade
- **Lesions**: maculopapular then reticulated
- **Distribution**: slapped cheeks then to trunk and exts
ERYTHEMA INFECTIOSUM

- Etiology: parvovirus B19
- Treatment:
  - Hemolytic disorders closely follow
  - Immunocompromised – bone marrow suppression - IVIG
  - Supportive care
WHAT IS THE DIAGNOSIS

• Varicella Eruption
• Papulovesicular Acral Syndrome
• Eruptive Juvenile Xanthogranulomomas
• Classic Cutaneous Mastocytosis (urticaria pigmentosa)
PAPULOVESICULAR ACRAL SYNDROME – GIANOTTI-CROSTI

- **Incubation**: 3-6 dys
- **Age**: 1-6 yrs
- **Prodrome**: none
- **Fever**: low-grade
- **Lesions**: papulovesicular
- **Distribution**: face, buttocks and lower exts
PAPULOVESICULAR ACRAL SYNDROME

- Etiology: multiple viral infections have been associated
- Treatment: antipruritics, supportive care, can last 8 – 12 weeks
OTHER CUTANEOUS INFLAMMATORY DISORDERS
WHAT IS THE DIAGNOSIS

- Pityriasis Rosea
- Lichen Planus
- Syphilis
- Psoriasis
LICHEN PLANUS

- Etiology unknown – proposed cell mediated autoimmune response
- Morphology – shiny, flat-topped polygonal violaceous papules
- Distribution – can be diffuse but most commonly lower extremities
- In children 40% of the cases will have mucosal lesions
- Consider lichen planus drug eruptions
  - in children likely medications are griseofulvin, NSAIDs and phenytoin
- Skin biopsy is usually diagnostic
- Treatment:
  - Topical steroids, topical calcineurin inhibitors
  - UV therapy
  - Antihistamines for pruritus
WHAT IS THE DIAGNOSIS

- Pityriasis Rosea
- Syphilis
- Pityriasis Lichenoides Chronica
- Psoriasis
PITYRIASIS LICHENOIDEIS

- Acute and chronic forms
- Etiology unknown, but T cell clonality is known
- Considered a benign lympho-proliferative disorder in which the host immune reaction prevents evolution to lymphoma
- Rare reports of Cutaneous T cell Lymphoma in patients with history pityriasis lichenoides dictate close follow up in these patients
- Treatment may be systemic antibiotics – azithromycin or tetracyclines
- Most effective treatment is UV light therapy
WHAT IS THE DIAGNOSIS

- Atopic Dermatitis
- Cutaneous T Cell Lymphoma
- Tinea versicolor
- Psoriasis
CUTANEOUS T CELL LYMPHOMA

- Primary cutaneous lymphoma
- Clinical presentation of CTCL in children is highly variable
  - Consider with any cutaneous eruption that is not resolving as expected
  - Alopecia mucinosis, pityriasis lichenoides-like CTCL, purpuric CTCL
- Hypopigmented variant is most common in children
- Treatment – limited patch stage can be treated with potent topical steroids, UV light therapy and topical bexarotene (topical retinoid)
- Followed by pediatric oncology due to concern for increase risk of Hodgkin lymphoma
WHAT IS THE DIAGNOSIS

- Tinea facei/corporis
- Seborrheic dermatitis
- Atopic Dermatitis
- Neonatal Lupus Erythematosus
NEONATAL LUPUS ERYTHEMATOSUS

- Variant of lupus in neonates with mothers with or tendency toward SLE, Sjogrens or undifferentiated autoimmune syndrome
- NLE can range from in number of affected systems
- ½ of affected mothers are asymptomatic at time of birth and must be evaluated and followed
- 50-78% of affected babies will have skin findings presenting within weeks of birth and resolve by 6-12 months of age
- Anti- Ro(SS-A), anti- La (SS-B) and anti u1RNP – with Ron being the causative antibody in heart block
- Cutaneous lesions can be treated with topical steroids and calcineuron inhibitors, but this has not been shown to prevent scarring
WHAT IS THE DIAGNOSIS

- Systemic Lupus Erythematosus
- Juvenile Dermatomyositis
- Acute Urticaria
- Polymorphous Light Eruption
JUVENILE DERMATOMYOSITIS

- Immune mediated small vessel vasculopathy, myositis and dermatitis most likely due to environmental triggers and immune dysfunction
- 25% of DM patients are less than 18 years at the time of onset
- Cutaneous lesions are found in 75% of affected children at presentation
- Cutaneous findings:
  - Facial lesions, Gottron’s papules, periungual telangiectasias, shawl sign and can spare sun protected areas
- Treatment – systemic steroids, IVIG, methotrexate, rituximab
GENETIC SKIN DISORDERS
ICHThYOSIS
INCONTINENTIA PIGMENTI
CLINICAL FINDINGS ASSOCIATED WITH ATOPIC DERMATITIS THAT COMPLICATE THE CLINICAL PRESENTATION
PERIORAL DERMATITIS
AUTOECZEMATIZATION

- Very sudden onset often diffuse monomorphic eruption
  - Face, extensor surface of arms and hands and feet are common locations
- Severity of pruritus is variable
- Etiology is not completely understood
  - abnormal immune recognition of autologous skin antigens,
  - increased stimulation of normal T cells by altered skin constituents
  - dissemination of infectious antigen with a secondary response, and
  - hematogenous dissemination of cytokines from a primary site.
- Typical presentation
  - Cutaneous infection prior to eruption (i.e. tinea) initiate treatment and then eruption occurs
AUTOECZEMATIZATION

- Difficult to treat and most often allowing it’s natural progression about 2 to 4 weeks may be necessary
- Topical steroids may be helpful
- Must finish treatment of cutaneous infection, so differentiating from drug hypersensitivity is important
- Please consider a systemic allergic contact for example ingesting nickel containing foods in a patient with a nickel allergic contact allergy will present with very similar skin lesions and require limiting or excluding nickel from their diet
IRRITANT OR ALLERGIC CONTACT DERMATITIS

- This diagnosis may fit all of the atopic dermatitis criteria but please look for patterns that DO NOT fit the typical distribution in atopic dermatitis
- May have very well demarcated lesions, but if related to topical creams will not
- Very pruritic
- History may show this is a new area of involvement in an already diagnosed atopic dermatitis patient
- Remember to focus family to report all contacts to the skin not just new contacts
Be alert to this diagnosis

Atopic dermatitis patients are at higher risk due to skin barrier dysfunction and immune dysregulation

Try to remove or greatly limit contact

May try to use a barrier – thick cotton sock under shin guard

Once diagnosed may use topical steroid class three or below to affected site (except face) twice a day for 2 weeks once a day for two week then stop

If an allergic response may last three weeks after discontinuing contact

Consider consult for patch testing
Atopic dermatitis and psoriasis: two different immune diseases or one spectrum? Emma Guttman-Yassky and James G Krueger Current Opinion in Immunology, 2017-10-01, Volume 48, Pages 68-73, Copyright © 2017

Pathophysiology of Atopic Dermatitis and Psoriasis: Implications for Management in Children. Chovatiya R¹, Silverberg JI²

HAVE OUR EYES BEEN TRICKING US
HAVE OUR EYES BEEN TRICKING US

“...when considering the range of AD phenotypes, a case can be made that psoriasis and AD exist across a spectrum where polar T-cell axes can be variably present and create some overlapping disease characteristics.”

RSS Emma Guttman-Yassky and James G Krueger Current Opinion in Immunology, 2017-10-01, Volume 48, Pages 68-73, Copyright © 2017
VARIOUS AD PHENOTYPES
VARIOUS PSORIASIS PHENOTYPES
“The next decade of research will be dominated by extensive genetic, molecular, and clinical phenotyping of patients in order to understand which pathologic mechanisms are most relevant. The era for personalized medicine in AD and psoriasis is upon us!”

THE END ...THANKS