Pediatric Dermatology

Pediatric Dermatology

A Primer

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About this Book

This is a manual meant to accompany rotation or a course in pediatric dermatology. It covers the basics of treating common skin conditions in children, adolescents, and adults.

You will find:

- Skin conditions selected and placed specific to chapters, with colour-coded pages for quick access
- Syndromes presented in easy-to-read text, with accompanying pictures to allow an in-depth read of the material, or just a quick glance
- Boxed features highlighting important aspects of certain skin conditions
- Quizzes ending each chapter to test knowledge

It is important to note that this is not an exhaustive guide or reference and is only meant to be used as a quick reference presented in an efficient format.

Note: Medical knowledge is constantly changing, new information will become available and new treatments approved after the publishing of this book, which may make parts of it out of date. Even at the time of writing, this is not a complete or exhaustive list of diseases and therapeutic options, but is geared toward those conditions seen most frequently in clinic. It is designed to be read as a companion manual for a rotation in pediatric dermatology or an introduction to pediatric dermatology course.

Additional resources for information available online include:

Dermnetnz.org

Emedicine.medscape.com/dermatology

Merckmanuals.com/professional/dermatologic-disorders

UptoDate.com

Pedsderm.net/for-patients-families/patient-handouts/

This book is meant as one among many resources used by medical practitioners in their care of patients with skin disease. It does not substitute for the sound judgement of a practitioner, who knows both the patient and the medications available in their setting, to make the correct treatment recommendations.

Version 1.

Made possible by a generous grant from the Ellis Foundation

Author's Note

Skin disease is among the most common category of disease in children. Some skin conditions are very common. Atopic dermatitis can be seen in up to 15% of some populations. Other skin diseases are exceedingly rare, but bring significant morbidity to children affected. Pediatric dermatology is both a small and subspecialized field of medicine and at the same time covers one of the most common components of a primary care or pediatric practice.

There are many excellent and comprehensive textbooks on Pediatric Dermatology and these are listed in the References section. The goal of this manual is to provide an introduction to the field and review of some of the most frequently seen conditions in an accessible format while touching on a few of the more uncommon pediatric dermatology conditions. Each chapter is followed by a few multiple-choice questions to highlight key facts from the chapter. At the end is a section with some detail about skin care as well as review of several of the medications used frequently in dermatology as these might not be familiar to practitioners from other fields of medicine.

Dr. Wingfield E. Rehmus MD, MPH

I am originally from the United States and in 2006, my husband and I moved with our three young children to the Republic of Palau, an island country with a population of about 20,000 in the Western Pacific, for 2 years. Many of the images in this manual come from my time in Palau. I am ever grateful to the people of Palau who welcomed us and shared their beautiful country with us. The first iteration of this book was not focused on pediatrics, but on island dermatology, with emphasis on including images of skin of colour and medications available in that setting.

After living in Palau, I moved to Vancouver and began work in pediatric dermatology. Working in peds derm has been one of the great joys of my life. My job is all about human connection. Without discussing the latest Disney and Pixar movie, sparkly shoes, or the most recent sporting news, I have no therapeutic alliance. What's more, a key component of my job as a pediatric dermatologist is actually celebrating how beautiful each child is even as the world may tell them otherwise due to a visible skin condition.

I hope that this manual will serve as a small token of my thanks for the generosity that has been offered me by my patients and colleagues over the years. I also hope that it will spark a passion for pediatric dermatology in readers who can continue to provide care for kids with skin disease wherever they find themselves so that we can all work together to help kids feel more comfortable in their own skin.

CHAPTER 1: INTRODUCTION TO CLINICAL DERMATOLOGY

Skin Structure and Function

In order to understand the basic pathophysiology of skin disease, it is important to begin with a review of the components of the skin and their function. The skin has three main parts: the epidermis, dermis, and subcutaneous tissue.

The epidermis is comprised of several layers. The innermost basal layer is attached to the dermis at the complex dermal-epidermal (DE) junction. The basal layer contains primarily keratinocytes, but has occasional pigment producing melanocytes. Keratinocytes progress upwards through the epidermis to the outermost layer, the stratum corneum, before sloughing off. The outer layer has a crucial role in maintaining the barrier function of the skin. Within the middle layers of the epidermis are Langerhans cells, an important part of the skin's immune system that act as antigen presenting cells.

The dermis is the portion of the skin immediately under and connected to the epidermis. The dermis is primarily comprised of collagen with intervening elastic fibers, blood vessels, and nerves. The dermis provides structure and allows the skin to maintain elasticity and resist stress. Blood vessels within the skin provide nutrients and allow for transport of inflammatory cells to areas of infection or inflammation. Nerves within the skin mediate pain, touch, pressure, and itch sensation. The thickness and constitution of the dermis varies by body site, for example, with thinner dermis on the face and thicker on the back.

Beneath the dermis is the subcutaneous layer comprised of fat cells (adipocytes) held together in lobules separated by fibrous septa. The subcutaneous fat layer gives form and cushioning as well as functioning as an endocrine organ.

When the skin is affected by illness or injury, it is not able to perform these functions. Problems such as cancer, dehydration, hyperthermia, hypothermia, infarction, infection, and pruritus may result.

In addition, the skin is an important component of appearance, and healthy skin is frequently seen to convey beauty. Patients with skin disease, particularly when it is visible to others, may feel stigmatized. This psychological impact of skin disease can be out of proportion to what others expect and can even outweigh the physical impact of the disease.

Using standard terminology in the description of skin lesions allows for easy communication between health practitioners regarding the nature of the lesions being evaluated. This terminology is commonly referred to as morphology.

The skin has many important functions. The 5 basic functions of the skin are:

1. Barrier formation
2. Thermoregulation
3. Photoprotection
4. Cutaneous circulation

5. Immunological protection

Morphology: Primary Lesions

Primary Lesions

The primary lesion is the true state of the illness when it first appears or is unchanged by outside forces such as infection or scratching. When evaluating skin disease, it is helpful to find the primary lesions.

Macule	A flat lesion with no surface change <1cm in diameter.
Patch	A flat lesion with no surface change >1cm in diameter.
Papule	A raised or scaly lesion <1cm in diameter.
Plaque	A raised or scaly lesion >1cm in diameter.
Vesicle	A fluid-filled lesion <1cm in diameter.
Bullae	A fluid filled lesion >1cm in diameter. **Flacid bullae**: Thin walled, ruptures easily, rarely seen intact. **Tense bullae**: Thick walled, appears tense.
Pustule	A superficial cavity containing purulent material, usually <1cm in diameter.
Nodule	A raised, solid lesion involving the dermis and/or subcutaneous tissue, usually >1cm diameter.
Wheal	A transient, elevated lesion due to superficial edema, often pink to red with surrounding pallor.
Telangiectasia	Persistent dilation of superficial blood vessels in the skin.
Comedone	Plugged secretions of a pilosebaceous unit (a hair follicle and its accompanying sebaceous gland) **Open comedone:** Small 1-2mm white to skin coloured papule **Closed comedone:** Small 1-2mm papules with a brown-black central opening.

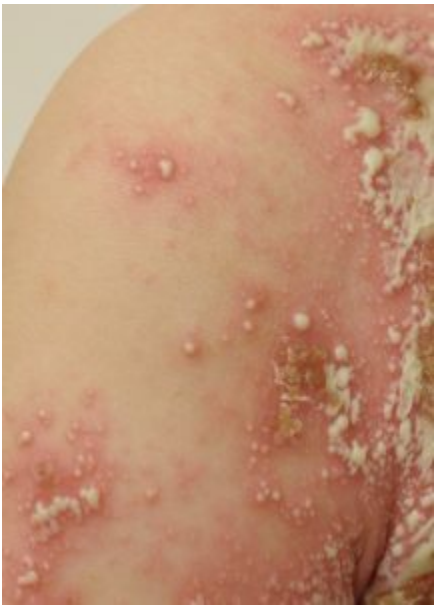

Image 1.1: Pustules of varying sizes

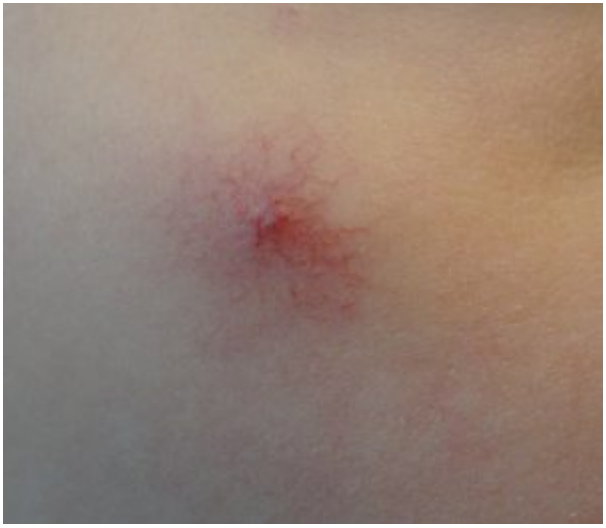

Image 1.2: Telangiectasias surrounding a vascular papule

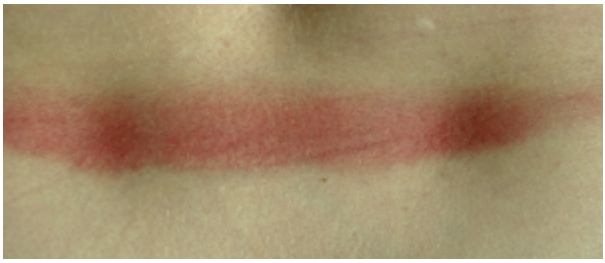

Image 1.3: A patch of sunburn

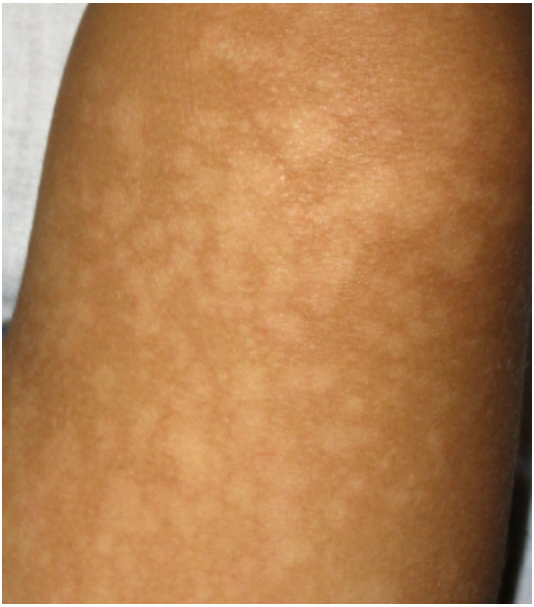

Image 1.4: Hypopigmented macules coalescing into patches

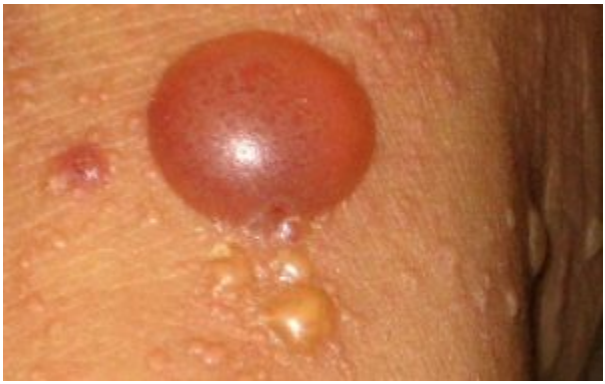

Image 1.5: Vesicles with a single bullae

Morphology: Secondary Lesions

Secondary Lesions

Secondary features occur when the basic form of the lesion has changed over time. This may be from a variety of factors, such as scratching or rubbing by the patient, infection or trauma.

Crust	Dried serum, pus or blood on the surface of a lesion.
Scale	Visible flakes of stratum corneum — scale can be thin or thick, adherent or flaky. It may be white, silvery or yellow in colour.
Erosion	A slightly depressed area of loss of epidermis. Heals without scar formation.
Ulcer	A depressed area corresponding to loss of epidermis and dermis (and possibly the subcutis) — heals with scar formation.
Scar	Fibrous tissue which forms a new surface after the healing process.
Atrophy	Thinning of one or more layers of the skin — notable by the appearance of a thin, shiny surface, sometimes with visible blood vessels below (epidermal atrophy), or a depression (dermal atrophy).
Lichenification	Thickening of the epidermis with exaggeration of skin markings.
Fissure	A linear cleavage in the skin. It may be dry or moist.
Excoriation	Loss of the epidermis and superficial dermis due to scratching, may be linear or punctate.

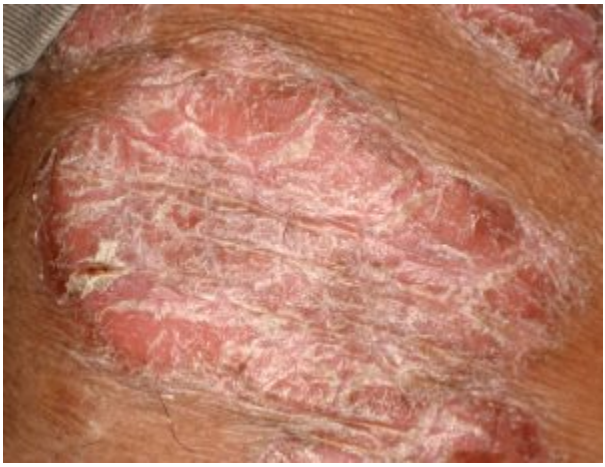

Image 1.7: Plaque with overlying scale

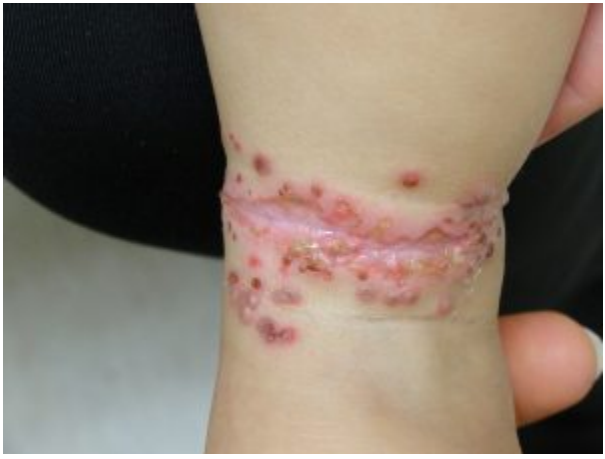

Image 1.8: Herpetic vesicles leading to erosions and ulceration

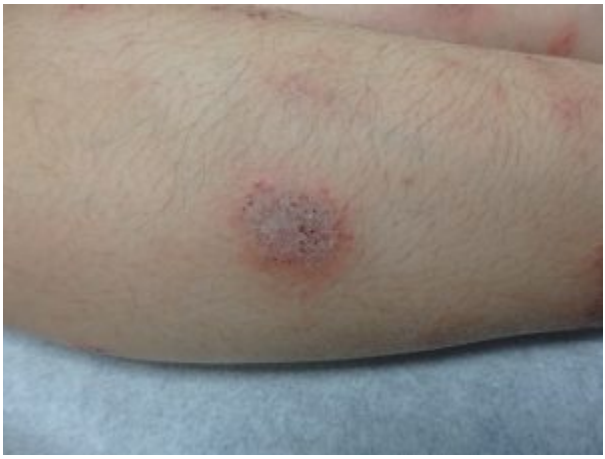

Image 1.9: Erythematous plaque with scale and crust

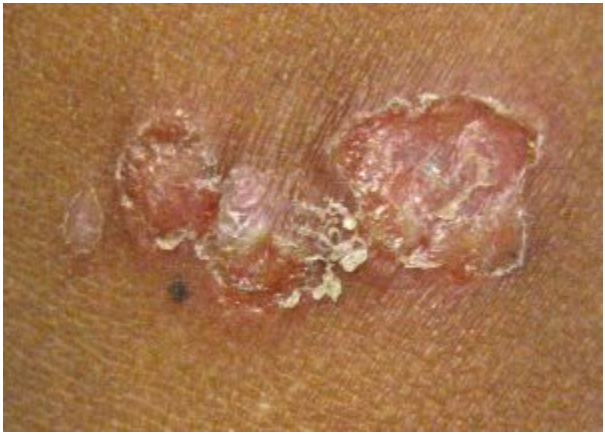

Image 1.10: Crusted plaques

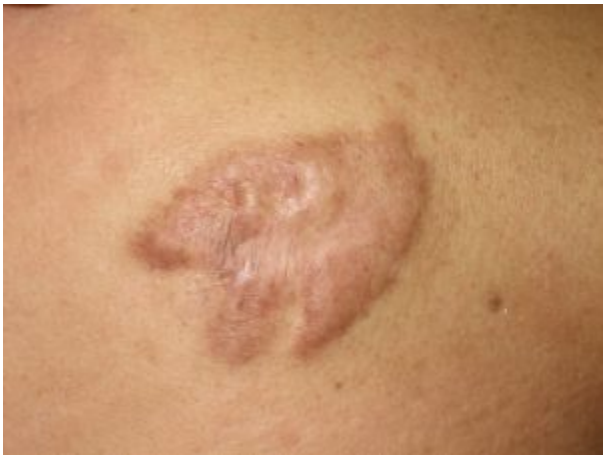

Image 1.11: Scar with hyperpigmentation and dermal thickening

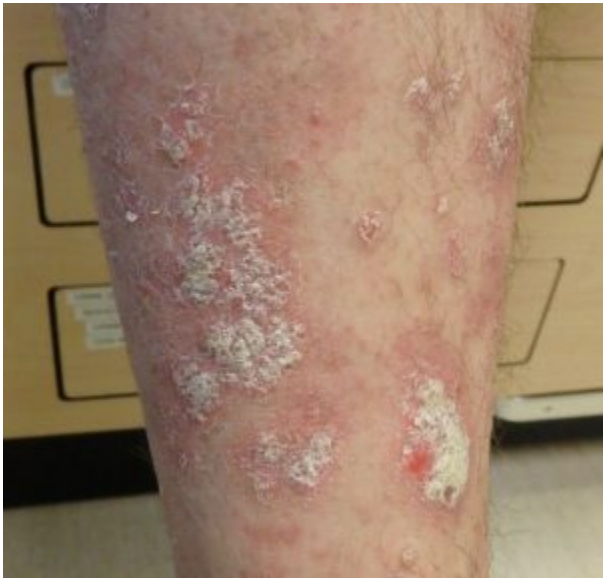

Image 1.12: Erythematous plaques with thick scale

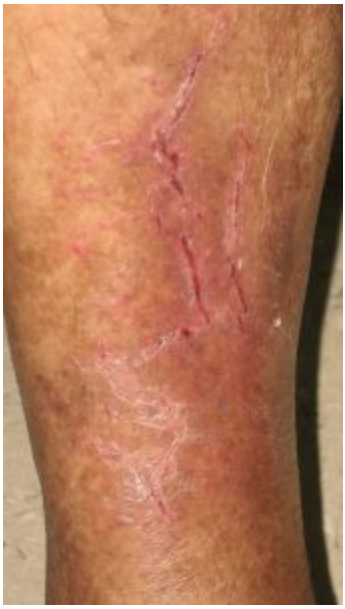

Image 1.13: Fissures due to extremely dry skin

Morphology: Surface Change and Shape

Surface Change and Shape

The skin is a three-dimensional structure and there are several terms that can be used to describe the surface texture or shape of lesions.

Lichenoid	Flat-topped and slightly scaly
Dome-shaped	Smoothly rounded
Verrucous	A rough and irregular or bumpy surface
Ulbilicated	Has central depression
Filiform	Thread-like
Pedunculated	On a narrow stalk

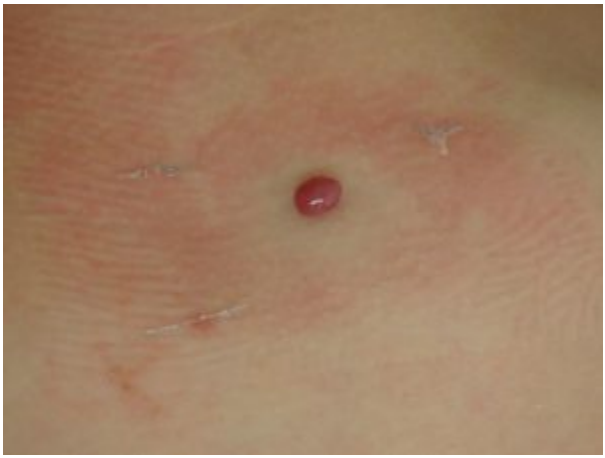

Image 1.14: Pedunculated vascular papule

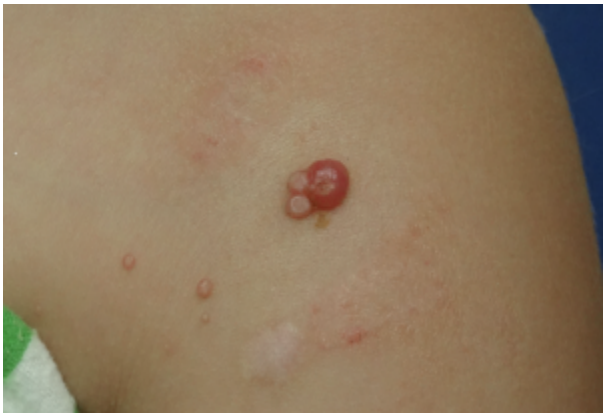

Image 1.15: Cluster of umbilicated papules with surrounding dermatitis

Morphology: Grouping of Lesions

Grouping of Lesions

In addition to describing the appearance of the individual lesions and any changes which have occurred, it is often helpful to describe the shape of the lesion or the pattern of distribution with multiple lesions.

Discrete	Individual lesions remain separate from each other.
Grouped or clustered	Multiple individual lesions appearing in one area.
Confluent	Individual lesions tend to blend together where they touch to form larger lesions.
Annular	Ring shaped; arranged in a circle with prominence of features on the periphery
Arcuate	Arranged in an arc-like formation
Nummular	Coin-shaped lesions; round and discrete but usually not annular
Reticulated	Net-like or lacy pattern
Guttate	Drop-like lesions, usually referring to flares of psoriasis with small plaques
Morbilliform	Appearing in a measles-like fashion with diffuse macular and papular lesions
Dermaromal	Appearing in an area which corresponds to a single sensory nerve root
Linear	Arranged in a line
Serpentine	Arranged in a snake-line linear pattern

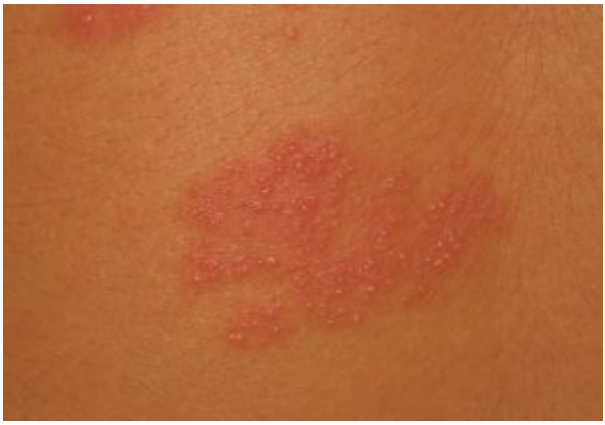

Image 1.16: Clustered vesicles on an erythematous base

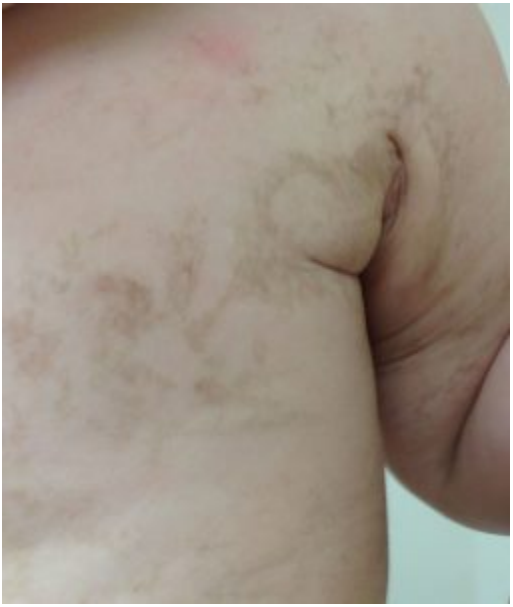

Image 1.17: Hyperpigmentation following the lines of Blaschko

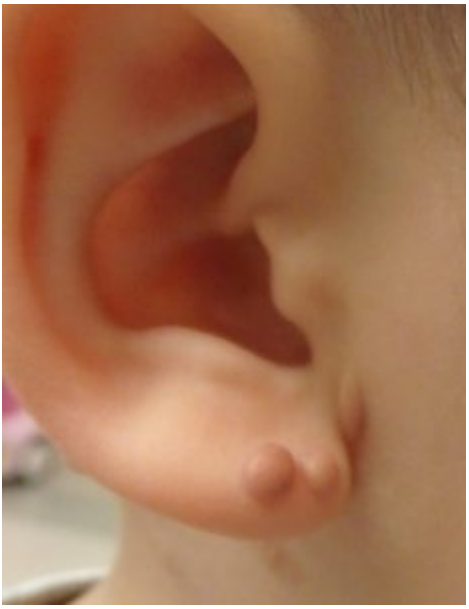

Image 1.18: Dome shaped papules on the ear

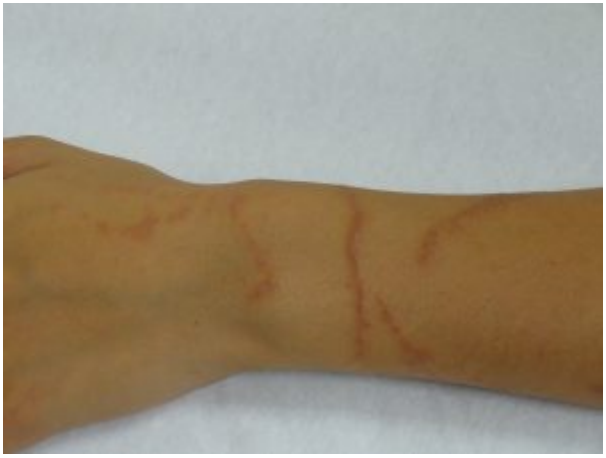

Image 1.19: Linear vesicles from sea jelly sting

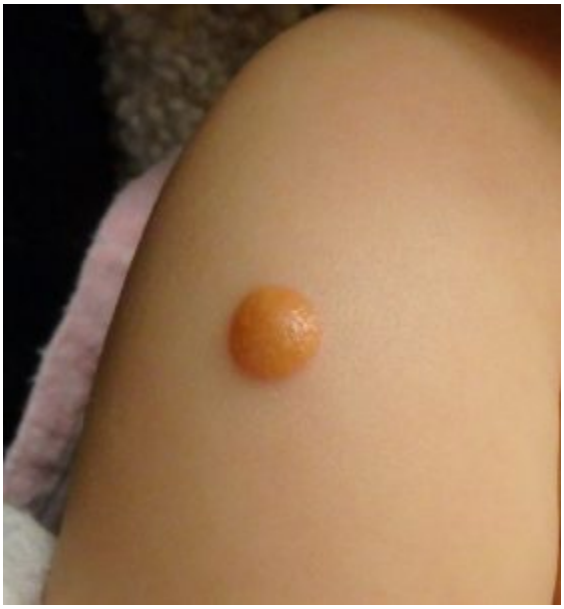

Image 1.20: Dome shaped papule

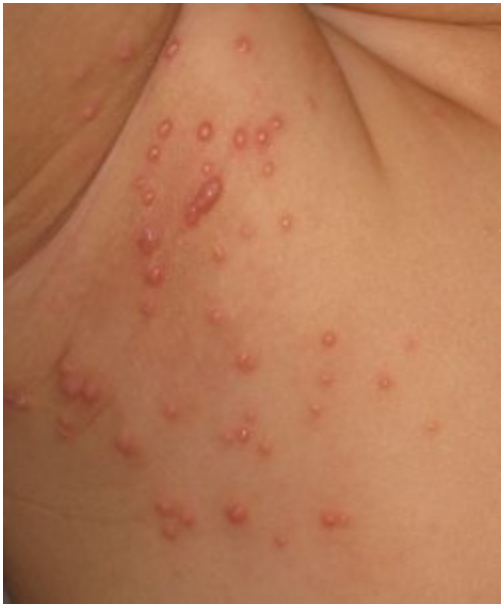

Image 1.21: Discrete papules that do not coalesce

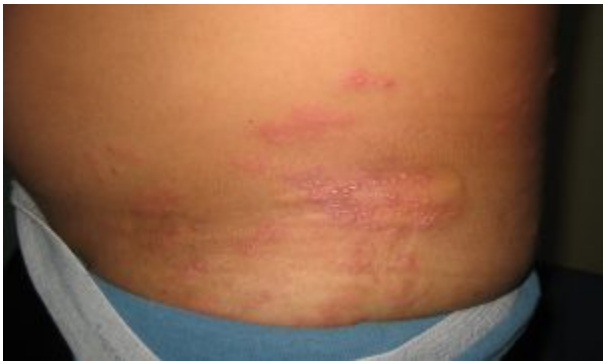

Image 1.22: Vesicles in a dermatomal distribution

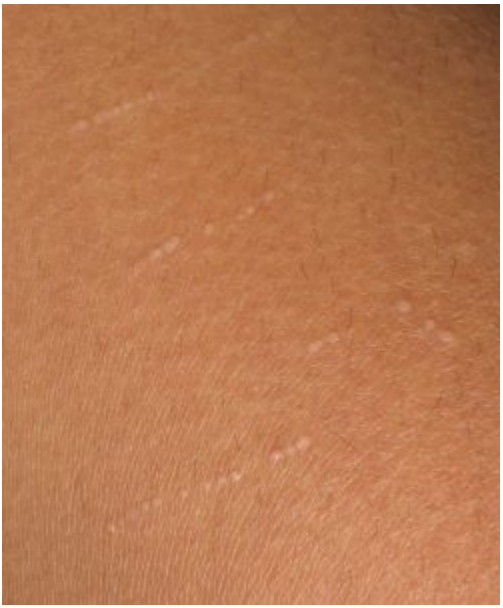

Image 1.23: A linear array of tiny papules

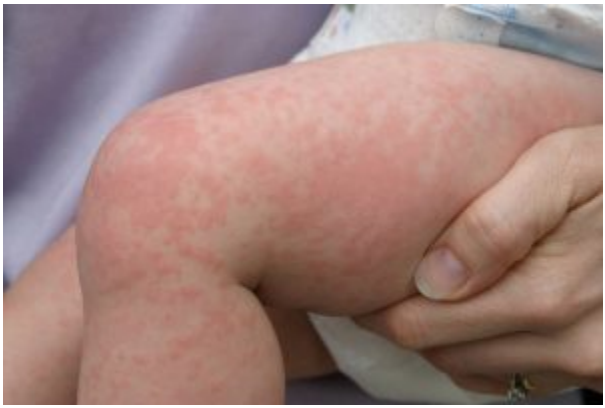

Image 1.24: A morbilliform eruption

Selected Differential Diagnosis

In dermatology, the differential diagnoses are most often organized by the morphology of the lesions seen on physical exam. Other variables considered include the distribution of lesions, relevant exposures, as well as the age and overall health of the patient. Below are common conditions encountered in pediatric dermatology for the select morphologies.

Scaly Papules or Plaques
• Dermatitis
  ◦ Atopic dermatitis
  ◦ Nummular dermatitis
  ◦ Contact dermatitis
• Seborrheic dermatitis
• Psoriasis
• Tinea corposis
• Pityriasis rosea
• Pityriasis lichenoides
	Hyperpigmentated
	• Post-inflammatory hyperpigmentation
	• Café-au-lait macules
	• Dermal melanocytosis
	• Acanthosis nigricans
	• Nevi: acquired and congenital
	• Retention hyperkeratosis
	• Lentigo

Hypopigmented or Depigmented • Pityriasis versicolor • Pityriasis alba • Vitiligo • Post-inflammatory hypopigmentation • Nevus depigmentosis • Nevus anemicus	**Solitary Papules** • Molluscum contagiosum • Dermatofibroma • Verruca vulgaris (wart) • Skin tags • Arthropod bites • Pilomatricoma • Prurigo nodules • Scabetic nodules • Juvenile xanthogranuloma
Vascular Appearing Papules and Plaques • Infantile hemangioma • Congenital hemangioma • Pyogenic granuloma • Spider angiomas • Spitz nevus • Amelanotic melanoma	**Morbilliform Eruption** • Morbilliform drug eruption • Viral exanthem • Kawasaki disease • DRESS or early SJS/TEN • Connective tissue disease
Vesicles and Bullae • Viral infection • VZV • HSV • Hand foot and mouth • Bullous impetigo • Acute contact dermatitis • Drug reactions – SJS/TEN • Erythema multiforme • Epidermolysis bullosa • Incontinentia pigmenti	**Pustules** • Acne vulgaris • Folliculitis • Furuncles • AGEP (Drug reaction) • Impetigo • Candidiasis • Hidradenitis suppurativa • Scabies • Pustular psoriasis

Diffuse Erythema • Viral exanthems • Drug reactions • Sunburn • Atopic dermatitis • Psoriasis • Pityriasis rubra pilaris	

Quiz

1. The innermost basal layer is attached to the dermis at the stratum corneum.

a. True
b. False

2. Within the middle layers of the epidermis are _____ cells, an important part of the skin's immune system that act as antigen presenting cells.

a. Keratinocytes
b. Melanocytes
c. Langerhans
d. Adipocytes

3. Which of the following is NOT a primary lesion?

a. Bullae
b. Comedone
c. Fissure
d. Telangiectasia

4. The thickness and constitution of the dermis varies by body site.

a. True
b. False

5. Which of the following describes a thickening of the epidermis with exaggeration of skin markings?

a. Lichenification
b. Atrophy
c. Erosion
d. Excoriation

6. What does morbilliform grouping describe?

a. Individual lesions that tend to blend together where they touch to form larger lesions
b. Appearing in an area which corresponds to a single sensory nerve root
c. Individual lesions remain separate from each other
d. Appearing in a measles-like fashion with diffuse macular and papular lesions

7. Differential diagnoses are often organized by lesion morphology during a physical exam. What other variables are considered?

a. Relevant exposures
b. Age of patient
c. Overall patient health
d. Distribution of lesions
e. All of the above

8. Which of the following conditions may cause morbilliform eruptions?

a. DRESS, Kwasaki disease, and viral exanthem
b. Psoriasis, connective tissue disease, and skin tags
c. Nevus anemicus, sunburn, and early SJS/TEN
d. Morbilliform drug eruption, HSV, and impetigo

9. What kind of cell gives form and cushioning as well as functioning as an endocrine organ?

a. Keratinocytes
b. Melanocytes

c. Adipocytes
d. Langerhans

Answers: 1. B 2. C 3 .C 4 .A 5 .A 6. D 7 .E 8. A 9. D

CHAPTER 2: RASHES OF THE NEWBORN

Benign Skin Changes of the Newborn

Erythema Toxicum Neonatorum

Erythema Toxicum Neonatorum (ETN) is a common benign skin disorder that occurs in nearly half of full-term neonates and usually appears in the first 3 days of life. It is less common in premature infants.

ETN is usually not present at birth, but begins between 1-2 days of life. It presents with tiny papules, pustules or vesicles (1-2mm) with a blush of redness around them. They distributed mostly on the trunk, occasionally involving the face, buttocks and extremities. The palms and soles are almost never affected.

No management is required, as the rash is asymptomatic and resolves spontaneously. Alternate diagnoses should be considered if the rash is present immediately from birth, does not resolve with the expected time course, or the neonate is systemically unwell.

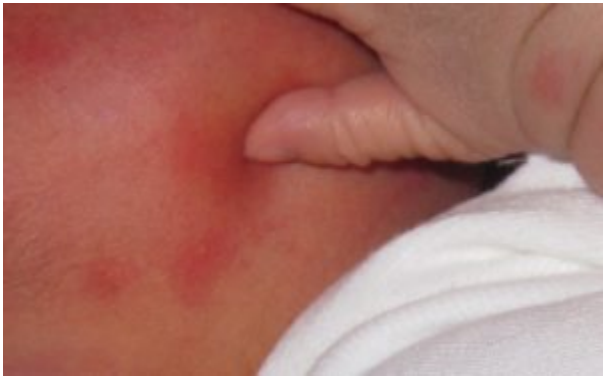

Image 2.1: Erythema toxicum with tiny papules surrounded by a blush of erythema – Image credit to Dr. Joseph Lam

Transient Neonatal Pustular Melanosis

Transient neonatal pustular melanosis is usually present from birth and affects ~5% children with dark skin. It also presents with superficial pustules, however these are larger than those seen in ETN. When they resolve, they leave behind a collarette of scale and characteristic brown spots. As with ETN, it is a benign and self-resolving.

Neonatal Cephalic Pustulosis

Neonatal cephalic pustulosis, more commonly known as neonatal acne, is a pustular rash that usually starts between 2-3 weeks of life and resolves by ~3 months. It is distributed on the face but does not have comedones ("blackheads" and "whiteheads") like typical acne. It is thought caused by a reaction to Malassezia yeast. As the rash is self-limited, treatment is usually unnecessary.

Infantile Acne

Infantile acne is a form of acne that occurs slightly later than this (between 2-12 months of age) and differs in that there are frequently comedones in addition to pustules. It can result in scarring so treatments similar to those for adolescent acne are recommended (similar approach to adolescent acne). If severe, evaluation for precocious puberty is recommended.

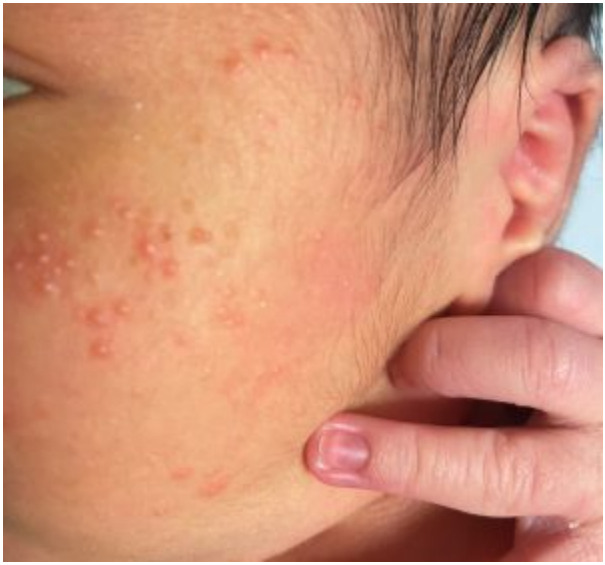

Image 2.2: Neonatal acne with inflammatory papules and pustules but no comedones

Neonatal Candidiasis

Neonatal candidiasisis a yeast infection of the skin acquired during or shortly after delivery. It usually presents around 1 week of age and affects the diaper area, but may also be seen in body folds and on the face. It consists of red patches with satellite papules and pustules. Topical antifungals are usually sufficient. Less commonly, the infection is acquired in utero and is present at birth (congenital candidiasis). This rash is more widespread and premature or unwell neonates may require IV antifungals due to a risk of systemic infection.

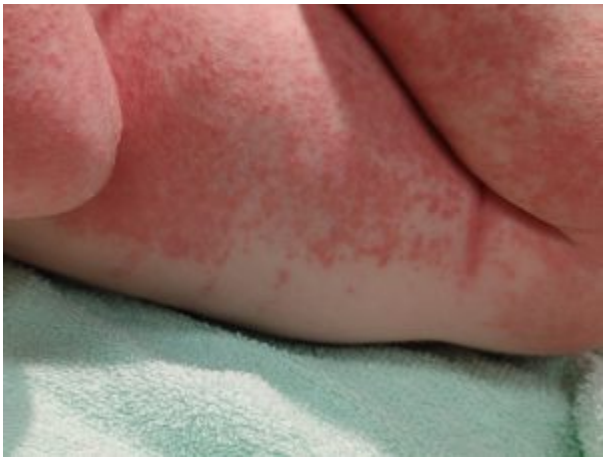

Image 1.3: Ch.2: Congenital candidiasis with tiny erythematous pustules and papules

Miliaria

Miliaria affects ~15% of newborns. It occurs due to obstruction of sweat ducts and may present as small "dew drop-like" vesicles, pustules or red bumps depending on the depth of blockage. It commonly occurs on the head, neck and upper trunk and may follow occlusion and/or sweating (such as excess warming in an incubator or tight swaddling). It resolves without treatment.

Cutis Marmorata

Cutis marmorata is a normal physiologic skin change seen in ~50% of newborns, and occasionally lasting until later in life. It is caused by changes in the tone of superficial vessels in response to the ambient temperature. It presents with a mottled (lacy or net-like) blue to red discolouration that occurs when the body is exposed to cold temperatures. The rash usually fades away when the body is rewarmed. It is important to distinguish it from cutis marmorata telangiectatica congenita (CMTC), a vascular anomaly. CMTC differs from cutis marmorata in that it does not typically fade with rewarming, may be localized and may have atrophy of the affected area.

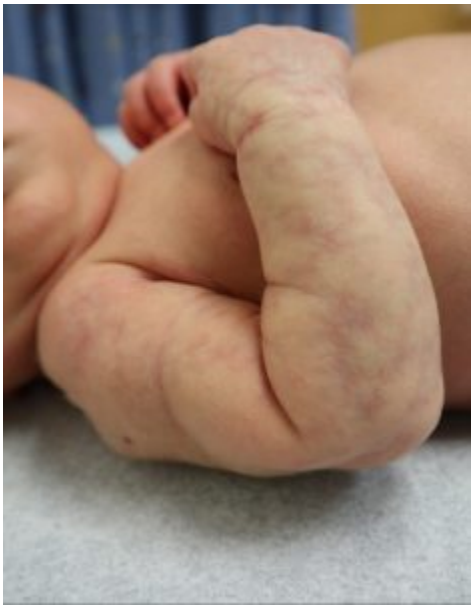

Image 2.4: Reticulate violaceous plaque with atrophy in CTMC – Image credit to Dr. Joseph Lam

Potentially Concerning Skin Changes in Newborns

Blisters

Blisterscan occur in neonates or a variety of reasons including infection, genetic blistering diseases (see Epidermolysis bullosa), and infiltration of the skin with mast cells. Appropriate testing to rule out infection is necessary and proper wound care is crucial to prevent secondary bacterial infection.

Neonatal Herpes Simplex

Neonatal herpes simplex usually presents with vesicles and occurs due to HSV exposure during vaginal delivery. Vesicles are seen most commonly on the presenting part of the baby such as the crown of the head. Neonatal HSV is more likely if the mother is experiencing her first episode of HSV, so she might not have a history of genital herpes. The rash may be present from birth if it is acquired in uterobut typically starts at least 5 days after birth. Infection may be complicated by encephalitis, and mortality is ~50% in these cases if not treated with IV acyclovir.

Neonatal Lupus

Neonatal lupusis seen in babies born to mothers with anti-Ro, anti-La, or U1RNP antibodies. The antibodies can cross the placenta and cause changes in the baby. Skin findings include annular plaques with fine scale especially on the head and neck and concentrated around the eyes. The lesions typically first appear by 2 months of age and worsen after sun exposure. While skin changes will self-resolve, babies with neonatal lupus are at risk for heart block, cytopenias, and liver function changes.

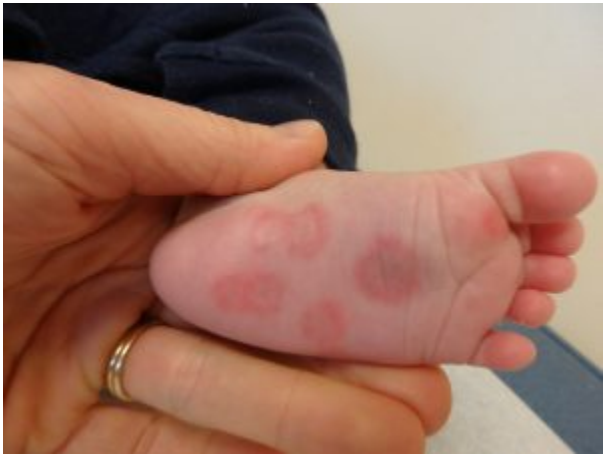

Image 2.4: Annular plaques of NLE on the feet. More typical location is the face.

Collodion Membrane

Collodion membrane is the name given to a parchment or plastic wrap-like membrane of skin that wraps some newborns. It can cause ectropion and/or eclabium. It may be the first sign of an ichthyosis, but also can be self-resolving. Treatment is with moisturizers, and possibly incubator, to help preserve skin function. The membrane will slough spontaneously and should not be removed.

Subcutaneous Fat Necrosis of the Newborn

Subcutaneous fat necrosis of the newborn occurs due to crystal formation in fat cells in newborn fat. It is seen most often in newborns who have required cooling and presents with tender red-brown nodules. Babies with extensive subcutaneous fat necrosis should not be given Vitamin D and should be followed for possible development of hypercalcemia.

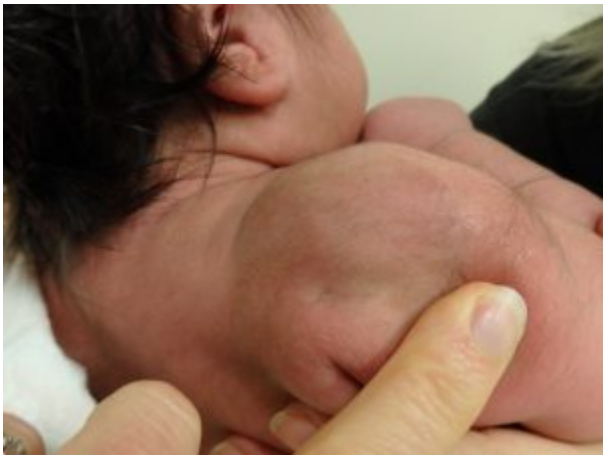

Image 2.5: Tender indurated plaque on the shoulder of a neonate with fat necrosis

Blueberry Muffin Baby

Blueberry muffin babydescribes the clinical finding of widespread red to purple papules and nodules in a newborn baby. There is a wide range of conditions that lead to the finding of blueberry muffin baby. The most common of these are congenital infections, but different forms of anemia and hematologic malignancy are among other potential causes. Evaluation for underlying cause of the nodules is imperative.

Selected causes of blueberry muffin baby:

Infections	Anemia and blood loss	Other
Congenital Rubella	Hemolytic Anemia	Leukemia Cutis
Toxoplasmosis	Twin-twin Transfusion	Neuroblastoma
Cytomegalovirus	Fetomaternal Hemorrhage	Langerhans Cell Histiocytosis
Coxsackievirus	Severe Internal Bleeding	
Parvovirus		

Quiz

1. When does erythema toxicum neonatorum (ETN) usually appear in neonates?

a. Within the first day of life
b. Within the first 2 weeks
c. Within the first 3 days of life
d. None of the above

2. What is true about neonatal candidasis?

a. It is acquired between 2-12 months of age
b. Consists of dome shaped papules and pustules
c. Infection is commonly acquired in utero
d. Topical antifungals may be sufficient for treatment

3. Where is milia often found on newborns?

a. Arms
b. Face
c. Legs
d. Abdomen

4. The superficial pustules seen in transient neonatal pustular melanosis are smaller than those seen in ETN (erythema toxicum neonatorum).

a. True
b. False

5. Which of the following are selected causes of Blueberry Muffin Baby?

a. Parvovirus
b. Hemolytic Anemia
c. Neuroblastoma
d. Severe internal bleeding
e. None of the above

6. What is true about neonatal lupus?

a. Lesions appear by 2 months of age
b. It does not worsen with sun exposure
c. Babies with it are at a risk of cytopenia
d. It can be seen with mothers with U1RNP antibodies

7. Which of the following is used to treat neonatal acne in a healthy baby?

a. Topical antifungals
b. Topical antibiotics
c. Steroid cream
d. Regular moisturizer

8. What is the biggest difference between cutis marmorata and cutis marmorata telangiectactica congenita (CMTC)?

a. Cutis marmorata is treated with topical cream and CMTC is not
b. CMTC does not fade with rewarming, while cutis marmorata does
c. Babies with CMTC should not be given Vitamin D
d. None of the above are true

9. What is the reason for undertaking immediate testing when blisters are found?

a. To see if mast cells infiltrated the skin
b. To rule out infection
c. To see if it is genetic (Epidermolysis bullosa)
d. Testing is not necessary

10. Why does miliaria occur?

a. Antibodies that cross the placenta before birth
b. Obstruction of sweat ducts
c. Yeast infection
d. Crystal formation in fat cells in newborns

Answers: 1. C 2. D 3. B 4. B 5. E 6.B 7.A 8. B 9. B 10. B

CHAPTER 3: ECZEMATOUS DISORDERS

Atopic Dermatitis

What is it?

Atopic dermatitis is a very common skin problem seen mostly in children; up to 15-20% of children are estimated to have it. Atopic dermatitis patients have higher than normal rates of asthma and allergies and may have family members who have asthma, allergies or atopic dermatitis. Atopic dermatitis is often called eczema and causes itchy red areas to appear on the skin. It is caused by a complex interaction between the environment, skin and immune system. It tends to come and go and sometimes will be itchy even before the rash is seen. Most patients with atopic dermatitis present as young children and many improve with time. Some continue to have severe skin problems into adulthood.

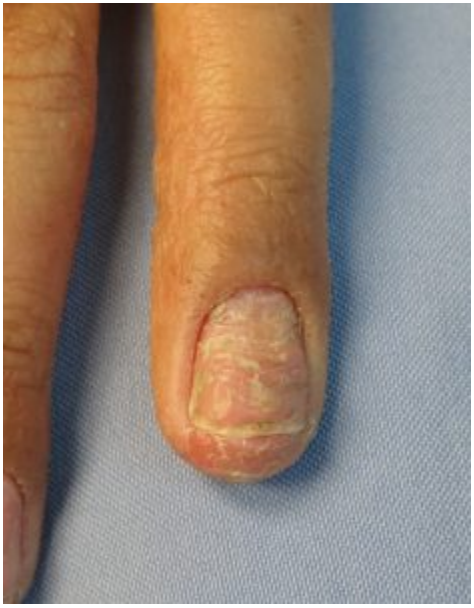

Image 3.1: Nail changes due to severe hand eczema

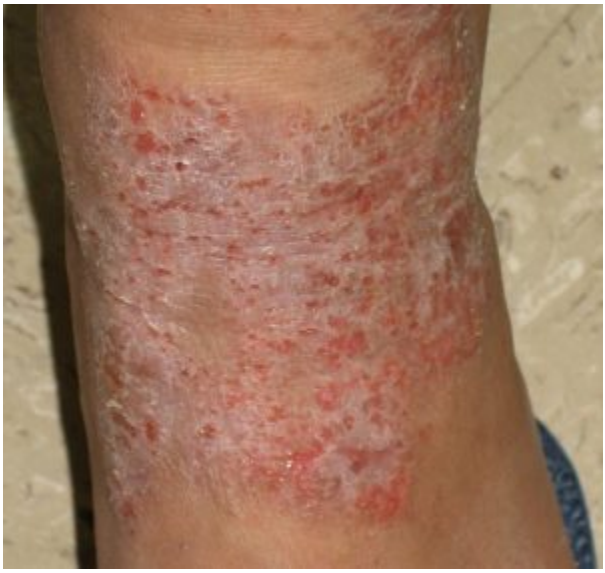

Image 3.2: With secondary infection

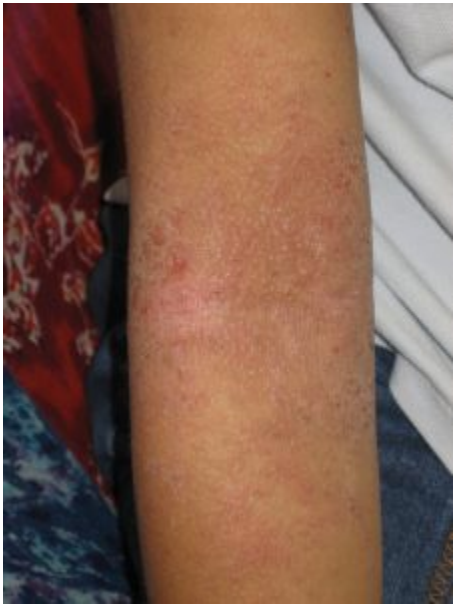

Image 3.3: Typical anticubital fossa plaque

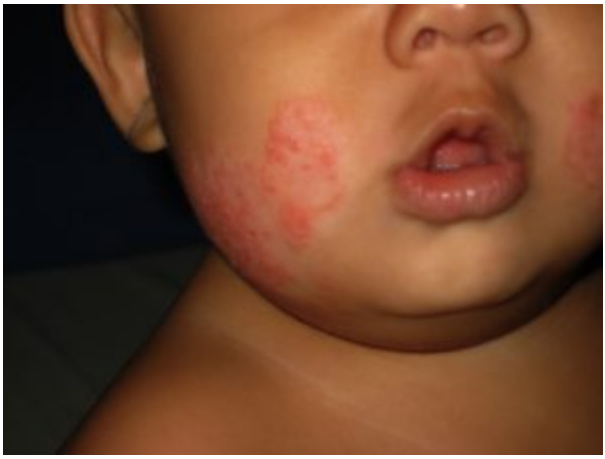

Image 3.4: With background hypopigmentation

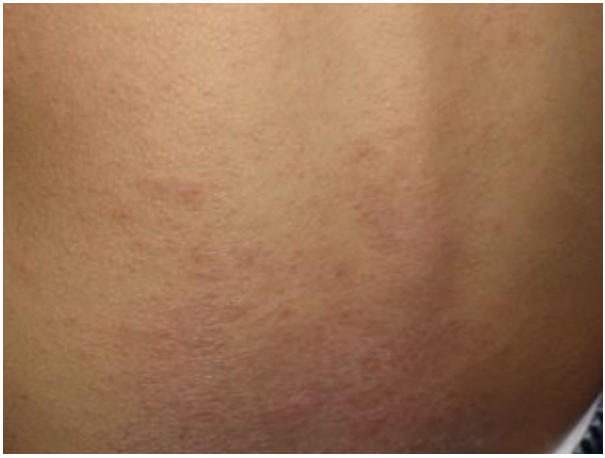

Image 3.5: With follicular prominence

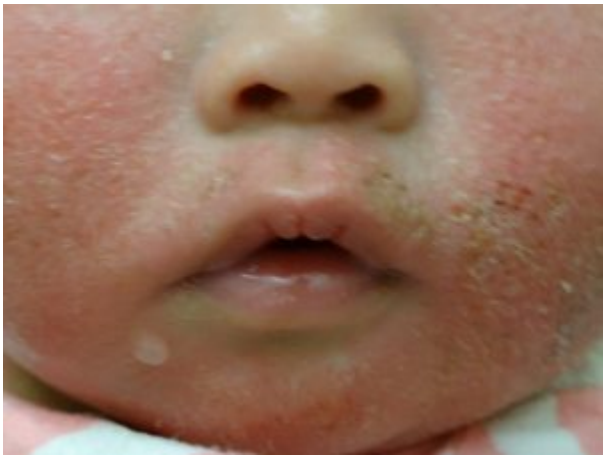

Image 3.6: Typical facial plaques in infancy

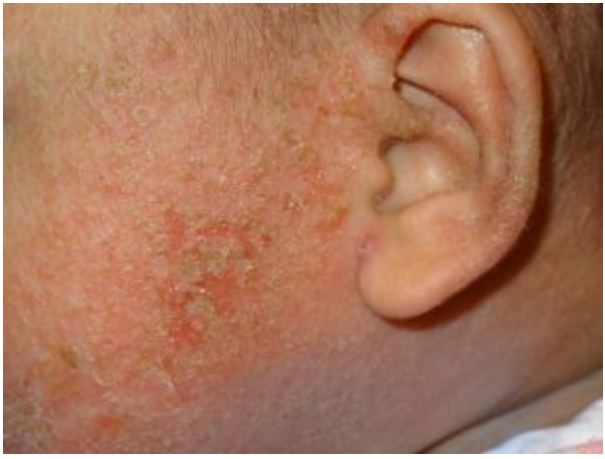

Image 3.7: Note the fissure on the earlobe

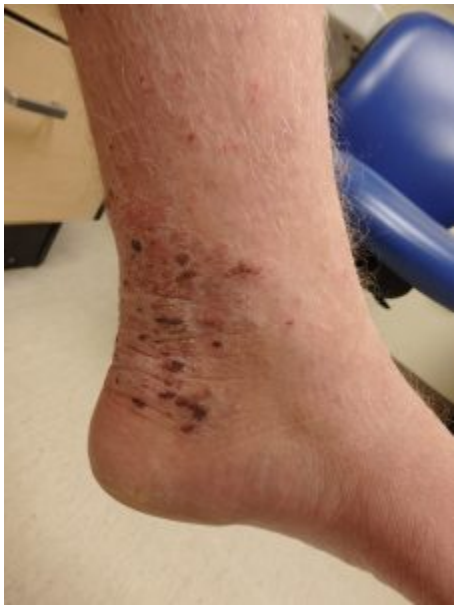

Image 3.8: Lichenified and eroded plaque

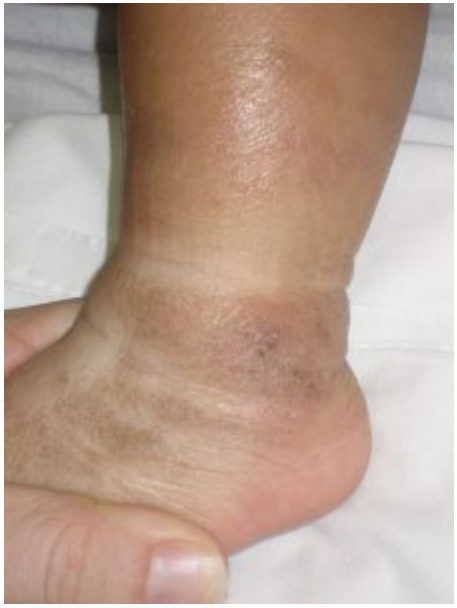

Image 3.9: Lichenification at the ankle

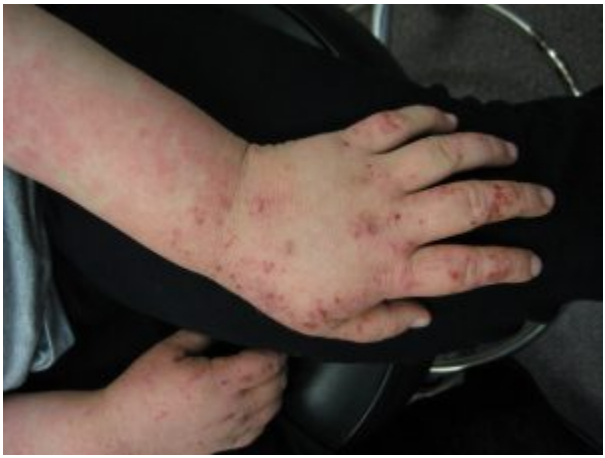

Image 3.10: Excoriations on the dorsal hands

What does it look like?

In young children, it is most common on the face, elbows and knees but can be anywhere. It tends to spare the diaper area. In older children and adults, it often goes to the bend of the elbow (antecubital fossae) or the creases behind the knee (popliteal fossae). Palmoplantar skin and eyelid dermatitis are areas often involved in older children. Patients with atopic dermatitis often have very dry skin, and their skin can still look dry even if they apply moisturizer several times a day. Often the skin around the hair follicles is a bit noticeable, usually because it's slightly raised and hypopigmented (follicular prominence). Areas affected by the eczema might become lighter or darker (post-inflammatory hypo- and hyper-pigmentation).

What makes it worse?

Certain triggers such as fragrance and harsh soaps can make the rash worse and should be avoided. Each person with atopic dermatitis are at increased risk of food allergy, but atopic dermatitis is not caused by food allergy, though certain foods might make it flare. In particular, food such as tomato products can cause worsening on the face, primarily due to contact with the food. In general, food avoidance/elimination is not recommended and should be discussed with an allergist to avoid unnecessary complications including the risk of malnutrition or anaphylaxis upon re-exposure.

Common Triggers

- Hot and/or dry weather
- Hot water and strong soaps
- Products with added fragrance, including dryer sheets
- Saltwater or chlorine left on the skin after swimming
- Rough fabrics such as wool
- Known environmental allergens such as dust mites, grass, pollens, and animal dander

How is it treated?

There is no cure for atopic dermatitis, but treatment can improve the symptoms, while maintenance therapy and a good daily skin care routine can help prevent flares.

- Daily bath with warm, not hot water and soap limited to areas such as hands, feet, axillae, and groin. Apply moisturizer immediately after the bath.
- Topical medications (see below) can be applied twice daily to affected areas until clear. Sometimes this might require using medicine on all of the skin for a short period of time. Maintenance therapy of twice weekly application is helpful to prevent flares. Oral antihistamines are not particularly effective in controlling the itch associated with atopic dermatitis. Some are sedating and can be used in extreme flares as a sleep aid, but melatonin is likely a safer alternative.

Systemic Treatments: When not responsive to topical therapy, systemic treatment might be needed.

- Phototherapy – Narrow band UVB, usually 2-3 times per week.
- Systemic immunomodulators such as methotrexate, cyclosporine, MMF, IL4/IL13 blockers (dupilumab, tralokinumab), or JAK inhibitor (upadacitinib, abrocitinib) may be necessary.

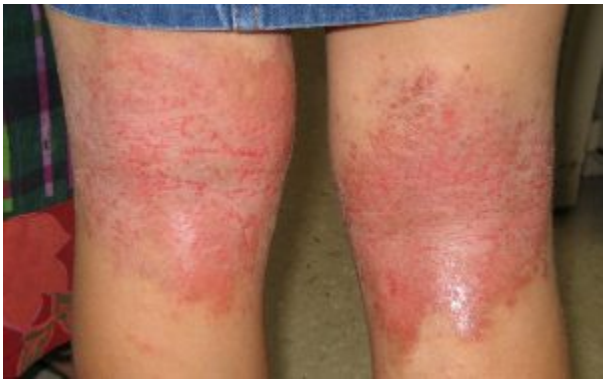

Image 3.11: Before treatment

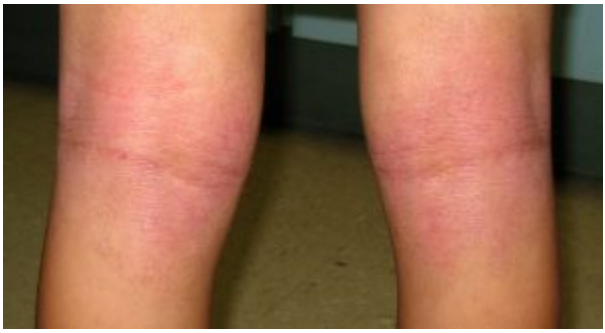

Image 3.12: After treatment

Topical Therapies	Indications
Corticosteroids	**Low potency** (hydrocortisone 0.5-2.5%, desonide) for mild eczema, face, neck and groin areas. **Mid potency** (betamethasone valerate 0.1%, mometasone 0.1%) for moderate eczema or unresponsive to low potency. Avoid long term use on large body surface areas. **High potency** (clobetasol 0.05%, betamethasone diproprionate) for thick areas of eczema unresponsive to the lower/ mid potency topical corticosteroids, palms and soles. **Scalp solutions** (fluocinolone oil or betamethasone valerate, mometasone furoate and clobetasol scalp solutions) in order of potency.
Calcineurin inhibitors	Tacrolimus ointment 0.03 or 0.1% and pimecrolimus cream 1%. Can be used on all locations including face, neck and groin, with no risk of skin atrophy. Their strength is close to a mid-potency corticosteroid. Might sting on application.
Crisaborole	Non steroid anti-inflammatory that can be used with no risk of atrophy. May be particularly helpful on thick skin such as hands and feet. Might feel hot on application.

Complications?

Complications of atopic dermatitis include loss of sleep, distractibility during the day due to itch, stress due to chronic relapsing and remitting nature of the condition, and infection. *Staphylococcus aureus* is the most common pathogen, but *Streptococcus pyogenes* can be seen as well. Secondarily infected plaques have a yellow honey-coloured crust on top and may lead to widespread worsening of the eczema. Secondary infections are usually treated with systemic antibiotics such as cephalexin. A culture with sensitivities can direct proper antibiotic therapy if there is concern for MRSA. Dilute bleach baths, using ¼ cup bleach per tub of water for a 10 min soak 2-3 times per week, might be helpful and has both antimicrobial and anti-inflammatory benefits. Eczema herpeticum is the explosive development of blisters due to herpes simplex virus (that otherwise causes cold sores) in patients with atopic dermatitis. This eruption can be quite severe and must be treated aggressively acyclovir. Eczema coxsackium, is a similar condition and morphologically can be confused with eczema herpeticum, caused by coxsackie

virus and only requires supportive therapy and treatment of the underlying eczema. Lesions on hands, feet, and on oral mucosa aids in diagnosis.

Nummular Eczema/ Nummular Dermatitis

What is it?

Nummular dermatitis is a form of eczema characterized by coin-shaped eczematous plaques that can occur as a solitary plaque or can be multiple and widespread.

What does it look like?

Nummular dermatitis is round to oval in shape and is intensely itchy. They are seen most commonly on the extremities and usually measure only 1-3 cm in diameter. They have minute papules and vesicles that are seen within the plaque. Unlike tinea corporis, which is annular in morphology, the skin changes are not accentuated at the periphery but involve the entire lesion. Early on, they may be quite inflamed with vesicles and weeping. Given the intense itching which causes frequent scratching and rubbing, they are often seen to be lichenified and have associated pigmentary change if they have been present for a long time.

How is it treated?

Treatment requires mid– to high– potency corticosteroids. In early lesions, it may be possible to clear with betamethasone valerate or mometasone furoate cream or ointment. Once lichenification has occurred, however, it is usually necessary to use a high potency corticosteroid such as clobetasol proprionate or betamethasone diproprionate. Several days of corticosteroid under occlusion may also be helpful. The occlusion is best achieved by applying the medication then covering the area with plastic wrap, which can be held in place by tape or a sock, or with a plastic band-aid. Secondary bacterial infection is common and antibacterials should be considered if the plaques are crusted.

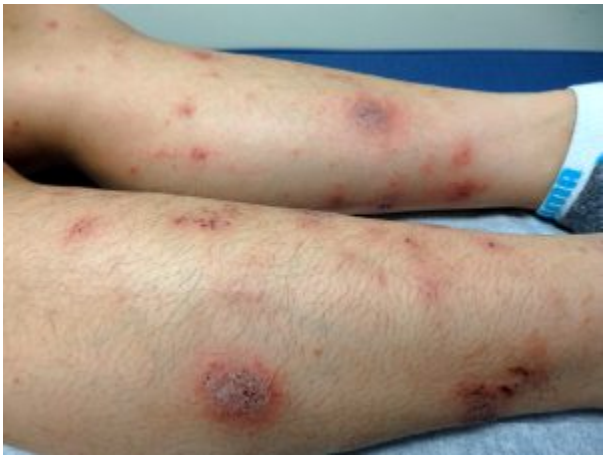

Image 3.14: Nummular eczema

Contact Dermatitis

What is it?

Contact dermatitis can be broken into two forms: allergic contact dermatitis (20% of cases) and irritant contact dermatitis (80% of cases). In both cases, contact with a particular substance leads to changes in the skin making it itchy, especially in cases when the exposure happens over a long time.

What does it look like?

The classic features of allergic contact dermatitis are redness and blister formation at the site of contact with well-defined margins clearly showing the area of exposure. In some cases, the distribution may be linear as when a liquid runs down the skin or the patient brushes past a leaf or branch to which he/she is allergic. In other cases, the distribution is found to be a plaque that corresponds to exposure. After longer term exposure, the area usually becomes more scaly and thicker plaques with lichenification can be seen.

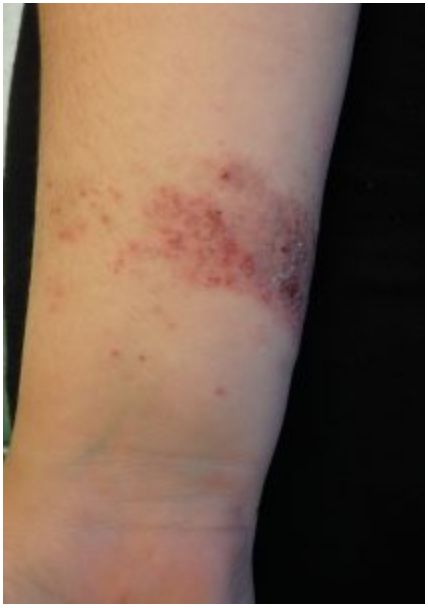

Image 3.15: Circumferential eczematous plaque on the wrist from chronic allergic contact dermatitis due to a bangle

What causes it?

Allergic contact dermatitis is an immune-medicated reaction that occurs after sensitization. The first episode might take around 1 to 2 weeks for a reaction to develop. With re-exposure this can occur within few days. Chronic contact dermatitis can last for weeks due to frequent contact

with the allergen. There are numerous allergens that can casue allergic contact dermatitis. Common encountered allergens include:

- Nickel – Found in many earrings, necklaces, bracelets, watches, cell phones and snaps/buckles of pants.
- Rubber – Rubber contains many substances such as latex and rubber accelerators used in processing. This can be seen in patients with allergy to the rubber in shoes.
- Plants – (rhus family) poison tree, poison oak, poison ivy, and mango (the underside of the skin has a compound similar to poison ivy).
- Paraphenylenediamine- found in hair dyes and black henna.
- Methylchloroisothiazolinone (MCI)/methylisothiazolinone (MI)- in diaper area found in wet wipes.
- Fragrances and dyes – including perfume, hair dye, and fragrance/color added to shampoo/soap/cosmetics/laundry detergent and etc.
- Medications – topical antibiotics like polymyxin B, bacitracin, neomycin, gentamycin are common cause of allergic contact dermatitis.
- Adhesives- found on dressings, this should not be confused with irritant contact dermatitis from wound discharge or topical antiseptic under the dressings.
- Toilet seat- can cause both allergic and irritant contact dermatitis, the former from the paint or material used and the later usually from the antiseptics or bleach used to clean them.

Irritant Contact dermatitis: Irritant dermatitis is not caused by an allergic, immune mediated response to a substance, but by direct injury due to exposure to a toxic substance, most commonly a chemical exposure. Immediate findings include a burning sensation, redness, swelling and at times blistering and peeling. Long-term, low-level exposures can cause redness and peeling with itch and burning sensation. The most common cause of irritant contact dermatitis is frequent work with detergents and in water. Oils, organic solvents, cement, and food handling (particularly raw meat) can cause irritant contact dermatitis.

How is it diagnosed?

Diagnosis of contact dermatitis is usually by clinical appearance and history. Patch testing (application of small amounts of the allergen in question to the skin in a small chamber) can be used to demonstrate allergy and can narrow down the cause of the allergy. A standard allergy patch test usually contains numerous allergens that can be tested at the same time. The test is different than the prick test which identifies type I reactions or IgE mediated hypersensitivity compared to patch test which is a type IV delayed hypersensitivity reaction.

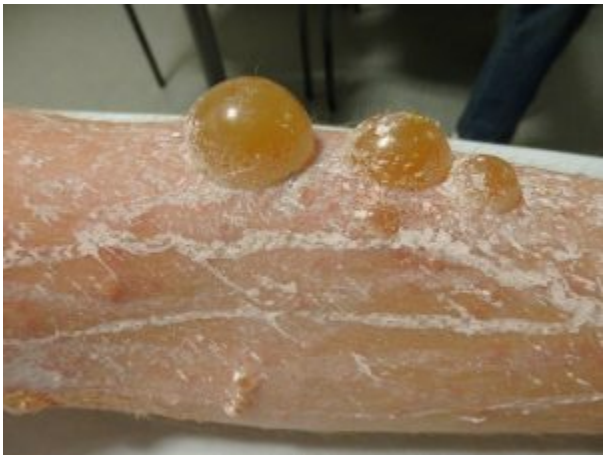

Image 3.16: Severe contact dermatitis with bullae

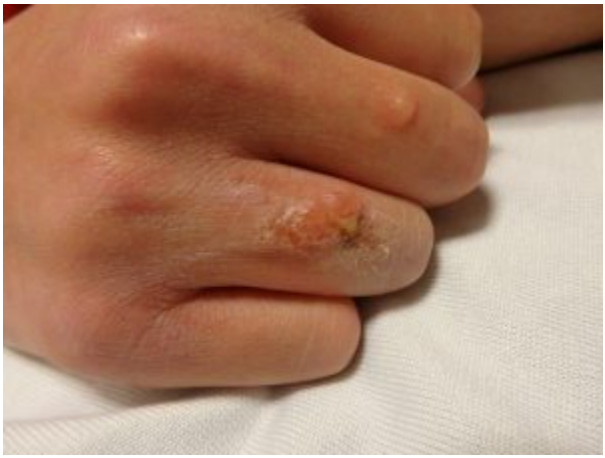

Image 3.17: Vesicles from acute contact dermatitis

How is it treated?

The appropriate treatment is avoidance of skin contact with the substance that causes the rash. In some cases, this is very difficult, and protecting the skin is the best alternative. For example, use gloves to avoid contact with known chemicals at work, or coat all nickel products with clear nail polish to prevent skin contact. Topical corticosteroids are the most common treatments for acute lesions. For short term use in acute situations, mid-potentcy corticosteroids are appropriate. For those with extensive involvement, oral steroids may be necessary and should be continued as a tapering dose over 2 weeks to prevent a return of symptoms at the end of treatment. For more chronic reactions, choice of topical steroid or steroid sparing agent mimics choices made in treatment of atopic dermatitis.

Seborrheic Dermatitis

What is it?

Seborrheic dermatitis is the cause of common dandruff. It has been attributed to *Malassezia furfur*, a yeast that is commonly found on the scalp, but the cause is not completely understood. It appears most commonly in areas where there are large numbers of sebaceous glands such as the scalp, face, and chest.

What does it look like?

In newborns it presents as cradle cap and can be quite widespread including the diaper area. It causes widespread erythematous papules with occasional scale and can be difficult to differentiate from atopic dermatitis. Involvement of the diaper area in seborrheic dermatitis is a good clue. In older children, seborrheic dermatitis can present with mild itching and flaking on the scalp. In more severe cases, erythematous scaly plaques with a yellow-greasy scale are most commonly seen in the scalp, eyebrows, and nasal ala.

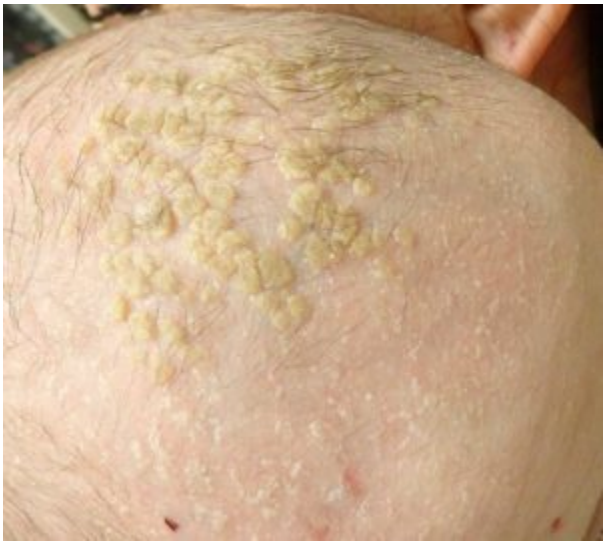

Image 3.18: SD: Yellow greasy scale

How is it treated?

Seborrheic dermatitis is treated with a combination of corticosteroids and anti-seborrheic shampoos. For facial involvement, hydrocortisone is usually sufficient to control the erythema. The combination of hydrocortisone with 2% ketoconazole cream is a common prescription that is safe for all ages. For eyebrows and scalp, a liquid form of corticosteroid, such as fluocinolone oil or betamethasone scalp solution, is often easier to apply. Anti-dandruff shampoos are also useful and may be anti-inflammatory, keratolytic or have anti-yeast properties. All anti-dandruff shampoos must be left on for 5-10 minutes before rinsing. For patients with thick scale,

application of an oil such as mineral oil to the area overnight (with a shower cap to increase the effect of the oil and prevent staining sheets) with a shower in the morning is effective in removing the thick scale.

Asteatotic Dermatitis

What is it?

It is a type of eczema that appears as a cracked dry riverbed from excessive dryness. Exposure to hot water, using harsh soaps or malnutrition can result in astetotic eczema. It is thought to be secondary to loss of epidermal lipids and natural moisturizers.

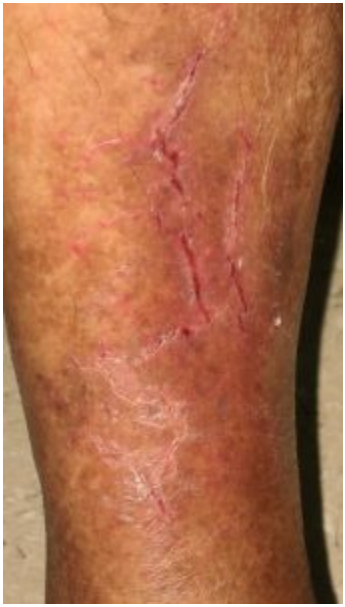

Image 3.19: Fissures and dermatitis from asteotosis

What does it look like?

Dry skin that progress to form superficial cracks or fissures on a background of faint erythema. Often seen on the lower extremities, where scales are thick or on the upper back in patients exposed to hot showers.

How is it treated?

Topical steroids, preferably in an ointment base, rapidly clears the skin. Calcineurin inhibitors can also be used. Aggravating factors like hot showers, harsh soaps and bath scrubs should be avoided. For prevention, multiple daily applications of a thick moisturizer, preferably petrolatum jelly or moisturizers that contain ceramides and humectants.

Juvenile Plantar Dermatosis

What is it?

Juvenile Plantar Dermatosis is not an uncommon problem on the feet of children that is more common in boys than girls and is sometimes called “sweaty sock syndrome”.

What does it look like?

Shiny, dry skin that often cracks. It is most often on the bottom of the foot especially on the ball of the foot and the bottom of the big toe.

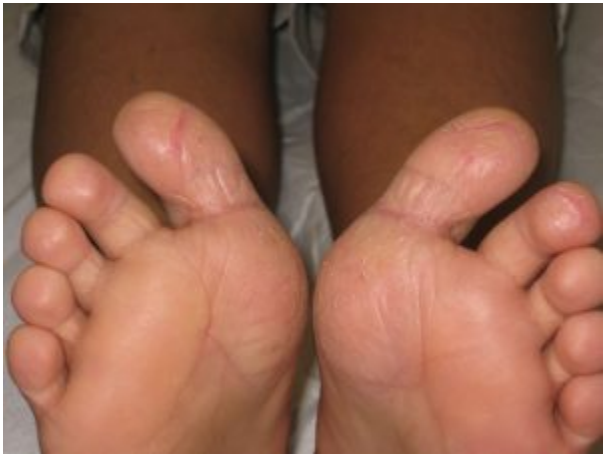

Image 3.20: JPD: Shiny skin with fissures and peeling

What causes it?

Juvenile plantar dermatosis is more commonly seen in children who have atopic dermatitis and/or who have particularly sweaty feet. It is worsened by prolonged contact with damp socks or to increased friction.

How is it treated?

Frequent sock changes, frequent moisturizer application, topical corticosteroids, avoidance of shoes that cause a lot of friction such as plastic or rubber shoes worn without socks, and taking rest days to allow the skin to heal.

Id Reaction (Autosensitization)

What is it?

A diffuse secondary eczema occurring due to a localized severe skin reaction such as caused by contact dermatitis, or tinea corporis.

What does it look like?

A widespread, symmetrical eczematous papules and plaques that occurs days to weeks following a localized dermatitis.

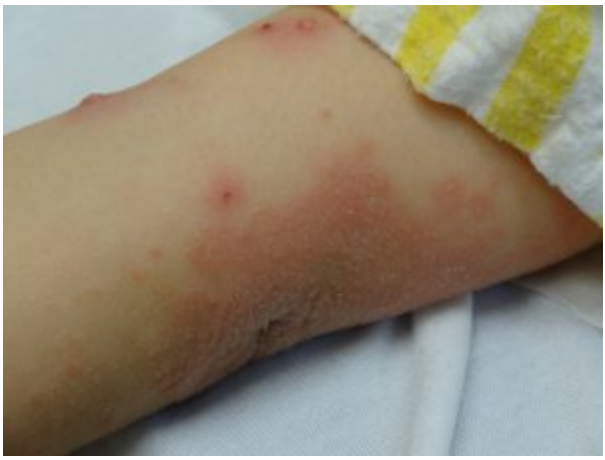

Image 3.21: ID reaction secondary to inflamed molluscum

How is it treated?

Searching for and treating the primary skin problem is the first component of therapy. For mild to moderate involvement, topical corticosteroids can be sufficient and help control symptoms. If severe, a short course of prednisone or prednisolone for 1-2 weeks might be necessary.

Diaper Dermatitis

What is it?

Diaper dermatitis is dermatitis occurring in the diaper area. Most commonly this is due to irritation from urine and stool in the moist environment of the diaper, however, there is a longer and important differential diagnosis. The location and morphology of the lesions can help indicate an underlying cause.

Cause	Clinical clue
Irritant contact dermatitis	Most common. The eruption is erythematous often spares the folds.
Allergic contact dermatitis	Confined to exposure area, similar to irritant contact dermatitis. Napkin wipes are potential cause.
Candida infection	Erythematous plaques with satellite papules (pustules) favour the folds.
Streptococcal infection	Bright red, well-demarcated plaques, that can be painful and can be associated with bad odour. Perianal region most often involved.
Psoriasis	Sharply demarcated plaques with scalloped edge. Associated psoriasis in other location including scalp, nails and skin.
Seborrheic dermatitis	Usually bright red-orange and can resemble psoriasis. Often seen in conjunction with scalp involvement.
Atopic dermatitis	Not commonly seen except in setting of erythroderma. Otherwise diaper area is usually spared.
Langerhans cell histiocytosis	Rare. Associated involvement of other locations like scalp and body. They are resistant to treatment. Needs biopsy.
Jacquet's dermatitis	An erosive dermatitis, with punched out erosions and ulcers. It is a result of severe irritant contact dermatitis.

How is it treated?

Understanding the underlying cause can help direct management of diaper dermatitis. For irritant contact dermatitis, barrier protection using zinc-based creams and petrolatum jelly is useful for prevention. They should be applied generously as if icing a cake and should not be completely removed with diaper changes as wiping them off vigorously can damage the underlying skin. If the skin is particularly inflamed, 1% hydrocortisone is useful. Secondary candida infections can occur and can be treated with clotrimazole or other anti-yeast preparations. It should be emphasized that only low potency cortisones should be used under the diaper due to risk of skin thinning with and stronger cortisones. Wipes could also be a source of irritation. Cleaning with water on a soft cloth or mineral oil on a cotton ball are alternatives.

Quiz

1. Which of the following is false about atopic dermatitis?

a. Up to 20% of children are estimated to have it
b. Patients may have higher than normal rates of asthma and allergies
c. It is often called eczema
d. All of the above are true

2. Which of the following can help treat nummular eczema?

a. Low-potency corticosteroids
b. Elimination diet
c. Antibacterials alone
d. Mometasone furoate

3. Which is a common trigger of atopic dermatitis?

a. Humid weather
b. Unfragranced products
c. Soft fabrics
d. Hot water

4. Seborrheic dermatitis has been attributed to the yeast Malassezia furfur.

a. True
b. False

5. What are classic features of acute contact dermatitis?

a. Undefined margins (area of exposure unclear)
b. Swelling
c. Blister formation
d. Cracked skin
e. None of the above

6. Which medication is used to treat asteatotic dermatitis?

a. Anabolic steroids
b. Topical steroids
c. Topical antibiotics
d. None of the above

7. What are some causes of contact dermatitis?

a. Fragrances/dyes
b. Toilet seats
c. Rubber
d. Nickel
e. All of the above

8. Irritant contact dermatitis is least commonly seen in diaper dermatitis.

a. True
b. False

9. What is false about Juvenile Plantar Dermatosis?

a. Can be caused by frequently sweaty feet
b. It consists of shiny, dry, skin that often cracks
c. It is more common in boys than girls
d. Frequent sock changes worsen it

10. How is diaper dermatitis best treated?

a. Zinc-based creams
b. Clotrimazole
c. Mid- to high- potency cortisones
d. It depends on the underlying cause

Answers: 1. D 2. D 3. D 4. A 5. C 6. B 7. E 8. B 9. D 10. D

CHAPTER 4: PAPULOSQUAMOUS DISORDERS

Psoriasis

What is it?

Psoriasis is an inflammatory disorder that causes thick, red, and scaly plaques to appear on the skin. The tendency to get psoriasis is passed in families, but there is usually a trigger such as an infection that causes it to appear for the first time. Psoriasis is more common in adults. In the pediatric population, the prevalence increases with age, being highest in teenagers. Rarely psoriasis can start at birth or in the infancy period.

There are many systemic disorders linked to psoriasis, including arthritis, obesity, and metabolic syndrome. Psoriasis has a major impact on quality of life.

What does it look like?

The distribution of the psoriatic plaques is often symmetrical and distributed on the elbows, knees, lower back, and scalp. The plaques are erythematous to salmon in colour with a sharp demarcation. The scales in psoriasis are very thick and can become silver in colour (Micaceous scale). More than 50% of patients have pruritus but not as severe as in atopic dermatitis.

Scalp plaques are thick and can lead to tinea amiantacea, a term used when hairs clump with thick scale. The hair line is a common site of involvement. The external auditory canal and post auricular skin are often involved. An important site to examine in patients with psoriasis are the nails. This helps support the diagnosis. Nail involvement can be the solo presentation.

Nail sign	Description
Pitting	Small circular depressions on the nail plate
Onycholysis	Separation of the nail plate from the nail bed
Oil drop sign	Yellow orange discolouration under the nail plate
Subungual hyperkeratosis	Thickness under the distal nail

Koebner Phenomena

Development of psoriasis on areas of trauma, indicates that the condition is active. This also can be a feature of other cutaneous disorders like lichen planus, vitiligo and warts.

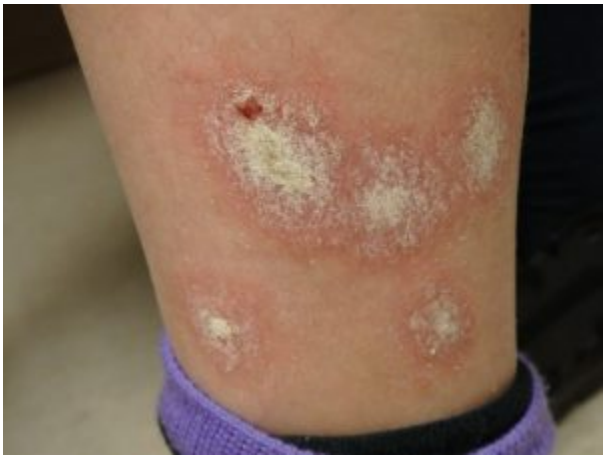

Image 4.1: Erythematous plaques with dry silvery scale

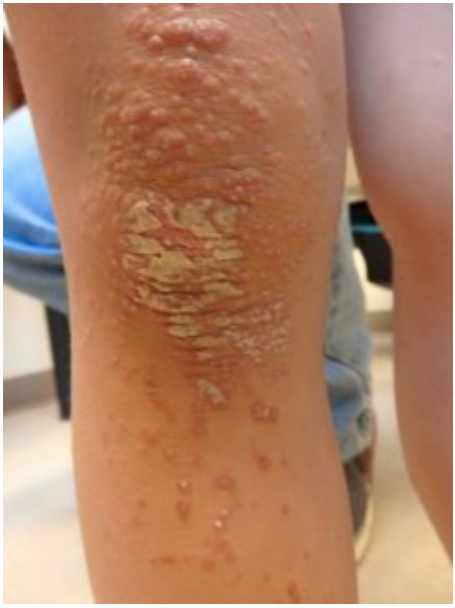

Image 4.2: Erythematous plaques with dry silvery scale

Subtypes of psoriasis:

1. Psoriasis vulgaris or chronic plaque psoriasis- see above
2. Guttate psoriasis: numerous raindrop-like psoriasis papules and plaques, often follows a streptococcal infection such as pharyngitis or perianal strep. Treatment includes both antibiotics and psoriasis therapies. Phototherapy is a good option if available.
3. Pustular psoriasis: a widespread pustular eruption with background of erythema. Can be widespread and associated with constitutional symptoms. The use of systemic corticosteroid in patients with psoriasis vulgaris or arthritis that can lead to a pustular psoriasis flare when the steroid is discontinued. Treatment with acitretin is often recommended.
4. Erythrodermic psoriasis: with wide-spread erythema > 80-90% body surface area and associated exfoliation. This can be the first presentation of psoriasis, clues to the diagnosis can be family history or nail involvement. Skin biopsy may be necessary

What causes psoriasis?

Psoriasis is a cutaneous disorder with an immune dysregulation. The high rate of epidermal proliferation triggered by the immune system causes to the thick plaques and associated scale. An increase in Th1 and Th17 cells leads to the inflammatory reaction and increased cytokines seen in psoriasis. These have been a target for new biologic therapies, which have shown great success in adult patients with psoriasis and psoriatic arthritis
What makes it worse?

In pediatric psoriasis, associated streptococcal infection of the throat or perianal skin should be evaluated. Other triggers include medications like NSAIDs, beta blockers, antimalarial, interferons and lithium. Ironically, tumour necrosis alpha inhibitors are used to treat psoriasis but can lead to a paradoxical psoriasis reaction, involving the palms, soles and scalp, when used to treat inflammatory bowel disease.

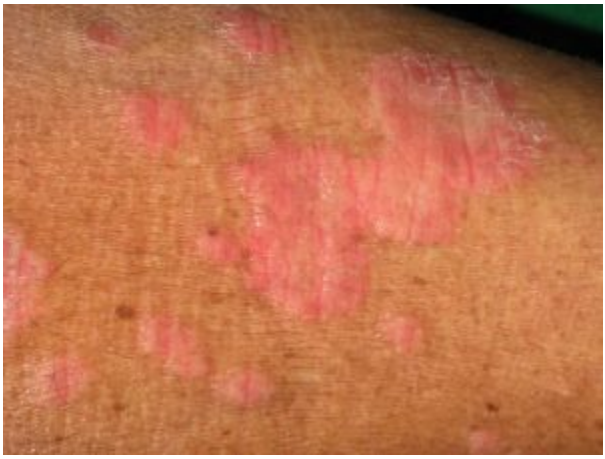

Image 4.3: Partially treated plaques without scale

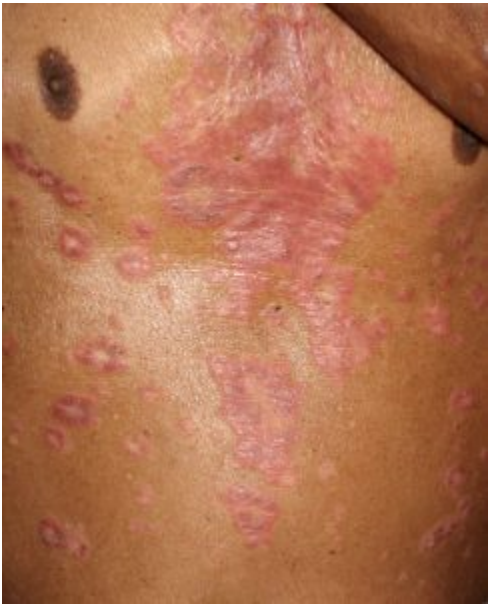

Image 4.4: Confluent plaques on the chest

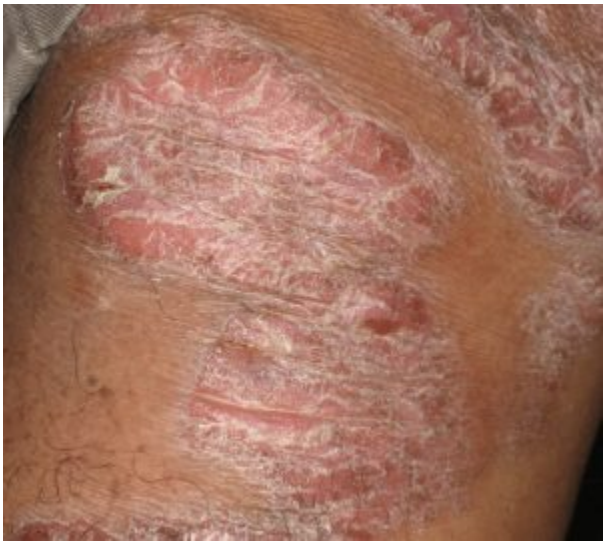

Image 4.5: Well demarcated plaques with silvery scale

How is it treated?

Treatment depends on how much of the skin is involved, what areas of skin are involved, and how thick the plaques are. Prednisone is avoided, because of the risk of developing pustular psoriasis when it is withdrawn.

Topical Therapies

- Mid-high potency corticosteroids like mometasone and clobetasol.
- Topical vitamin D derivatives (calcipotriene) ointment alone or in combination with betamethasone dipropionate.
- Betamethasone diproprionate with salicylic acid- for thick scales, the salicylic acid helps exfoliate the scale.

Face and genital region

Topical tacrolimus or pimecrolimus are very effective. Avoid using potent topical corticosteroids, because of risk of atrophy and striae formation.

Scalp involvement

Fluocinolone acetonide oil for mild scalp involvement to help life the scales. Betamethasone membrane or Clobetasol scalp lotion for thicker areas.

Phototherapy

When available, narrow band UVB (NBUVB) is the mode most often used. It is helpful for widespread involvement especially with thin plaques. Other options include broad band UVB, UVA/UVB and

Psoralen plus UVA (PUVA). Treatments are given 2-3 times a week for a duration of at least 3 months.

Systemic Therapy

Moderate to severe involvement >10% body surface area may require systemic treatments in combination with the above therapies.Common systemic agents used in psoriasis are methotrexate, cyclosporin, acitretin (vitamin A derivative) and biological therapies. The targeted biologic therapies including inhibitors of TNF alpha and IL 12/23 inhibitors. These are best directed under the care of a dermatologist when possible.

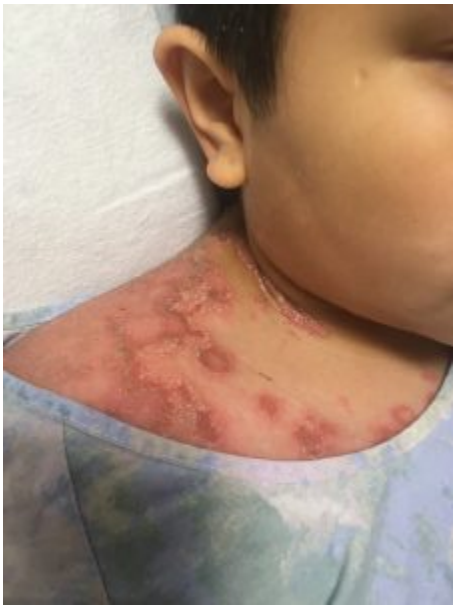

Image 4.6: Pustular psoriasis

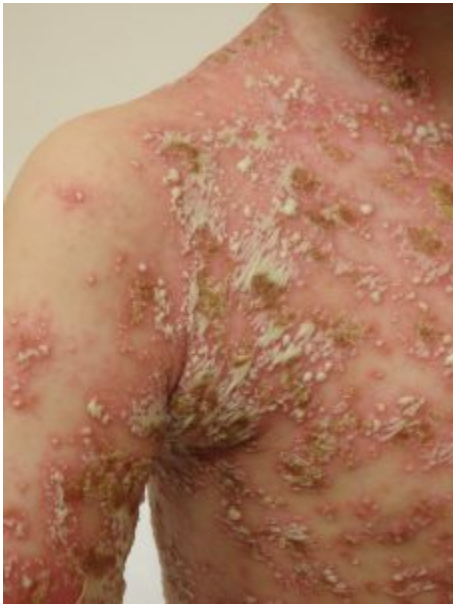

Image 4.7: Pustular psoriasis

	Atopic Dermatitis	Psoriasis
Incidence	Very common	Less common
Family history	Family history of atopy	Family history of psoriasis
Flexors vs extensor	Involves flexors (except infantile)	Extensors
Pruritis	Pruritus (must)	Pruritus very common >50%
Secondary infections	Higher risk of secondary infections	Less infections
Nail involvement	Nails are involved less often	Specific nail findings
Koebner phenomena	–	+
Joint involvement	–	+/- psoriatic arthritis

Lichen Planus

What is it?

Inflammatory skin disorder that can be chronic. It is known for the 5 P's (pruritus, papules, purple, planar and polygonal). It can involve all ages but is more common in adults. Involvement includes the skin, hair, nails and mucous membranes. The trigger is usually unknown but vaccines, medications, infections HCV and allergens have been linked.

The 5 P's of Lichen Planus

- Pruritus
- Papules
- Purple
- Planar
- Polygonal

What does it look like?

The lesions present as small to medium sized, shiny, flat-topped purple papules that can coalesce to form plaques. Secondary scale can develop and the characteristic thin white lines in the lesions are called Wickham striae. The most common sites of involvement include the ankles, wrists, lower back and genital skin. They are often pruritic and Koebner phenomenon can be seen. Mucous membranes can be involved, and the most common presentation is a lacy reticulated white line on the inner aspect of the cheeks.

How is it treated?

For mild involvement, topical corticosteroids (mid potency) are used. Calcineurin inhibitors are another option. For more widespread disease, phototherapy is usually very effective. Prednisone can be used for short periods. Other systemic agents, including acitretin, methotrexate, cyclosporine, griseofulvin and metronidazole have been trialed in small studies.

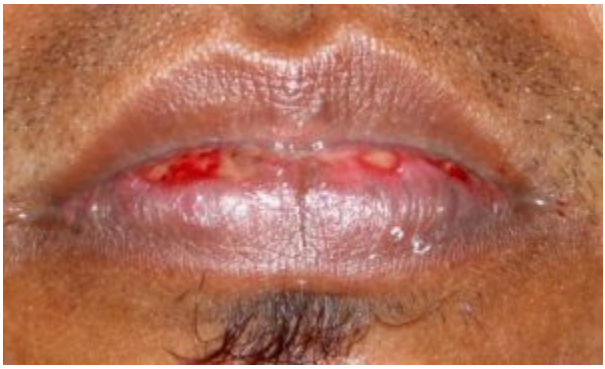

Image 4.8: Oral erosions seen in erosive lichen planus

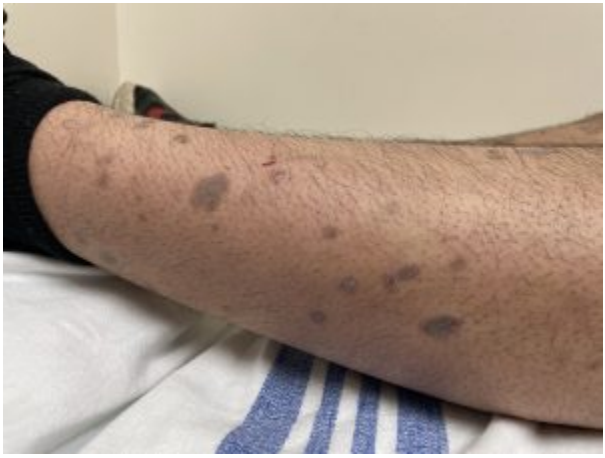

Image 4.9: Violaceous papules and plaques on lower legs in lichen planus

Pityriasis Rubra Pilaris

What is it?

PRP is an uncommon chronic inflammatory disorder that can be seen in both adults and pediatric patients. It can mimic psoriasis but has a distinct morphology with orange-red follicular papules that join together (coalesce) into large plaques. The cause is unknown.

What does it look like?

Small follicular papules that coalesce to form disseminated yellowish-pink scaly plaques with characteristic islands of sparing. The palms and soles develop yellowish thick waxy scales that can fissure.

How is it treated?

Similar to psoriasis, except for phototherapy which can flare the condition. Most cases resolve in 2-3 years, but systemic therapy might be required to control symptoms. Acitretin is one of the most effective therapies.

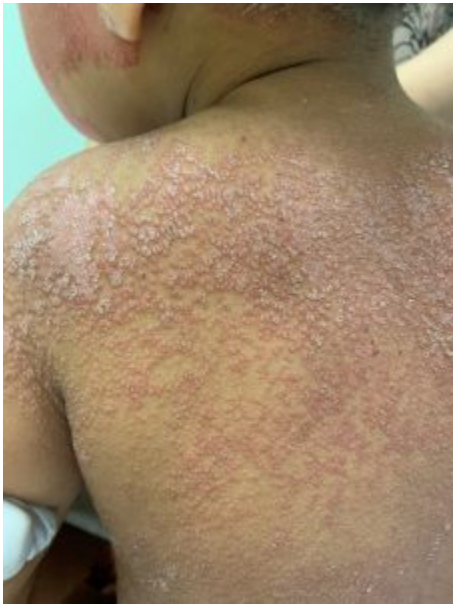

Image 4.10: PRP: Erythematous follicular papules coalescing into plaques with scale and islands of sparing

Lichen Striatus

What is it?

Lichen striatus is a transient linear rash that is seen in school age children. The cause is unknown, but it is seen more commonly in girls than boys.

What does it look like?

Lichen striatus presents with a linear band of erythematous papules with slight scale. It is commonly seen presenting in a stripe down and arm or leg, but can be seen on the face or trunk. Over time, the lesions fade and often leave hypo- or hyper-pigmentation that resolves slowly over months.

How is it treated?

No treatment is necessary, and families can be reassured. For some children, there is associated pruritus and mid-potency topical steroids might be helpful.

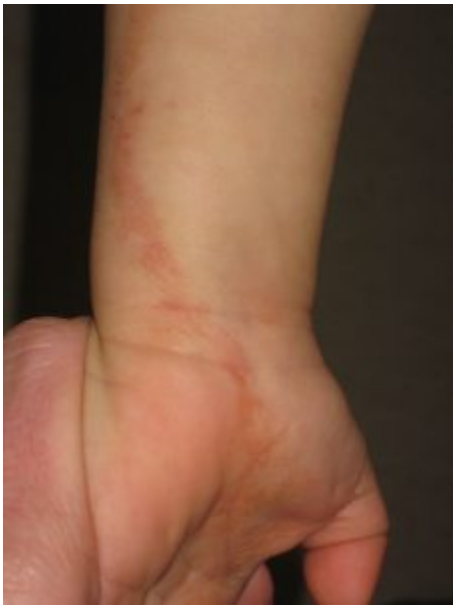

Image 4.11: Linear array of erythematous papules with scale in lichen striatus

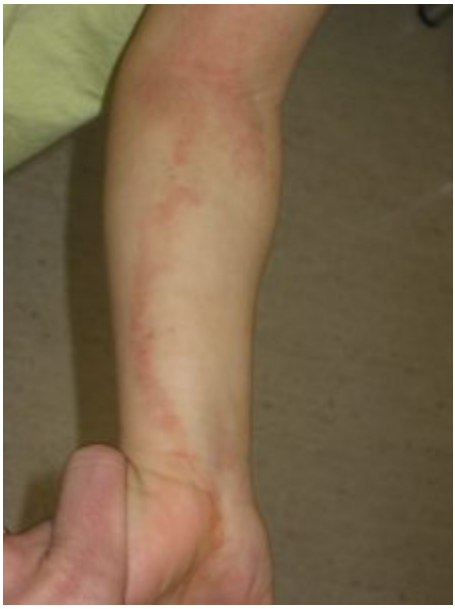

Image 4.12: Linear array of erythematous papules with scale in lichen striatus

Pityriasis Lichenoides (PLC/PLEVA)

What is it?

Pityriasis lichenoides is an inflammatory skin reaction that can be seen in either chronic (Pityriasis lichenoides chronica/PLC) or acute (Pityriasis lichenoides et varioliform acuta/PLEVA) forms. It is seen most commonly in teens and young adults but can occur in younger children. The cause is unknown, but cases are known to occur after viral infections.

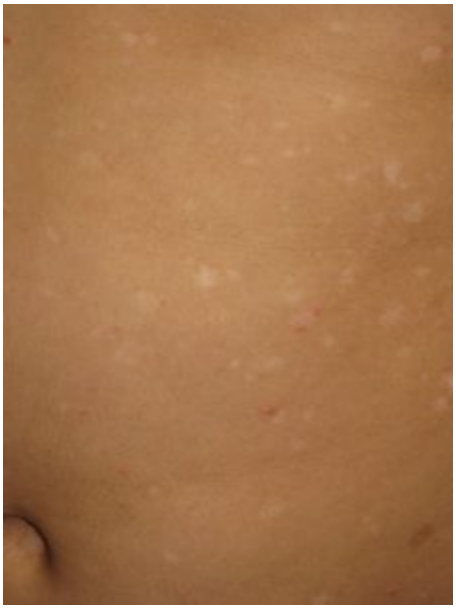

Image 4.13: PLC: Small erythematous papules with subtle scale and hypopigmented papules

What does it look like?

PLC presents with small erythematous papules with slight scale scattered widely over the body. The trunk is most commonly affected, and the papules often resolve leaving post-inflammatory hypo- or hyper- pigmentation. Lesions in different stages are often visible. Symptoms are usually minimal. PLEVA presents with larger inflammatory papules which also have central scale, but may have pus or blood within the center. The lesions are painful, can ulcerate, and may be associated with the presence of systemic symptoms.

How is it treated?

Topical steroids can be utilized to minimize symptoms, but do not generally clear the problem. When available, phototherapy is first line treatment. If not available, ambient sun with care to avoid sunburn is an option. Oral antibiotics such as erythromycin or tetracyclines can be trialed. Patients should be monitored with regular follow-up due to the rare occurrence of

transformation to cutaneous T cell lymphoma. Acitretin, methotrexate, cyclosporine, griseofulvin and metronidazole have been trialed in small studies.

Pitryriasis Rosea

Pitryriasis rosea is an acute, benign eruption that is self-limiting. Peak incidence during adolescence and during spring or fall seasons. It has been connected to Human Herpes Virus 7 & 6 reactivation, with eruption resolves after 6-8 weeks (see viral exanthems as well).

What does it look like?

The so-called herald patch is the first manifestation of the eruption. It appears on the trunk, upper arm, neck or thigh, is several centimeters in diameter, and presents as an erythematous plaque often with a collarette of scale. Subsequently, after 1-2 weeks multiple 0.5 to 2 cm, oval to oblong, red-tan papules with a fine scale. They are characteristically arranged parallel to skin tension lines (Christmas-tree pattern). Mild prodromal symptoms can occur. Plaques are discrete, have peripheral scale, and are generally thinner than in psoriasis.

How is it treated?

No treatment required. Pruritis can be treated with low to mid potency topical corticosteroids.

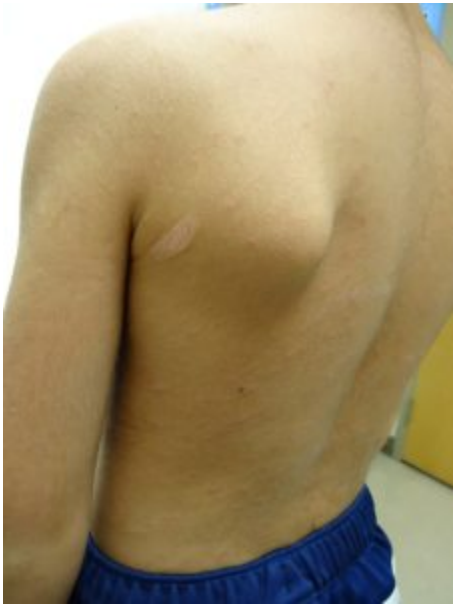

Image 4.14: Herald patch near the axilla and widespread exanthem due to pityriasis rosea

Quiz

1. What is false about psoriasis?

a. It is more common in adults
b. Often starts at birth/infancy
c. It is an inflammatory disorder
d. Has a pustular variant

2. What are some triggers of psoriasis?

a. NSAIDs
b. Beta blockers
c. Lithium
d. Interferons
e. All of the above

3. Which of the following is not treatment for lichen planus?

a. Phototherapy
b. Topical corticosteroids
c. Prednisone
d. Calcineurin inhibitors
e. All of the above treatments

4. What are the white net-like lines seen on skin lesions and buccal mucosa in lichen planus called?

a. Koebner's lines
b. Wickham striae
c. Blashko's lines
d. Oral aphthae

5. Lichen striatus must be treated because it can leave atropic scars.

a. True
b. False

6. True or False: The cause of pityriasis lichenoides is unknown.

a. True
b. False

7. Where does pityriasis rosea not appear?

a. Trunk
b. Neck
c. Upper arm
d. Thigh
e. It appears on all of the above

Answers: 1. B 2. E 3. E 4. B 5. B 6. A 7. E

CHAPTER 5: ACNEIFORM DISORDERS

Acne Vulgaris

What is acne?

Acne vulgaris is one of the most common skin conditions worldwide. It is most common in teenagers but can be seen in preteens and adults as well. Most people can recognize acne by its blackheads, whiteheads, and pimples. It is most often seen on the face, back, and chest.

What causes it?

There are several factors that contribute to the development of acne.

1. Increased sebum production in response to androgens.
2. Follicular hyperkeratosis that blocks the opening of hair follicles and causes comedones (whiteheads – or closed comedones, and blackheads – or open comedones).
3. *Cutibacterium acnes* (*C. acnes*), formerly known as *Proprionibacterium acnes*, proliferation around the hair follicle.
4. Inflammation, which causes pustules and nodules. These inflammatory lesions may lead to scarring.

There are many myths or misconceptions about acne. It is helpful to reassure patients that:

- Acne is not caused by dirty skin. In fact, washing the face too often can make acne worse because of irritation.
- Diet does not have a large role in acne formation. There is some evidence that high glycemic diets may worsen acne, but this is not the underlying cause. There are also other health benefits to following a lower glycemic diet.
- Stress does not cause acne, but it can make it flare.

What does it look like?

Mild acne presents with comedones primarily on the cheeks and forehead. These can be open (blackheads) or closed (whiteheads) and have little inflammation associated with them. In moderate acne, there are inflammatory papules and pustules, and sometimes nodules, which are deeper than the comedones and may involve the back and chest. In more severe acne cystic lesions appear, and scarring results as these heal.

How is it treated?

Treatment of acne requires long-term therapy.

Mild acne: Topical therapy is often sufficient. These may include over-the-counter salicylic acid or benzoyl peroxide washes, creams, and wipes. For primarily comedonal acne, topical retinoids work well. For small inflammatory lesions, benzoyl peroxide, topical antibiotics or combination products are more effective. An alternate agent is azelaic acid.

Moderate acne generally requires oral therapy, often in combination with topicals. For papular/ pustular and nodular acne oral antibiotics such as doxycycline taken for several months are often recommended. In female patients a combined oral contraceptive pill may be a good option. These may be used in combination with the topical products described above.

For acne that is severe, scarring or unresponsive to the above treatments, isotretinoin is the first line therapy. Isotretinoin has the best chance of "curing" acne, though some patients do need more than one course. Due to the side effect profile, patients taking isotretinoin must be carefully counselled and monitored. Lab monitoring includes liver function, lipids and pregnancy tests.

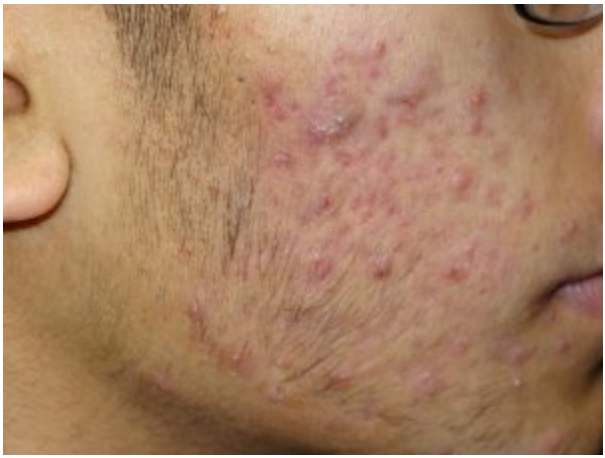

Image 5.1: Inflammatory papules and pustules

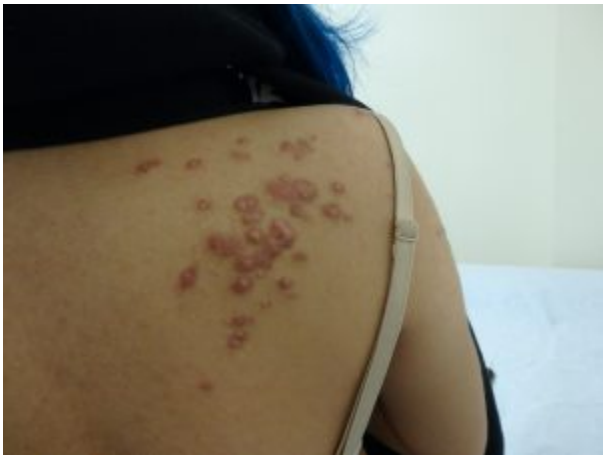

Image 5.2: Keloid scars following mild acne

Acne Variants: Conglobata, Fulminans, and Medication-induced

Acne Conglobata

Acne conglobata is a severe form of nodulocystic acne without systemic symptoms. Some patients may develop acne conglobate as part of the "Follicular Occlusion Tetrad" along with dissecting cellulitis of scalp, hidradenitis suppurativa and pilonidal sinus. Isotretinoin is used to treat acne conglobata and may require concomitant oral corticosteroids, especially at the beginning of the course when the acne can flare as isotretinoin is started.

Acne Fulminans

Acne fulminans is a severe form of acne characterized by the abrupt onset of nodular and cystic acne lesions with systemic symptoms including fever, arthralgia and myalgia, osteolytic bone lesions and hepatosplenomegaly. This requires prompt treatment with oral corticosteroids, followed by isotretinoin, initiated at a low dose and then increased.

Medication-induced Acne

Common medications known to cause acne include anabolic steroids, lithium, corticosteroids, unopposed progestin, and phenytoin. If the causative medication cannot be discontinued, then the acne can be treated as above. In steroid-induced acne, the lesions are quite monomorphous. In some instances, pityrosporum yeast is implicated and topical antifungals are helpful.

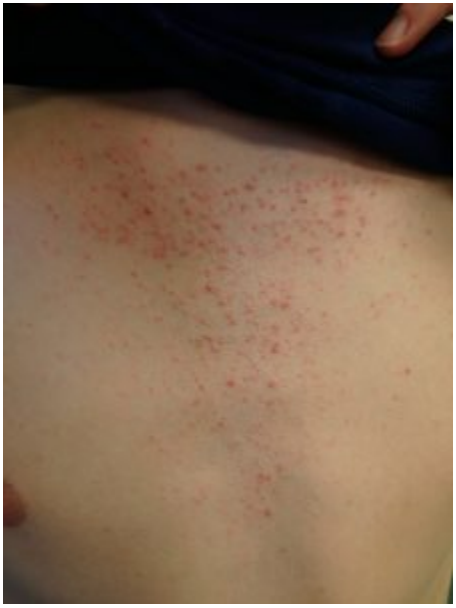

Image 5.3: Steroid-induced acne

Periorificial Dermatitis

What is it?

Periorificial dermatitis is a common skin eruption on the face. It tends to occur most commonly in adult women, but also affects children of all ages. The exact cause of periorifical dermatitis is not known. In some cases, it may be triggered by the use of corticosteroids. These may be applied directly to the face, from unintentional contact after using the hands to apply a TCS elsewhere, or due to use of nebulized steroid.

What does it look like?

There are groups of small, pink to red papules and pinpoint pustules in the perioral, perinasal and/or periocular area. Sometimes the background skin is red or may have scale. Some patients report a burning sensation.

In contrast to rosacea, which can look similar, periorificial dermatitis spares the cheeks and forehead, and does not involve background telangiectasia or flushing.

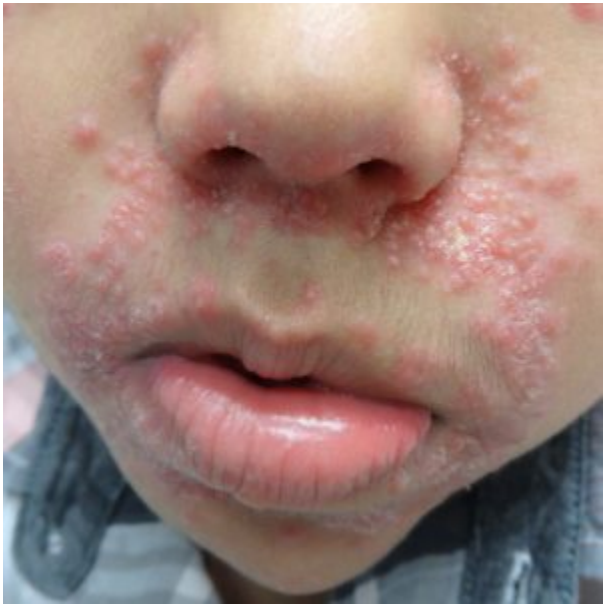

Image 5.4: Small perinasal inflammatory papules without comedones

How is it treated?

The treatment is very similar to that of rosacea. Topical treatments include calcineurin inhibitors, topical antibiotics, and/or azelaic acid. Oral tetracycline or macrolide antibiotics can be used for refractory cases for a 4-8 week course. Any topical corticosteroids being used on the

face should be discontinued. If potent TCS are being used then these should be tapered slowly and replaced with a less potent agent to prevent a flare.

Hidradenitis Suppurativa

What is it?

Hidradenitis suppurativa (HS) is a chronic inflammatory condition that leads to development of comedones, abscesses, and scarring primarily in the axilla and groin. The cause is unknown, but it is increased in frequency in patients with inflammatory bowel disease, obesity, and other conditions characterized by occlusion of the hair follicles. The disease has a significant negative impact on mental wellbeing.

What does it look like?

HS presents with abscesses in the axilla and groin area. These are often quite painful and might drain pus. Comedones and sinus tracts as well as scars are visible in the affected areas.

How is it treated?

HS is a chronic condition and is difficult to treat. Weight loss and smoking cessation are recommended when relevant. For early disease, simple measures such as use of antibacterial washes and unscented antiperspirant, wearing loose fitting clothing, decreasing friction, and pain control may be sufficient. In more extensive disease, systemic therapies including antibiotics, retinoids, anti-inflammatories (methotrexate/cyclosporin) or biologics (esp adalimumab) are necessary. Individual lesions can be treated with intralesional triamcinolone or drainage. Some patients find that laser hair removal is beneficial. Surgical removal of affected skin is sometimes required.

Folliculitis

What is it?

Folliculitis refers to inflammation of the hair follicle. This can be caused by both infectious and non-infectious causes. Bacterial folliculitis from S. Aureus is the most common cause. Dermatophytes and Malassezia yeast can also cause folliculitis. Other infectious causes are rare. Non-infectious etiologies include irritant folliculitis, related usually to occlusive products or friction, culture-negative folliculitis, and more rarely can be drug-induced or eosinophilic folliculitis.

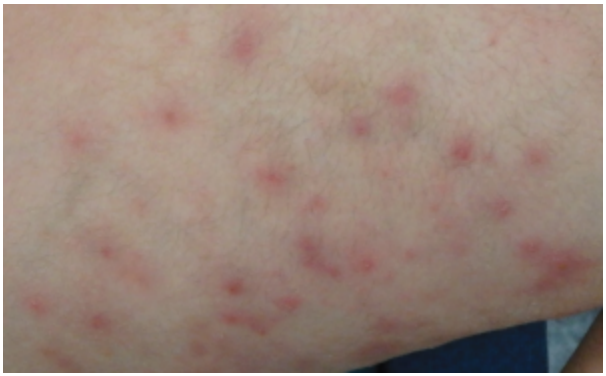

Image 5.5: Folliculocentric inflammatory papules and pustules in fungal folliculitis

What does it look like?

There are follicular-based pustules with an erythematous rim. It is most common on the head and neck, especially the scalp and beard, as well as the upper trunk, buttocks, thighs, axillae and groin. Hot tub folliculitis refers to infection with *Pseudomonas aeruginosa* and occurs from using a hot tub that has not been properly maintained. It occurs primarily on the back and legs that have been exposed to the water. It is often pruritic. Fungal folliculitis is often called Majocchi granuloma. This presents as follicular pustules, papules and nodules. It is commonly seen following improper treatment of tinea corporis or cruris with a topical corticosteroid.

How is it treated?

Folliculitis can be treated by cleansing of the area with an antibacterial soap several times per day and the use of a benzoyl peroxide wash. Topical antibiotics can be added if deeper lesions or more wide-spread area of involvement is noted. If severe, or systemic symptoms are present oral antibiotics may be necessary. A swab should be taken prior to initiating oral treatment. Topical antifungals are helpful in folliculitis caused by yeast, but fungal folliculitis requires oral therapy.

Quiz

1. Patients with mild, inflammatory acne can be treated with which of the following medications?

a. Benzoyl peroxide
b. Salicylic acid
c. Tretinoin
d. All of the above

2. Female patients with severe acne being considered for Isotretinoin treatment must have which of the following screening blood tests performed?

a. CBC, Chem 7, Hepatitis B and C
b. Fasting glucose, Urinalysis
c. Fasting triglycerides, LFTs, pregnancy test
d. CBC, Fasting glucose, LFT's, pregnancy test

3. What micro-organism contributes to the development of acne vulgaris?

a. Staphylococcus aureus
b. HSV1
c. Cutibacterium acnes
d. Malasezzia furfur

4. Successful treatment of acne during teenage years means that the individual will not develop acne as an adult.

a. True
b. False

5. Which of the following are systemic symptoms that may be seen in acne fulminans?

a. Fever
b. Arthralgia and myalgia
c. Osteolytic bone lesions
d. Hepatosplenomegaly
e. All of the above

6. Which of the following are not used to treat periorificial dermatitis?

a. Topical corticosteroids
b. Topical antibiotics
c. Topical calcineurin inhibitors
d. Azelaic acid

7. Which micro-organism is the most common cause of Hot tub folliculitis?

a. Staphylococcus aureus
b. Pseudomonas aeruginosa
c. Cutibacterium acnes
d. Group A streptococcus

8. What is a possible outcome of treating a fungal infection with topical steroids?

a. Development of resistance fungi
b. Resolution of the folliculitis
c. Majocchi granuloma
d. Fungemia

9. Which body areas is acne vulgaris commonly seen?

a. Face only
b. Thighs and buttocks
c. Face, chest, back and thighs
d. Face, chest and back

Answers: 1. D. 2. C, 3. C, 4. B, 5. E, 6. A, 7. B, 8. C, 9. D

CHAPTER 6: INFECTIONS AND INFESTATIONS

Bacterial Infections: Impetigo

Impetigo is a common superficial bacterial infection of the skin, which is most often seen in children and is contagious. There are bullous and non-bullous forms.

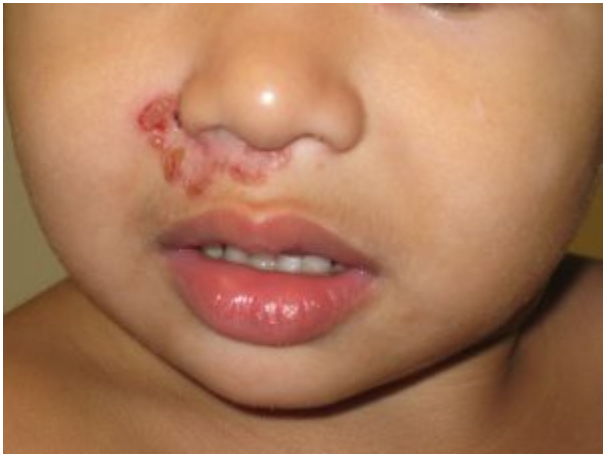

Image 6.1: Impetigo: Crusted erythematous plaques

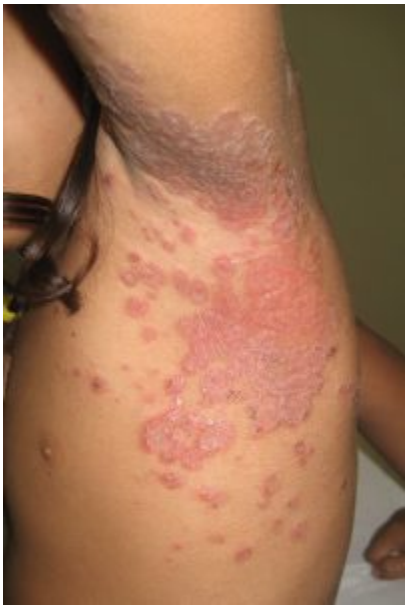

Image 6.2: Impetigo: Crusted papules and plaques in the axilla

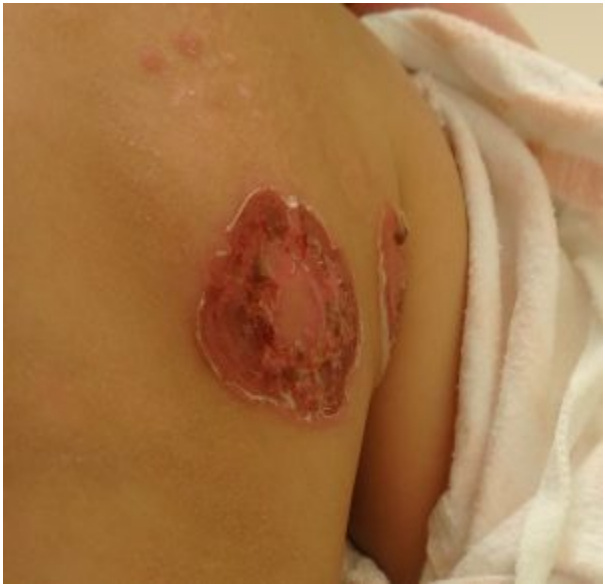

Image 6.3: Large plaque of impetigo with erosion and rim of desquamation

What causes it?

Impetigo is caused by several bacteria, most commonly *Staphylococcus aureus* or *Streptococcus pyogenes*. The infection often starts where there is a break in the skin such as a bite, scrape, cut or area affected by eczema; however, once it starts, it can spread to adjacent areas with intact skin. It is contagious and can be spread from person to person quite easily.

What does it look like?

Impetigo is characterized by a honey-coloured crust on the surface of the skin. The areas are usually red and open underneath and covered with the yellowish crust on the surface. The lesions can develop on any part of the body but are most common on exposed surfaces such as the arms, legs, and face. Sometimes impetigo develops with blisters. These rupture and leave a ring (collarette) of scale at the border. This form of impetigo is called bullous impetigo and is almost always caused by *S. aureus*. It can be commonly seen in the diaper area as well as on exposed surfaces as in non-bullous impetigo.

How is it diagnosed?

Lesions with classic honey-crusting can often be diagnosed clinically. Swabs for culture and sensitivity can be performed, particularly if there are risk factors for methicillin-resistant S. aureus (MRSA). If impetigo is suspected, treatment should not be delayed while waiting for results to become available.

How is it treated?

Impetigo is generally treated with oral antibiotics (cephalexin, erythromycin, dicloxacillin, or clindamycin). Soaks with warm soapy water or in bath water with 1/4 cup of bleach added to the tub can cut down on spreading and help to heal the lesions (see Appendix for instructions on bleach baths). Topical antibiotics such as bacitracin, polymyxin, erythromycin, neomycin, mupirocin or fusidic acid are helpful for very localized disease, but are usually not sufficient for more extensive disease. Treatment continues for 7-10 days.

MSSA / *S. pyogenes*

Cephalexin:

- Adults – 250-500 mg PO QID
- Pediatrics – 15mg/kg/dose PO TID to QID (max 4g/day)

Some patients carry *S. aureus* in the nose or perianal area and develop recurrent infections on their skin as a result. In these cases, treatment of the nostrils and perianal area with mupirocin ointment twice a day for 2 weeks along with use of antibacterial soaps and general house cleaning can cut down on recurrences.

Are there any complications?

Yes, if the impetigo is caused by *S. pyogenes* the patient is at risk of developing either scarlet fever or post-streptococcal glomerulonephritis. Unfortunately, neither of these conditions seems to be prevented by appropriate antibiotic therapy for the impetigo. Today, more patients are developing skin infections caused by MRSA. Treatment is frequently with clindamycin, trimethoprim-sulfamethoxazole (Septra), or doxycyline and can be guided by susceptibilities obtained from swabs.

MRSA

Clindamycin:

- Adults: 150-450 mg PO q6h.
- Pediatrics: 30-40 mg/kg/day PO div q6-8h

Septra:

- Adults: 160mg TMP/800mg SMX/dose PO q6h
- Pediatrics: 4-6mg TMP/20-30mg SMX/kg/dose PO q12h

Bacterial Infections: Cellulitis

Cellulitis is a common bacterial infection affecting the deeper layers of the skin and subcutaneous tissue.

What causes it?

Similar to impetigo, cellulitis is usually caused by *Staphylococcus aureus* and *Streptococcus pyogenes*. However, the infection occurs deeper than impetigo which is a superficial infection. As with impetigo, it usually starts at sites of trauma or where the skin is broken down (e.g. due to fungal infection).

What does it look like?

Cellulitis is characterized by redness, swelling, warmth and pain. It can occur anywhere, but the extremities are the most common site and it is nearly always unilateral. The area of redness is usually poorly defined and expands while the disease is active. There may be systemic symptoms such as fever/chills and malaise; white blood cells are often elevated.

How is it treated?

Oral antibiotics targeting *S. aureus* and *S. pyogenes* are the treatment of choice (cephalexin, erythromycin, cloxacillin, etc.). If patients have systemic symptoms or there is concern about sepsis, IV antibiotics may be necessary. If MRSA is a concern, appropriate antibiotics should be utilized as above.

Bacterial Infections: Other Skin Conditions Caused by S. Aureus and S. Pyogenes

In addition to cellulitis and impetigo, S. *aureus* and S. *pyogenes* can cause a variety of other skin conditions. Which skin manifestation is seen generally depends on the depth at which bacterial infection occurs, or – in the case of staphylococcal scalded skin syndrome – if a toxin is present in the blood.

Erysipelas is a bacterial infection typically caused by S. pyogenes. It affects lymphatics within the dermis (i.e. deeper than the level of impetigo but more superficial than cellulitis). It is typically seen on the face or lower extremity. It presents as a very well defined, bright red, tender plaque.

Ecthyma is a deeper form of impetigo caused by S. *pyogenes*. It often starts superficially but extends into the deeper layers of the skin and can result in ulceration and scarring. It commonly begins as small fluid-filled vesicles, often seen on the lower extremities and buttocks, which rupture and form shallow crusted ulcers.

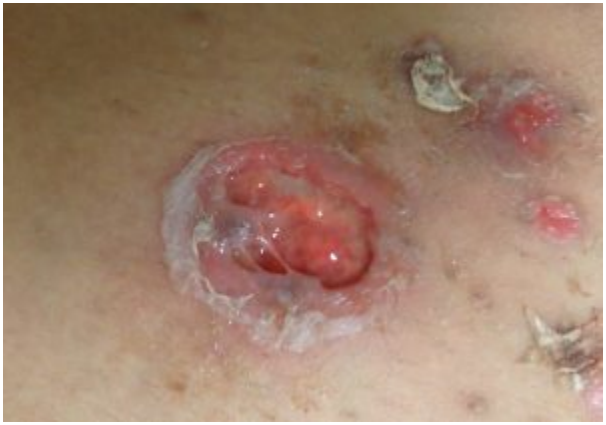

Image 6.4: Ecthyma: Ulceration at site of bacterial infection following varicella

Furuncles ("boils") and **abscesses** are walled-off collections of pus, usually caused by S. *aureus*. Whereas an abscess can occur anywhere in the body, a furuncle is, by definition, associated with a hair follicle. They are most commonly seen on the neck, axilla, and buttock but may appear anywhere. Most furuncles eventually come to the surface and rupture. For early/small furuncles, treatment with warm compresses and oral antibiotics may be sufficient. If lesions appear deep and may not rupture spontaneously, incision with drainage and packing with iodoform or Vaseline gauze are required for clearance. Without the packing, the wound can heal from the top leaving an empty space inside that can become re-infected.

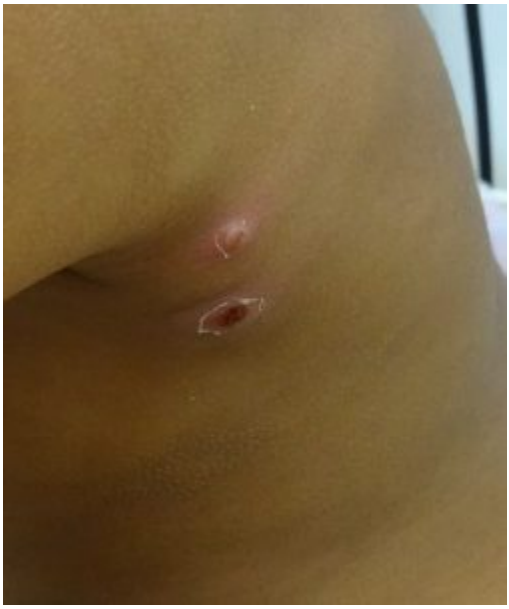

Image 6.5: Furuncle

Necrotizing Fasciitis is a deep infection involving the fascia located beneath the subcutaneous tissue. This is a life-threatening condition, and the extent of disease is often not evident from the findings seen on the skin. *S. pyogenes* is the most common cause, and infection usually enters the skin through a site of injury, although this may not always be the case. It often resembles cellulitis initially, however, rapid progression and pain out of proportion to skin findings are hallmark findings. If suspected, patients should be urgently seen in a tertiary centre for antibiotic and surgical treatment.

Staphylococcal scalded skin syndrome is a blistering skin condition most often seen in children under 5 years old. It is caused by toxins released by *S. aureus*. Although there is often a localized focus of infection such as the nasopharynx or conjunctivae, the areas of blistering are generally sterile. The rash often starts as redness around the mouth and within the skin folds. Flaccid, easily ruptured blisters develop. The skin around the mouth may develop characteristic "radial fissures". Skin tenderness, fever and irritability are often present. Patients generally require hospitalization for supportive care as well as to receive IV antibiotics covering *S. aureus*. Tape should be avoided as the skin will often peel off when the tape is removed.

Image 6.6: SSSS: perioral desquamation

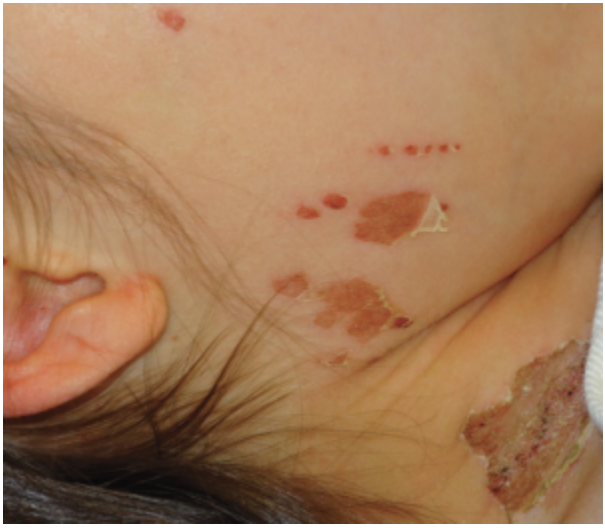

Image 6.7: SSSS: superficial peeling at a distant site

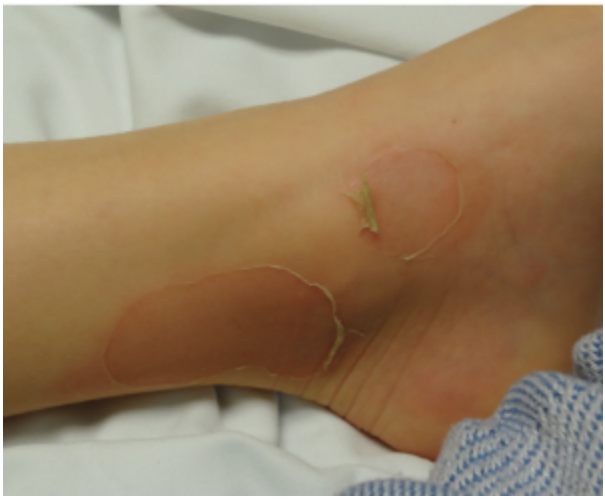

Image 6.8: Superficial peeling at a distant site

Bacterial Infections: Erythrasma

Erythrasma is a superficial bacterial infection that commonly affects the skin between the toes, in the groin and in the axillae.

What causes it?

It is caused by the bacterium *Corynebacterium minutissimum*. It occurs in healthy individuals but is more common in those with diabetes mellitus or who are immunocompromised. It may also be more common with warmer climates, excess sweating and poor hygiene.

What does it look like?

Erythrasma presents as well-defined pink to brown patches in the axillae, groin or web spaces between the toes. There may be fine scaling present and the rash may be slightly itchy but is often asymptomatic. Wood's lamp examination is a helpful tool to confirm the diagnosis as the bacteria cause a coral-pink fluorescence. The differential includes other skin conditions affecting the skin folds including intertrigo, inverse psoriasis, tinea, and candida infections.

How is it treated?

Mild disease can be treated with topical therapies such as clindamycin, erythromycin, fusidic acid or mupirocin. Widespread or resistant disease can be treated with oral antibiotics such as doxycycline or erythromycin. Antibacterial soaps can be used to help prevent recurrence.

Viral Infections: Verrucae (Warts)

What causes them?

Verrucae (warts) are a common condition caused by human papilloma virus (HPV). There are many sub-types of HPV, and each is most commonly seen in a characteristic location on the skin. Warts can occur anywhere on the skin, from the thick skin on the soles of the feet to the mucosal skin of the lips and genitals. Most warts are little more than an annoyance, but others can be associated with cancer formation.

There are 4 basic types of warts:

- **Verruca Vulgaris (common warts)** – Usually seen on the backs of the hands or around fingernails but can be anywhere. Very common in children. Raised, rough-surfaced lesions. May be single or in a cluster.
- **Verruca Plana (flat warts)** – Most common on face, neck, arms, and legs. Often seen in a straight line where skin was scratched (Koebnerization). Smooth, flat-topped papules often seen in clusters. People may have hundreds in one area.
- **Verruca Plantaris (plantar warts)** – Appear on the bottom of the feet. Often grow inward and more deeply than other warts. Most symptomatic of all warts due to pressure when standing. May lead to altered gait in children.
- **Condyloma Acuminata (genital warts)** – Seen around the anogenital track. Skin coloured, soft papules from 1-5 mm. Some subtypes are associated with cancer, especially cervical cancer. In very young children, spread is usually incidental, but in children between ages 5-12, the possibility of spread through sexual abuse should be considered.

How does someone get them?

Warts are passed from person to person. Usually this occurs by skin contact, especially if the

person had a small cut or scrape in the area to allow viral penetration. Individuals with decreased immune function due to cancer or HIV can have a large number of warts.

How are they treated?

There is no specific anti-viral therapy for HPV. Warts that are not bothersome to the patient can be watched in the hope that the patient's own immune system will recognize and clear the wart virus. Most therapies work by causing irritation, which increases the speed of this recognition by the patient's own immune system. There are many different treatments available for warts and each requires diligence.

- Over-the-counter salicylic acid preparations must be applied daily and work best when occluded with tape or a bandage unless the medication is formulated into an acrylic.
- Duct tape occlusion for five days before changing has also been shown to be effective. When it is removed, the wart is softened by soaking and then worn down with a nail file or pumice stone before a new piece of tape is applied.
- Topical retinoids such as tretinoin can be useful for flat warts
- Topical imiquimod or sinecatecinins can be used for condylomata acuminata

There are several in-office treatments available as well:

- Liquid nitrogen (cryotherapy) is the mainstay of therapy where it is available, but this treatment is painful and requires multiple visits and applications.
- Paring with a 15 blade scalpel decreases the pain of walking on plantar warts and can be followed by application of silver nitrate, which may leave a stain on the skin, but is an effective therapy.
- Canthardin can be applied in office, but increases risk of ring wart (central clearance with peripheral spread of wart) development. It should be washed off in 2-4 hours after application and should not be prescribed for home application.

In many places, HPV vaccine is given to males and females in young adolescents as part of the routine immunization program. This vaccine covers 9 strains of HPV, which cause ~90% of cervical cancers and the majority of anogenital warts. It is also indicated for other at-risk populations who may not have received it as part of the routine immunization program.

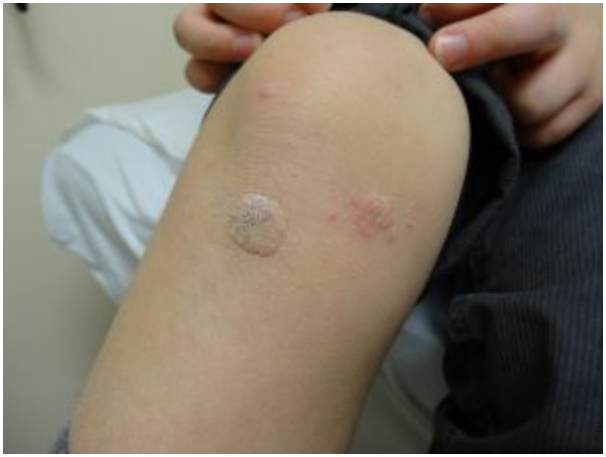

Image 6.9: Verrucous papules on the knee

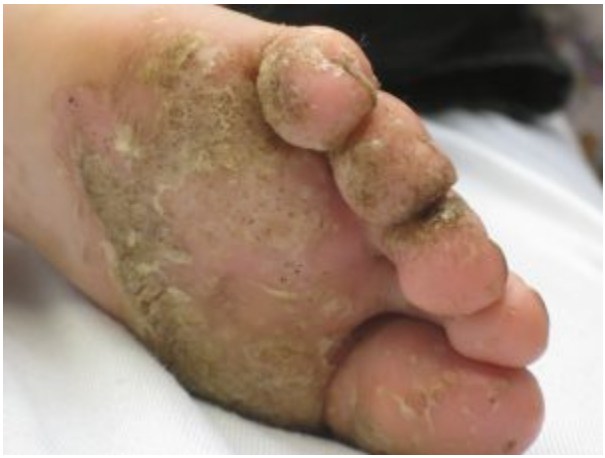

Image 6.10: Plantar warts in an immunocompromised patient

Viral Infections: Molluscum Contagiosum

What causes it?

Molluscum contagiosum is a common skin infection caused by a virus in the pox virus family. It is seen most commonly in children and is spread through skin-to-skin contact or through contact with fomites such as sharing towels. It is often spread from the initial site of infection to other sites in the same child (autoinoculation). A second peak of molluscum contagiosum is seen in young adults as a sexually transmitted disease with lesions primarily in the suprapubic area. Sporadic cases can occur in healthy adults as well as in association with HIV or other forms of immunosuppression.

What does it look like?

Molluscum presents as pearly, skin-coloured to pink papules. The classic lesions have a central umbilication. Molluscum lesions are 2-8 mm in size and are usually asymptomatic. They can occur in clusters, in linear configurations, or as solitary lesions. Although they can be seen anywhere, they are most common in areas of rubbing or moist skin such as the axilla, popliteal fossae, and groin. The lesions sometimes cluster in areas of atopic dermatitis (eczema) and may themselves cause dermatitis in the surrounding skin. They may develop significant erythema (redness) and some tenderness, which usually represents the body developing an immune reaction to the infection and may signal impending clearance of the lesions. They may leave pitted scars after resolution.

How are they treated?

Most molluscum lesions resolve spontaneously without treatment over the course of a year or more. Parents are often quite anxious about the lesions and treatment may be requested. The treatment can hasten the resolution, but aggressive therapy can lead to increased scarring. In-office therapies including cantharidin, which can be applied painlessly and then washed off after 2-4 hours. This may include a blistering reaction. The degree of blistering can be variable, so only a few should be treated at first visit. Liquid nitrogen can also be used, but is painful, especially for young children. At home treatment include mild irritants such as vinegar, tea-tree, or hydrogen peroxide.

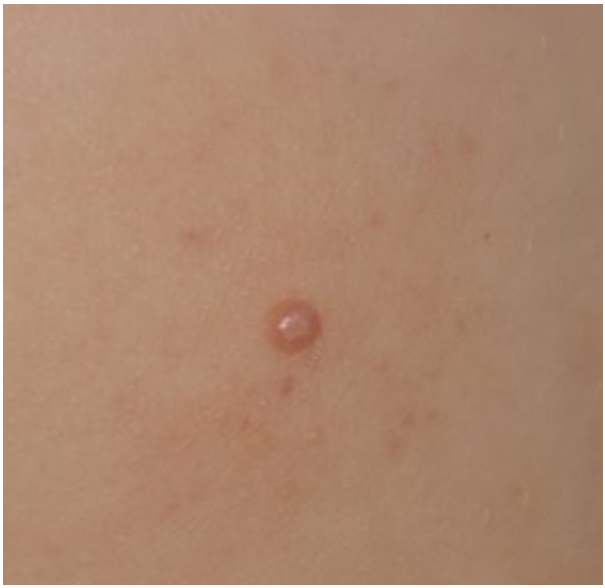

Image 6.11: Molluscum: Pearly umbilicated papule with mild surrounding dermatitis

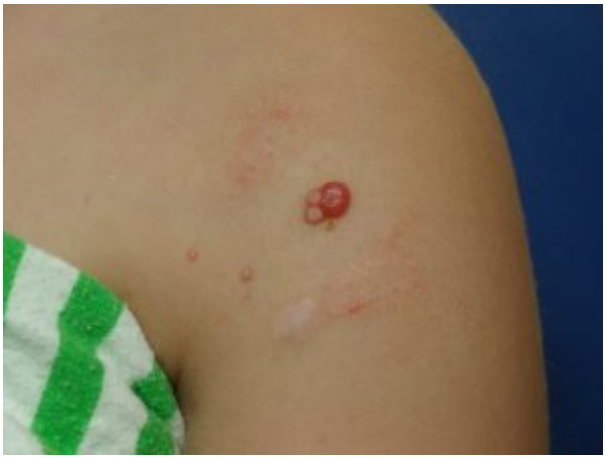

Image 6.12: Molluscum: Cluster of umbilicated papules with surrounding dermatitis

Viral Infections: Human Herpes virus (HHV)

Herpes viruses are double-stranded DNA viruses that replicate in the cell nucleus. They often have the ability to cause latent infections which can appear at a later point in the patient's life. The majority of patients with latent infections are asymptomatic. The important herpes viruses include HSV 1 and HSV 2, HHV 6 and 7, Varicella-Zoster virus, Cytomegalovirus, and Epstein Barr virus.

HHV	Other Name	Clinical Significance
1	Herpes Simplex Virus 1	Orolabial/genital herpes, herpetic whitlow, etc.
2	Herpes Simplex Virus 2	Orolabial/genital herpes, herpetic whitlow, etc.
3	Varicella-Zoster Virus	Varicella (chickenpox), zoster (shingles)
4	Epstein-Barr Virus	Mononucleosis, EBV-associated leukemia/lymphoma, Gianotti-Crosti, oral hairy leukoplakia, etc.
5	Cytomegalovirus	Retinitis, mononucleosis-like infectious syndrome
6	Roseolovirus	Roseola infantum, pityriasis rosea, drug reaction with eosinophilia and systemic symptoms (DRESS)
7	Roseolovirus	Pityriasis rosea, roseola infantum, drug reaction with eosinophilia and systemic symptoms (DRESS)
8	Kaposi Sarcoma-associated HHV	Kaposi Sarcoma

Viral Infections: Herpes Simplex Virus 1 and 2

Herpes Simplex Virus infection is very common and can be caused by either HSV1 or HSV2. These two viruses have a predilection for mucosal skin and so are seen most commonly in the mouth and in the groin area. HSV 1 is the most common cause of orolabial HSV (commonly known as cold sores), while HSV 2 is more commonly associated with genital lesions and is seen most commonly as a sexually transmitted infection. However, either form of the virus can be seen in either location, and both have been associated with non-mucosal infections as well.

What does it look like?

Lesions appear as grouped vesicles on an erythematous base. They are often painful and may cause swelling of the lips. A prodrome of tingling or burning may be felt in the 24 hours before the blisters appear. Symptoms are generally worse with first infections and are somewhat milder with recurrent infections. Recurrences are frequent in times of stress, after significant sun exposure or when the immune system is weakened.

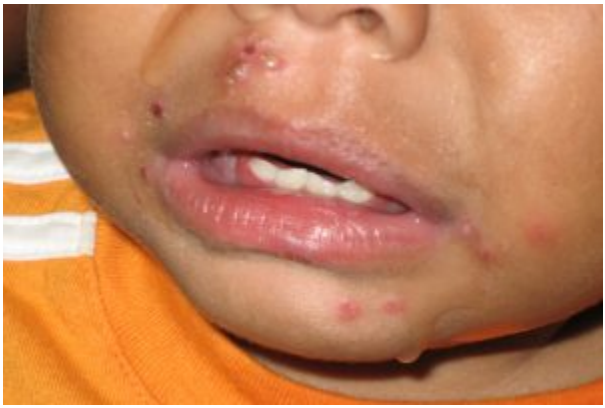

Image 6.13: HSV1: Vesicles and erosion in perioral distribution
Note small ulceration on mucosal lip

How is it diagnosed?

Classic lesions can be diagnosed clinically, but a viral swab for PCR is a rapid and sensitive method to confirm the diagnosis. The swab should sample the moist base of a recently ruptured vesicle. If the vesicle is still intact this will require deroofing, which can be achieved with a small needle.

How is it treated?

For first infections, treatment is with a short course of acyclovir. Treatment of most recurrent infections is supportive with topical anesthetics. Patients with frequent recurrences can be give na prescription that they begin when the tingling sensation is first noted. For patients with

particularly bothersome and frequent recurrences, suppression with acyclovir or valacylocvir can decrease the frequency of outbreaks.

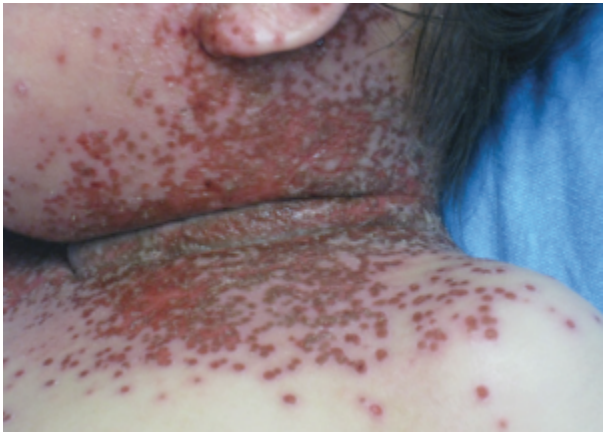

Image 6.14: Eczema herpeticum: Monomorphous punched-out ulcers

Other manifestations of HSV:

- **Herpetic Whitlow:** HSV lesions on the fingertips due to transfer from oral lesions by direct contact.
- **Eczema herpeticum:** Widespread skin involvement with HSV 1 in the setting of an underlying skin disease such as atopic dermatitis. These patients require hospitalization, isolation, and IV acyclovir therapy.
- **Recurrent erythema multiforme:** One of the most common causes of recurrent EM is HSV. These patients usually require treatment with suppressive acyclovir.
- **Neonatal HSV:** A medical emergency. Transmission from mother to neonate during delivery is most commonly seen in the mother's first infection and outbreak. Often mothers are asymptomatic at the time of delivery.

Viral Infections: Varicella-Zoster Virus

The Varicella-Zoster Virus causes both varicella (chicken pox) and zoster (shingles). Upon initial infection with the virus, the patient develops varicella with widespread involvement. As the infection clears, the virus goes into the nerve root where it stays in a latent form until a weakened immune system allows it to return as zoster.

Viral Infections: Varicella (Chickenpox)

What causes it?

Varicella is found worldwide and is most common in children during the late winter and spring. It is highly contagious both by direct contact and through respiratory secretions, especially in the few days before the rash appears and just afterward. The VZV vaccine has dramatically decreased the incidence of the disease.

What does it look like?

After an incubation period of 10-14 days, the patient often has mild headache, fever, and malaise for about 24 to 36 hours before the rash appears. The rash begins with red spots that soon turn to fluid-filled blisters. The bumps are said to look like a "dew drops on a rose petal" because the fluid-filled blister sits on a background of erythema. The rash usually begins on the scalp, face, or trunk and spreads to the extremities, but generally spares hands and feet. New spots continue to appear for 3-6 days. Old blisters crust over at the same time as new ones appear so the patient often has a mix of old and new lesions even in the same area of skin – the presence of lesions in multiple stages of development is a hallmark of this disease.

Is it dangerous?

For most children, chicken pox is an itchy, annoying sickness that has no complications. For some children – and more commonly for teenagers and adults – it can be dangerous. The complications of chicken pox are development of pneumonia, neurologic involvement, orchitis, and secondary infection with bacteria. Another concern is that it can affect a developing baby, so pregnant women should avoid contact with people who have the chicken pox. Also, once the spots are scratched open, they can become infected especially with Staphylococcal or Streptococcal species.

How is it treated?

For uncomplicated cases, the treatment is supportive: calamine, oatmeal baths, and antihistamines can help to minimize the itching down. Antipyretics can help with fevers. Topical antibiotics (e.g. mupirocin or bacitracin) on any scratched bumps can keep them from getting infected. For severe or complicated cases, or in immunocompromised patients, the treatment is with acyclovir. Patients should not return to school or to work until all of the lesions have crusted over.

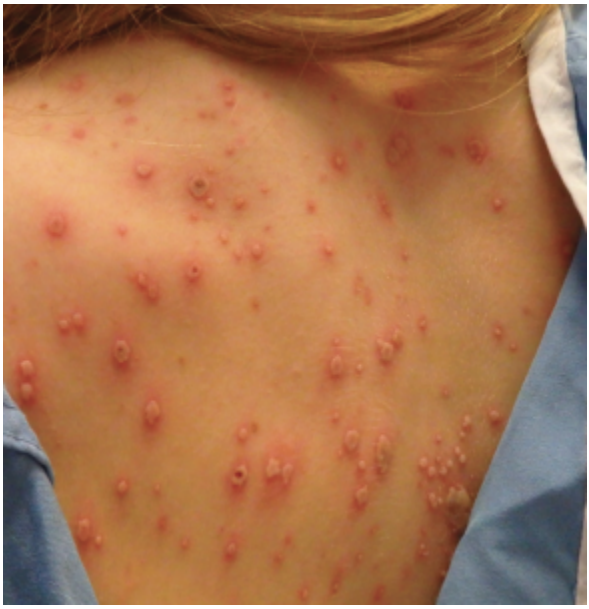

Image 6.15: Varicella: Crusted papules and vesicles

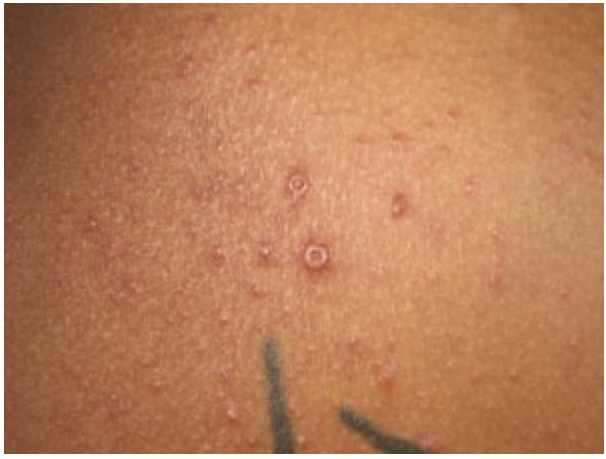

Image 6.16: Varicella: Vesicles with umbilicated appearance as they crust from the center outward

Viral Infections: Zoster (Shingles)

What causes it?

Zoster is caused by the reactivation of VZV and is uncommon in children. Following an episode of varicella, the virus travels down the nerves where it then stays, kept in check by the body's immune system. If there is a weakening of the immune system, whether from stress, illness, medication, or old age, the virus has a chance to multiply and travel back out the nerve to the skin where it causes blisters and pain. Vaccine-strain VZV can also cause zoster.

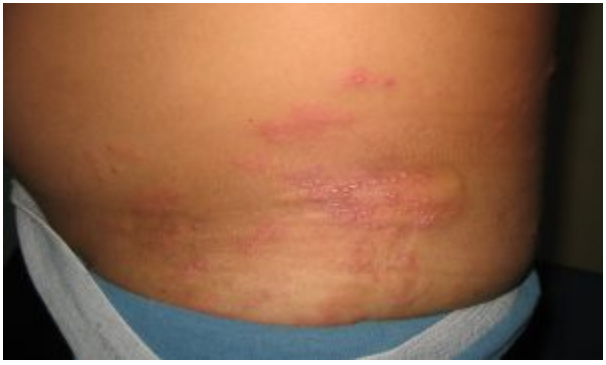

Image 6.17: Zoster: Vesicle on an erythematous base clustered in a dermatome

What does it look like?

Herpes Zoster is characterized by grouped clear vesicles on an erythematous (red) base. The lesions are found in a dermatomal distribution, which means that they appear on the skin in an area that gets its sensation from a single spinal nerve root. The rash rarely crosses over the midline of the body. Patients with zoster report significant pain at the site and do not have the itch that would be expected with other rashes.

How is it treated?

Getting rapid treatment is important in decreasing and controlling the symptoms of herpes zoster. Treatment with antiviral medications such as acyclovir should be started as soon as possible, ideally within the first 48 hours of lesions appearing. Pain control involves: moist dressings, NSAIDS, and local application of heat or pressure. IV acyclovir is indicated in cases of ophthalmic zoster or zoster in an immunocompromised person.

Viral Infections: Roseola Infantum

What is it?

Roseola infantum, also known as exanthem subitum, is a common childhood illness usually occurring between the ages of 6 and 36 months.

What does it look like?

The rash of roseola typically follows 3-4 days of high fevers, and its onset coincides with normalizing of body temperature. It consists of blanchable rose-coloured macules and papules on the neck, trunk, and buttocks with occasional involvement of the face and extremities. It usually resolves in a few days.

How is it managed?

Roseola can typically be diagnosed clinically and no lab investigations or further work up are necessary. Treatment is supportive and the illness is usually already resolving by the time the rash appears.

Viral Infections: Enteroviruses

The enteroviruses are a genus of single-stranded RNA viruses. They include several clinically significant viruses such as the echoviruses and coxsackie virus A/B.

Viral Infections: Hand-Foot-and-Mouth-Disease (HFMD)

What causes it?

HFMD is a common viral illness in children caused by coxsackie virus (usually coxsackie A-16) and other enteroviruses. It is most often seen in children between the ages of 1 and 4 but can be seen in older children and even adults. Transmission is usually fecal-oral but can be oral-oral as well.

What does it look like?

People with HFMD disease often have a prodrome of low-grade fever and malaise before developing any skin changes. The classic rash has red spots and blisters on the palms of the hands, soles of the feet, and in the mouth. The blisters are deep, have a grey appearance, and are often oval-shaped. They characteristically run along the skin lines on the fingers and toes. Patients can also have spots on the backs of the hands, tops of the feet, buttocks (especially in toddlers wearing diapers), and the knees. The mouth sores are often painful and can make it hard to eat and drink. Several weeks to months after HFMD, some children will develop nail changes called onychomadesis, which cause the nail to lift from the proximal edge.

How is it treated?

In most cases the virus goes away in about a week with no treatment other than pain medication and encouraging the person to eat and drink. There have been outbreaks with more dangerous strains, but these are rare. Treatment is supportive with the use of analgesics such as acetaminophen, topical anesthetics for painful oral lesions, and fluid administration to prevent dehydration. Eating can be difficult, and many people find that soft, bland foods and especially cold foods like ice cream, or even frozen vegetables (served still frozen) are soothing.

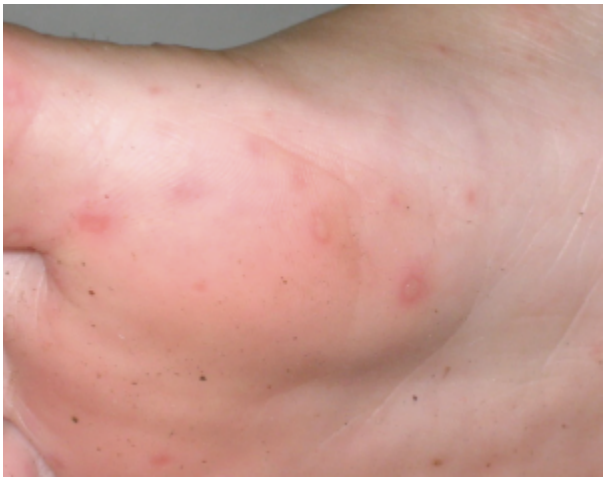

Image 6.17: HFMD: Grey vesicles with erythematous rim

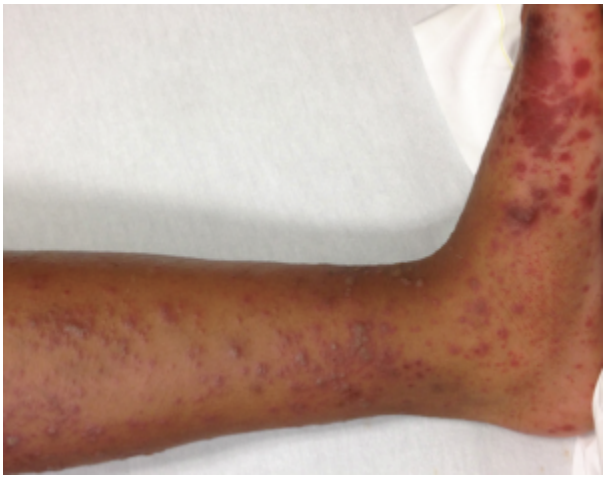

Image 6.18: HFMD: Note the deep red colour

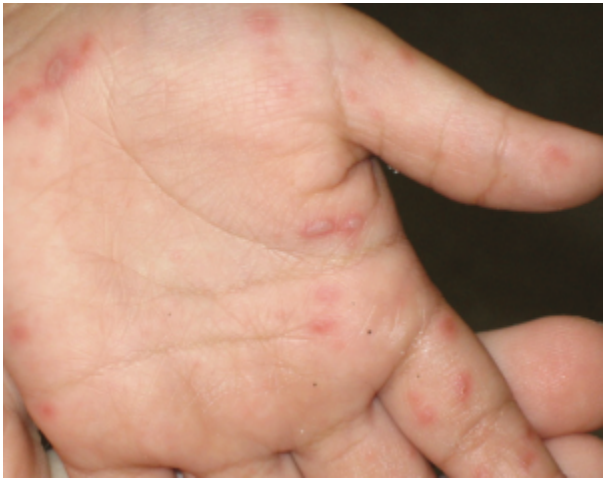

Image 6.19: HFMD: Note the oval shape of vesicles on the palm

Fungal Infections: Tinea Corporis

Tinea Corporis is a fungal infection localized to the uppermost layers of the skin. It is commonly known as "ringworm". The fungi that cause tinea corporis are called dermatophytes.

What does it look like?

The characteristic lesions are circular with a raised red border and associated scale. Most patients with tinea corporis are itchy, although the itch is typically less severe than that associated with nummular eczema.

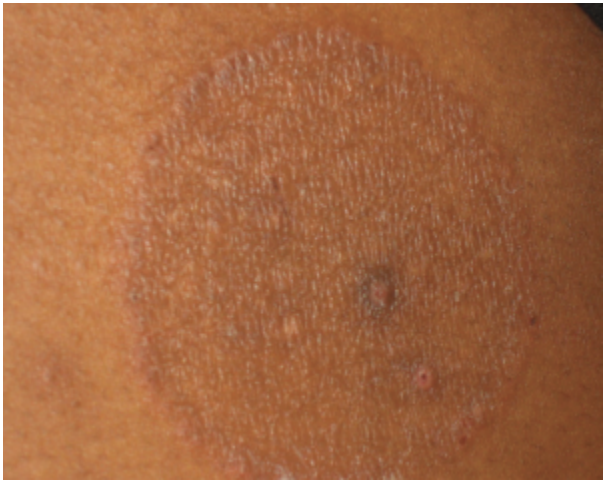

Image 6.20: Tinea Corporis: Annular plaque with scale

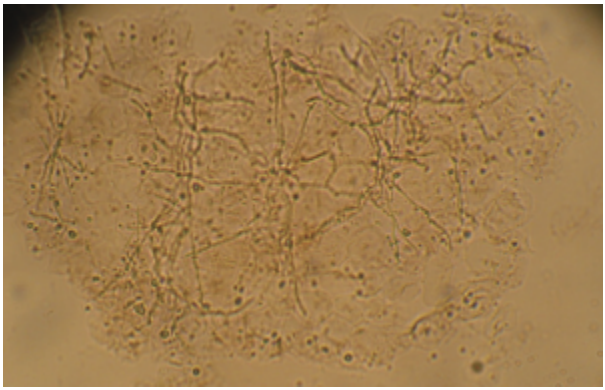

Image 6.21: KOH Prep with long branching hyphae

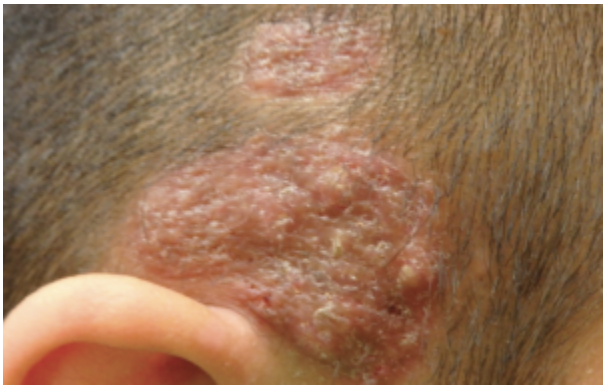

Image 6.22: Tinea capitis: Inflammatory and scaly plaque with hair loss

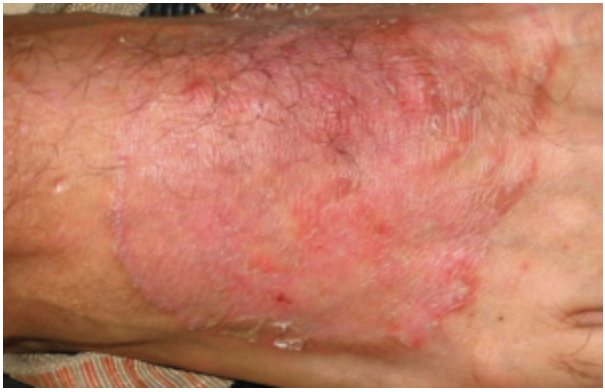

Image 6.23: Tinea pedis: Annular plaque with scale accentuated at border on the dorsal foot

What causes it?

There are several species of fungus associated with tinea corporis. Some of these are anthropophilic (meaning that they prefer to infect humans) and some are zoophilic (meaning that they prefer to infect animals). People get the infection when they come in contact with another person or an animal with the fungus on their skin.

How is it diagnosed?

In some cases, the presentation is very clear and it can be diagnosed clinically. In most cases though, it is difficult to tell apart from nummular eczema, which is also round, scaly, red, and itchy. For this reason, it is best to diagnose with a KOH prep, which is relatively quick and easy to do in clinic if there is an available microscope, or the scrapings can be sent to the laboratory for confirmation.

To perform a KOH prep, scrape the edge of one glass slide over the scaly edge of the lesion so that scale comes off onto a second glass slide. Cover with 1 drop of KOH and cover slip. The long branching hyphae are visible crossing the skin cells in the clump of skin seen on the slide. The hyphae become more clearly visible with time, so it is often helpful to set the slide aside and review at the end of clinic (see Appendix for further details).

How is it treated?

Unless it covers a large amount of the body, tinea corporis can be treated with antifungal creams. The most commonly used are terbinafine or the "–azoles" such as clotrimazole and ketoconazole. These can be used twice a day for 2-4 weeks. Topical steroids should not be used in treating tinea corporis because, while they may decrease the redness and scaling, they also diminish the patient's immune reaction to the fungus and allow the organism to multiply and may result in more resistant disease.

Oral therapy – indicated for tinea capitis, onychomycosis and extensive tinea corporis.

Specific terminology for other forms of fungal infection caused by dermatophytes:

Tinea faciei: Fungal infection of the face.

Tinea barbae: Fungal infection of the beard.

Tinea capitis: Fungal infection of the scalp – can develop into a large boggy lesion called a **kerion**.

Tinea cruris: Fungal infection of the groin.

Tinea pedis: Fungal infection of the feet.

Tinea manuum: Fungal infection of the hand (sometimes called "2-foot 1-hand" because it usually involves both feet but only one hand).

Tinea nigra: A fungal infection caused by one particular fungus, which makes the skin turn brown (*Hortaea werneckii*).

Tinea incognito: Fungal infection that has been treated with steroids. Since the inflammatory reaction is lessened, the infection looks better, but actually is getting worse. It can develop fungal folliculitis (Majocchi's granulomatosis) if the fungus tracks

into the hair follicles, which requires treatment with oral antifungals.

Onychomycosis: Fungal infection in the nail – does not clear without oral antifungals. Also sometimes referred to as **tinea unguium**.

Fungal Infections: Candidiasis

Candidal infections are most commonly caused by the yeast *Candida albicans*. They are seen in warm, moist areas such as in the diaper area of children or in skin folds.

What does it look like?

Cutaneous candidal infections are generally beefy red in color with satellite lesions that are pustulovesicular. Candida can also be seen in the mouth where it is known as thrush. This causes curd-like white plaques on the oral mucosa and tongue that can be scraped off with a tongue depressor. When located in the corners of the mouth, candida can cause perlèche (also known as angular cheilitis). This condition is associated with maceration and lip licking and causes the corners of the mouth to become erythematous and crack.

How is it diagnosed?

The diagnosis is clinical and can be suspected when bright-red plaques with satellite lesions are seen in intertriginous areas.

How is it treated?

Topical antifungals, such as miconazole or clotrimazole, are useful in the treatment of candidiasis but must be used twice a day in order to clear the infection.

Fungal Infections: Pityriasis Versicolor

Pityriasis versicolor is often called tinea versicolor; pityriasis is the correct term because it is caused by a yeast and not a dermatophyte.

What does it look like?

Patients with pityriasis versicolor usually present with light or dark spots that are round or oval in shape and vary in size from a few mm diameter to about 1cm in diameter. The surface of each macule has faintly visible scale or scale that can be seen after the macule is scratched lightly. The macules are classically found on the upper back and chest, but can be on the arms, abdomen, legs, and face as well.

What causes it?

Pityriasis versicolor is caused by the yeast *Malassezia furfur*. *M. furfur* is a normal resident on the skin and only causes problems when it overgrows. The yeast can overgrow in certain favorable conditions: high humidity, oily skin, treatment with steroids, and excess sweating. This condition can be as seen in as many as 20% of the population in tropical and subtropical areas.

How is it diagnosed?

Like dermatophyte infections, pityriasis versicolor is diagnosed clinically and confirmed with a KOH preparation if necessary. The yeast forms are much smaller than those seen in tinea and it is possible to see both spores and rounded hyphae (often said to resemble "spaghetti and meatballs") on the slide. This is different from tinea infections where long branching hyphae are seen. It is easiest to see the yeast at 40x power.

Is it contagious?

Not really. Since the yeast is present on everyone's skin already, touching someone with pityriasis versicolor doesn't increase the chances of having the condition.

How is it treated?

Selenium sulfide is the topical treatment of choice and either a lotion or shampoo can be used. The selenium sulfide must be left on for 10-15 minutes once a day before being washed off and should be used daily for 2 weeks. Patients may choose to use the shampoo or lotion once every few weeks on an ongoing basis because patients can relapse as the factors that led to the overgrowth of yeast are likely to be present in the future. It can also be treated with oral antifungals. Itraconzaole 400 mg in a single dose has proven effective, as has 300 mg fluconazole with a repeat dose at 2 weeks. With oral therapy, the effect is enhanced if the patient exercises to the point of a slight sweat 30 minutes after taking the medication and then waits overnight before showering. It is important to note that the scale and pruritus should resolve immediately after treatment, but the pigment change can take months to return to normal.

What is the differential diagnosis?

Pityriasis alba (*see Ch. 13*): This is a form of mild eczema where the skin is hypopigmented and slightly scaly. There is occasionally a tiny bit of associated redness. These areas are usually dry and there is often a history of eczema. The patches are usually larger and more ill-defined compared to pityriasis versicolor. It is most common on the face.

Vitiligo (*see Ch. 13*): This is typically characterized by white patches with complete loss of pigment (depigmentation) compared to the light patches with partial loss of pigment often seen in pityriasis versicolor (hypopigmentation). In vitiligo, there is no scaling associated with the white patches and they tend to occur bilaterally and in specific areas (e.g. around the eyes, on

the hands/feet, and in the groin). The size of the patches is variable from small confetti-like dots to virtually the entire body.

Post-inflammatory hypopigmentation (*see Ch. 13*): After a rash improves, the area can be left either light (hypopigmented) or dark (hyperpigmented). Usually there is a history of rash which precedes the pigment change in these cases.

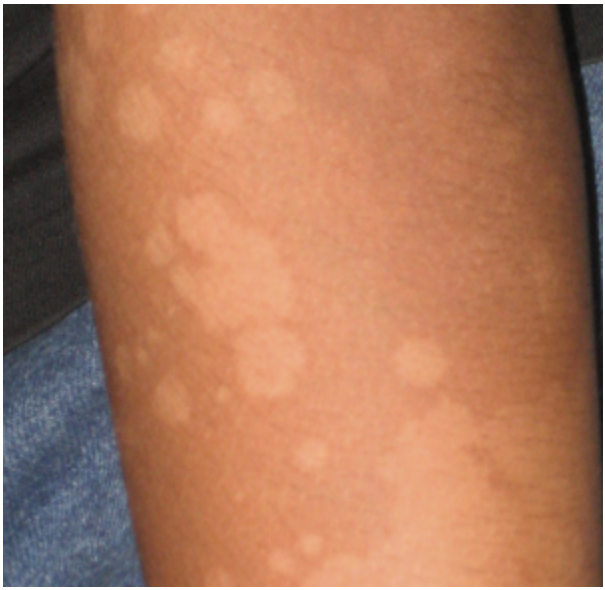

Image 6.24: PV: Hypopigmented macules coalescing into patches

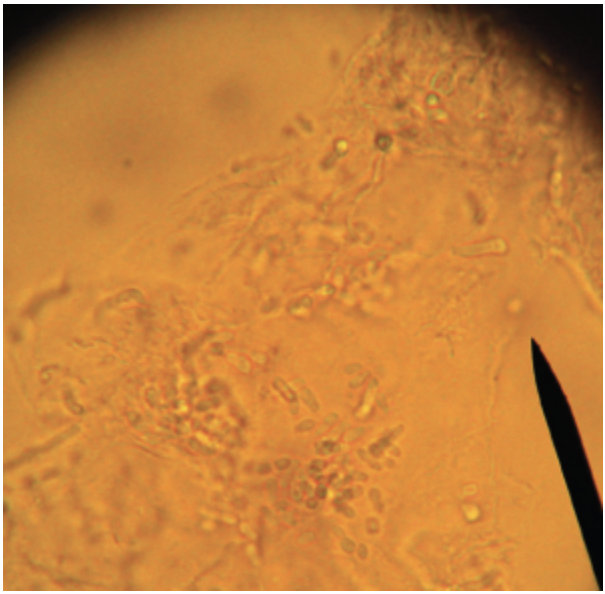

Image 6.25: KOH prep (40x power) shows spores and short hyphae

Infectious Exanthems

An exanthem is a widespread rash that can be triggered by an infection as well as other causes such as medications. Infectious exanthems are especially common in children and may have characteristic features depending on the causative organism.

Non-Specific Viral Exanthem

Non-specific viral exanthems are the most common exanthems in children. They present as red macules and papules that are blanchable (redness fades when pressure is applied), distributed widely on the trunk and extremities, and often coalesce. The rash is often associated with viral symptoms such as fever and might be difficult to distinguish from a morbilliform drug eruption. There are numerous viruses which cause non-specific viral exanthems including enterovirus, adenovirus, parainfluenza and respiratory syncytial virus.

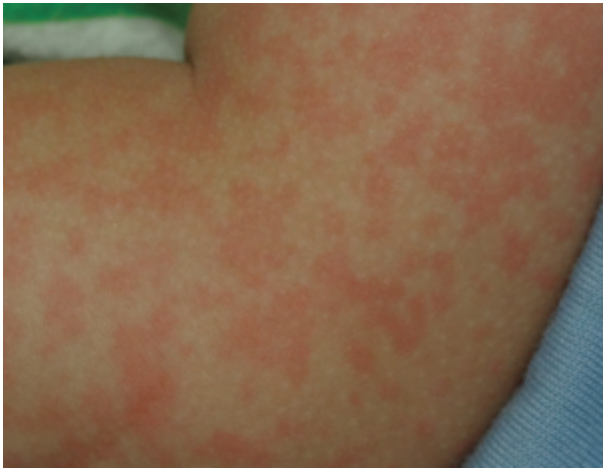

Image 6.26: Coalescing blanchable macules and thin papules in non-specific viral exanthem

Erythema Infectiosum

Erythema infectiosum, also known as "fifth disease", is caused by infection with Parvovirus B19. This is a common disease of school-aged children and typically occurs during the winter and spring. The exanthem occurs approximately 1-2 days following a prodrome of mild fever and headache. It begins as a distinct "slapped cheek" appearance with bright red patches to both cheeks. This is typically followed by a lacy red rash on the extremities that lasts for 1-3 weeks. There is no specific treatment and affected children can attend school, as the infectious stage occurs before the rash is evident.

Measles

With high vaccination rates in many countries, measles is becoming a less common disease worldwide. Still, it is a cause of significant morbidity and mortality globally and is highly contagious with up to 90% of susceptible people who get exposed contracting the disease.

Outbreaks continue to occur even in countries with high vaccination rates and have been seen frequently in recent years, especially in populations with high rates of vaccine avoidance. Measles is caused by a single-stranded RNA paramyxovirus. It is transmitted by air-borne droplets from 1-2 days before the onset of symptoms until 3-4 days after the rash appears. Patients experience a prodrome of cough, coryza (runny nose), and conjunctivitis. The first skin lesions are called *Koplik spot*sand are 1-2 mm blue-white macules on the oral mucosa (typically the inner cheeks). The rash appears about 2 weeks after exposure and 2-4 days after the beginning of symptoms. It is characterized by non-pruritic macules and papules beginning on the head and neck then spreading to the trunk and extremities (cephalocaudad spread). Treatment is with supportive care, vaccination of any unvaccinated contacts, and Vitamin A supplementation in children who contract the disease. This supplementation has been shown to decrease mortality by 30% in children and works to strengthen the mucosal barrier in the respiratory and gastrointestinal tracts. Complications can be serious and include pneumonia, encephalitis and myocarditis.

Rubella

As with measles, the incidence of rubella has decreased significantly with the advent of routine vaccination. As a result, it is very uncommon in most of the world; however, it is still of clinical importance due to some of its serious complications and the risk of fetal infection which can cause significant congenital abnormalities. A prodrome of fever, headache and malaise is followed 5 days later by an exanthem of "rose-pink" macules that starts at the head and travels downward (cephalocaudad spread). There may also be small red dots on the soft palate accompanying the exanthem which are known as *Forcheimer* spots. In most healthy children and adults, the disease is self-limiting and treatment is supportive.

Scarlet Fever

Scarlet fever is a bacterial illness due to toxins produced by *Streptococcus pyogenes* and was often fatal in the pre-antibiotic era. It most often occurs in children aged 4-8 and is associated with streptococcal pharyngitis ("strep throat") or impetigo (superficial skin infection with *S. pyogenes*). It typically begins with fever, sore throat and swollen neck glands with a distinct exanthem appearing 12-48 hours following this. The exanthem consists of tiny pink to red spots that cover most of the body and have a characteristic "sand paper" texture. The tongue is often swollen and red ("strawberry tongue"). Diagnosis can be assisted with a throat swab showing growth of *S. pyogenes*or with anti-streptolysin-O titres. The treatment of choice is penicillin for 10-14 days – a complete course is important to reduce the risk of complications such as rheumatic fever and post-streptococcal glomerulonephritis.

Gianotti-Crosti Syndrome

Gianotti-Crosti syndrome was initially associated with Hepatitis B infections but more recently has been shown in association with various other viral infections (EBV, CMV, adenovirus, etc.) and some non-viral infections (*S. pyogenes*, *Mycoplasma pneumonia*). It is seen in children aged 6 months-14 years and causes monomorphous (all the lesions have a similar appearance), flat-topped, pink/brown, edematous (swollen) papules most often located on the knees and elbows and less often on the face and buttocks. The trunk is typically spared. Lesions last for over 10 days, but do not require any treatment and resolve spontaneously. Rarely, the rash can last up to 8 weeks.

Kawasaki Disease

Kawasaki disease is a multisystem disease that generally affects children less than 5 years old. While the exact cause still remains unknown, infectious etiologies have been postulated. Kawasaki disease is significant for being the number one cause of acquired heart disease among children in North America. The classic findings include fever lasting more than 5 days, redness of the conjunctivae, unilateral cervical lymphadenopathy, swelling/redness of the hands/feet, and "strawberry tongue". An exanthem is present in ~80% of cases but its appearance is variable. Most commonly it is widespread red macules and papules similar to measles, and may favor the perineal area. If caught early enough the treatment of choice is intravenous immunoglobulin (IVIg). Patients should be referred to cardiology to assess for any cardiac involvement.

Infestations: Scabies

What is it?

Scabies is an infestation of the skin with a mite called *Sarcoptes scabiei* that lives under the top layer of the skin (stratum corneum). The itch and rash are caused by a hypersensitivity reaction to the mite and its feces/eggs.

What does it look like?

People with scabies are usually very itchy. The itching usually begins about 3 weeks after contact with the scabies mite and is usually worst in the evening/night. Skin findings include papules, nodules, burrows (lines in the skin where the mite has lived and traveled), and blisters/pustules. The most common locations are between the fingers, on the wrists, ankles, axillae, waist, groin, palms, and soles. In infants only, the lesions can also be seen on the head. Scabies nodules are a reactive process to the mite and are commonly seen in the groin and axillae (nodules on the scrotum or penis in a patient with diffuse itching are diagnostic).

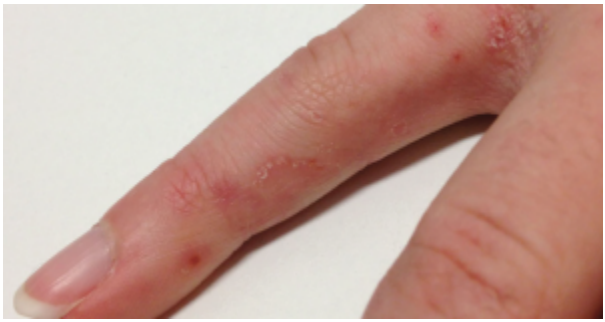

Image 6.27: Scabatic burrow on inner finger

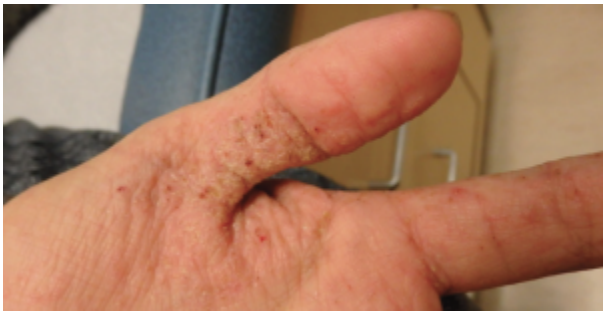

Image 6.28: Pruritic scaly and crusted plaque on hand

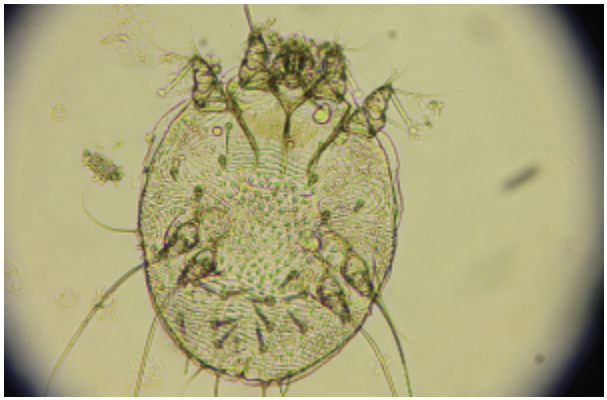

Image 6.29: Scabies prep showing adult mite

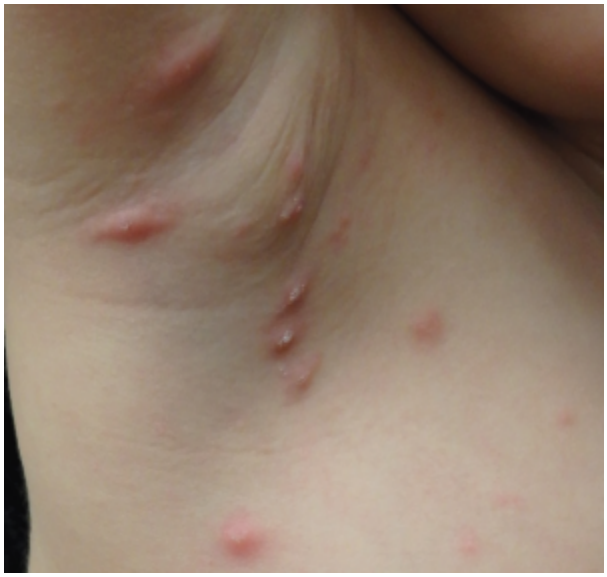

Image 6.30: Scabies: Axilliary nodules

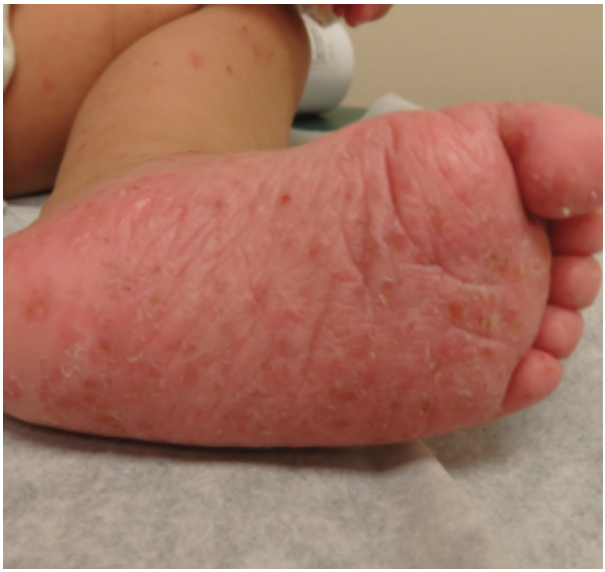

Image 6.31: Scabies: Papules, pustules, and burrows on an infant's foot

How do people get it?

Scabies mites are usually spread through skin-to-skin contact between people, but it is possible to get it from clothes or sheets that also have the mite. The mite can live for several days away from a person, so it is possible to contract scabies from contact with clothes or sheets that someone with scabies used several days before.

How is it diagnosed?

A diagnosis of scabies can often be made clinically based on a suggestive history (e.g. multiple cohabitants with similar rash) and with classic lesions such as burrows or scrotal nodules. However, in cases where the diagnosis is unclear, a scabies prep can be helpful. To do a scabies prep, clean a few suspected burrows and papules with alcohol and then scrape with a 15 blade scalpel. Because the mite lives under the stratum corneum, the scraping must be a bit more firm/deep than the very superficial scraping done to diagnose fungal infections; therefore, a small amount of bleeding is expected. The scraping is smeared on a glass slide and either KOH or mineral oil is placed on the slide before the cover slip is put in place. Mineral oil can also be placed on the skin or blade beforehand to help collect the scraped material more easily. With mineral oil, the mite will survive and may be seen moving on the slide. Additionally, it is easier to see eggs or feces when using mineral oil. Because each infested individual has only about 10 mites at any one time, it is usually necessary to scrape many papules at once to get a positive diagnosis. Scabies mite can also be seen with a dermatoscope. It is visible as a small dark triangle, known as the delta wing sign, at the end of a burrow.

How is it treated?

There are several treatments available for scabies. The most commonly used is 5% permethrin cream, which is applied to all skin from the neck down at bedtime. To be effective, the cream must be applied everywhere including between the fingers, under the nails, and in the groin

area. In infants, it must be also be used on the scalp and face, being careful not to get it in the eyes or mouth. In the morning after the application of the cream, it should be washed off and all sheets and clothing/undergarments should be washed. This procedure is repeated one week later. Because scabies is so contagious, it is recommended to treat all members of the family and others who have had close contact with the patient. All close contacts should be treated at the same time. Clothing and linens used within the previous week can be washed in hot water or stored in a bag for 10 days to kill any mites that may be living there.

Infestations: Pediculosis

Pediculosis

Pediculosis refers to an infestation of lice – flat, wingless insects that live on humans and may cause symptoms such as itch. Pediculosis capitis (head lice) is most common in children. Patients generally present with intense itching on the scalp. Lice may be identified visually, and nits or eggs will be visible along the hair shaft. Nits are firmly rooted to the hair and are not easily slid along the shaft. Treatment is with 1% permethrin lotion, ivermectin lotion, isopropyl myristate, and others. All treatments must be reapplied after 1 week because they cannot kill the eggs. Lice and nits should be physically removed from the hair as much as possible. Some children may require cutting or shaving the hair to allow for easier treatment. Pediculosis corporis (body lice) do not actually live on the skin or hair, but live in the seams of clothing and only move to the skin to bite for food. The lice may live in clothing for up to a month between meals, so infected clothing must be treated with high heat and not simply placed aside. Washing clothing and placing them in a hot dryer for 30 minutes or ironing is effective treatment. Pediculosis pubis (crab lice) are seen in pubic hair and less often in the thicker hairs on the chest, axilla, and eyelashes. They are spread through close physical contact, most commonly sexual. Treatment is as with head lice; sexual partners should be treated as well. Eyelashes can be treated with petrolatum twice daily for 8 days.

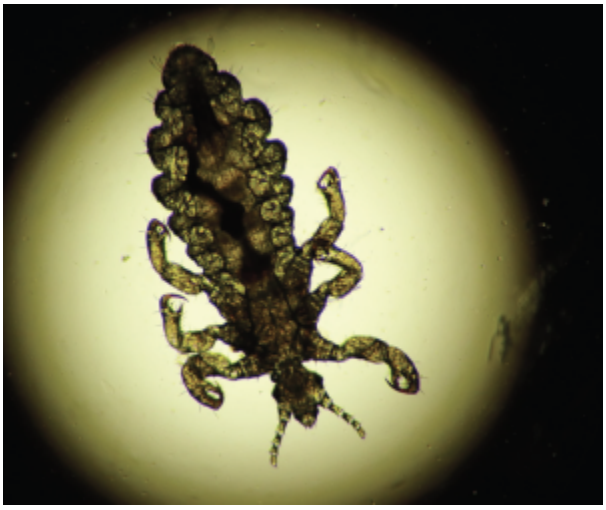

Image 6.32: Louse

Quiz

1. What is the incubation period of varicella?

a. 3-5 days
b. 10-14 days
c. 1-2 days
d. 5-7 days

2. Cellulitis is a bacterial infection.

a. True
b. False

3. Where are verruca plana (flat warts) not commonly found?

a. Face
b. Neck
c. Feet
d. Legs

4. HFMD is usually a childhood illness.

a. True
b. False

5. What type of fungi causes tinea corporis?

a. Candida albicans
b. Dermatophytes
c. Chytrids
d. Staphylococcus aureus

6. Which of the following oral antibiotic treats impetigo?

a. Cephalexin
b. Erythromycin
c. Dicloxacillin
d. Clindamycin
e. All of the above

7. Which of the following is not caused by a bacterial infection?

a. Erythrasma
b. Cellulitis
c. Verrucae
d. Impetigo
e. All are bacterial infections

8. How does one get verrucae?

a. Bacterial infection
b. Genetically inherited
c. Skin contact with someone who has it
d. Poor hygiene

9. Topical steroids can be used in treating tinea corporis.

a. True
b. False

10. All of the below are part of proper treatment of scabies in infants expect:

a. Permethrin 5% from neck down
b. Permethrin 5% to all skin including scalp
c. Treatment of all close contacts
d. Washing all clothing and linens in hot water

Answers: 1. B 2. A 3. C 4. A 5. B 6. E 7. C 8. C 9. B 10. A

CHAPTER 7: VASCULAR CONDITIONS

Salmon Patch

What is it?

Also called Nevus Simplex, Salmon patch is the most common vascular lesion in infants, occurring in 30-40% of newborns. When it is present on the nape of the neck it is often referred to as a "stork bite" and on the forehead or glabella as an "angel kiss". Salmon patch represents a benign capillary malformation.

What does it look like?

Salmon patches appear as a flat, pink to red blanchable patch with an indistinct and irregular border. They are most common on the forehead, glabella, upper eyelids, posterior neck and scalp. They often become pronounced with crying or physical exertion.

How is it treated?

No treatment is necessary. The majority of facial salmon patches will fade in the first 1-2 years of life. Lesions on the posterior neck may fade but are more likely to persist indefinitely. These are not usually of cosmetic concern as they are generally covered by hair.

Telangiectasia

Telangiectasia represent small, persistently dilated blood vessels in the skin. Telangiectasia can occur as a primary process, as a result of damage to the skin from the sun or following radiation therapy, or secondary to a systemic disease. Multiple telangiectasia should prompt referral to evaluate for an underlying cause such as hereditary hemorrhagic telangiectasia (HHT), ataxia-telangectiasia and others. Treatment of telangiectasia is often not necessary, but can include cosmetic camouflage, vascular laser, and sclerotherapy.

Spider Angioma

A spider angioma is a form of telangiectasia with a central feeding arteriole. They appear as a blanchable, central red papule surrounded by radially extending fine red lines. This gives it its name as a "spider" angioma. They are often located on the face, neck and upper chest. Solitary spider angiomas are common in children and often disappear with time, so do not require treatment. If treatment is desired for cosmesis, electrocautery or vascular laser can be used to destroy the central feeding vessel.

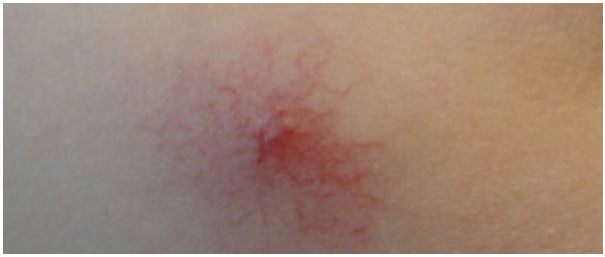

Image 7.1: Telangiectasias surrounding a vascular papule

Vascular Malformations: Port Wine Stain

What is it?

A port wine stain (PWS) is a congenital capillary malformation. There are several syndromes that are associated with PWS, including Sturge-Weber syndrome and overgrowth syndromes (Diffuse capillary malformation with overgrowth (DCMO), Klippel-Trenaunay syndrome, Megalencephaly-capillary malformation (MCAP), CLOVES syndrome, Proteus syndrome and PTEN Hamartoma syndrome). Facial or large PWS should prompt a work-up to rule out an associated syndrome.

What does it look like?

PWS are usually present at birth as well-demarcated, bright or deep red macules and patches. They are most commonly unilateral and often occur on the face. PWS grow in proportion with the child. Over time PWS can become darker in colour and develop skin thickening and nodules. These changes are most common in facial PWS and rarely seen in those on the trunk or extremities.

How is it diagnosed?

PWS do not spontaneously resolve or involute. Many patients want treatment due to the cosmetic appearance of these lesions and they can have a significant psychosocial impact. Treatment with a vascular laser is very effective although it does require multiple treatment sessions. If a PWS is associated with Sturge-Weber syndrome or an overgrowth syndrome, these patients require multidisciplinary care by the family physician, specialist pediatricians, and dermatology.

Vascular Malformations: Venous, Arteriovenous and Lymphatic Malformations

What are they?

Vascular malformations represent localized anomalous vessels and are categorized by the predominant vessel type. There are capillary malformations (discussed above, most commonly a port wine stain), venous, arteriovenous and lymphatic malformations. Another way to think of these is as slow flow (capillary, venous, and lymphatic malformation) or fast flow (arteriovenous malformations) and this can be seen by doppler ultrasound. Vascular malformations are congenital lesions that are typically present at birth and persist throughout life with either proportionate growth or a slow increase in size over time.

What do they look like?

Venous malformations are soft, blue papules or plaques that are compressible and fill with dependency. These can involve underlying muscle, bone and joints.

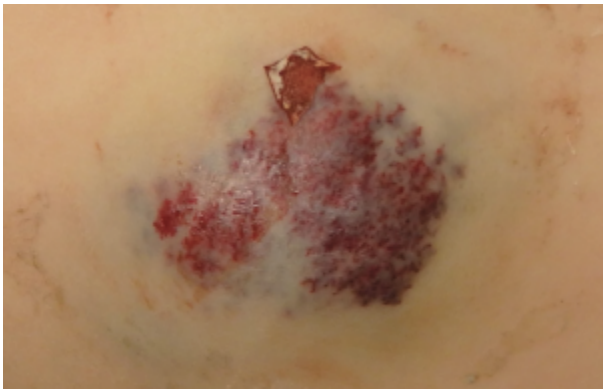

Image 7.2: Venous malformation with bleeding

Primary lymphedema presents as fluid accumulation most commonly in the lower extremities.

Microcystic lymphatic malformation, also known as lymphangioma circumscriptum, presents as clusters of clear or hemorrhagic vesicles. They are most common on the proximal limbs and chest but can occur anywhere including the oral cavity.

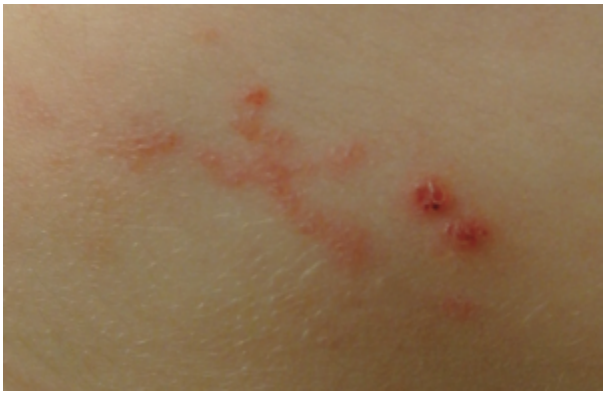

Image 7.3: Microcystic lymphatic malformation with superficial blebs

Macrocystic lymphatic malformation are most common on the neck, axilla and trunk and present as a large, soft, translucent mass underlying the skin. They often enlarge if the child has an infection.

Arteriovenous malformation (AVM) represent direct communications between arteries and veins which results in a fast flow shunt. These are rare vascular malformations, and unlike the other types only 40% are present at birth and the remainder appear later in life. The most common location is cephalic. They can cause complications such as skin necrosis or even high output cardiac failure.

There are many syndromes that are associated with vascular malformations. If a patient has multiple or large vascular malformations this should prompt a thorough work-up and involvement of a multidisciplinary team.

How are they treated?

Treatment of a vascular malformation depends on the size, location and other patient factors. Possible treatments include close observation, surgical excision, laser therapy, embolization, sclerotherapy, or oral medications such as mTOR inhibitors.

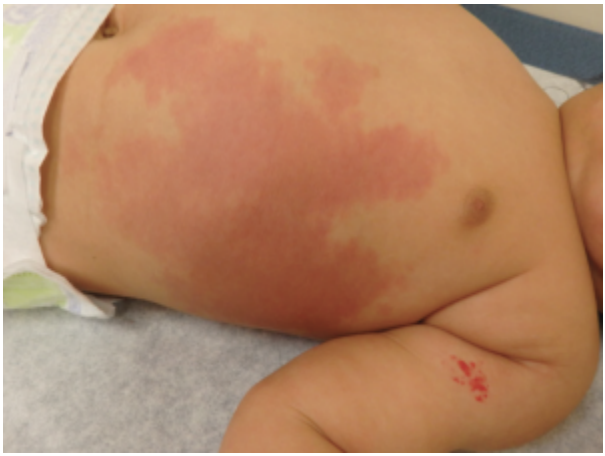

Image 7.4: Port wine stain on abdomen and flank.
Note: compare to hemangioma on arm

Vascular Tumours: Infantile Hemangioma

What is it?

Infantile hemangiomas (IH) are common, benign vascular tumors. They occur in approximately 5% of infants. Risk factors for IH include female sex, prematurity and low birth weight, placental insufficiency, multiple gestations and advanced maternal age. The cause of infantile hemangioma is incompletely understood and likely involves several mechanisms. It is thought that hypoxia plays a key role in initiation the growth of IH. Most are not present at birth, but appear by 3-4 weeks of age and grow rapidly within the first 3 months. The majority of growth happens by 6-9 months, followed by growth arrest. Spontaneous gradual involution starts around 1 year of age and continues until the child reaches 9 years old. There are several syndromes and specific complications that are associated with IH discussed below.

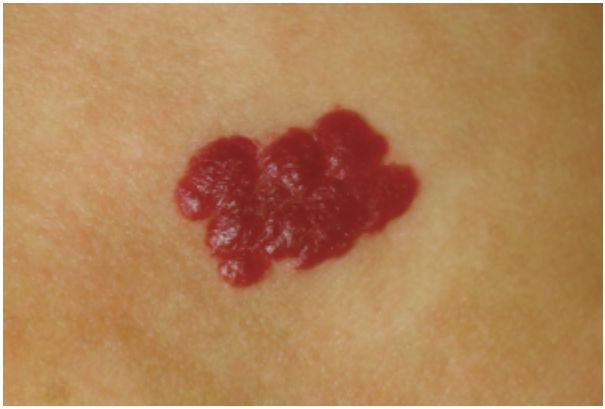

Image 7.5: A bright red vascular tumor on the chest of an infant typical of IH

What does it look like?

At birth, precursor lesions including pale areas, pink macules or bruise-like patches may be noted. More mature hemangiomas may be superficial, deep variants or mixed lesions with features of both. Superficial IH are bright red plaques with a finely lobulated surface leading to the name "strawberry hemangioma". Deep IH present as ill-defined blue masses which may have minimal or no overlying skin changes. Mixed lesions have a bright red superficial component overlying a deeper blue nodule. Hemangiomas, especially large or genital lesions, may develop central ulceration. Infantile hemangioma can be focal or segmental. The distribution and size of IH is important because of the risk of associated syndromes. Large, segmental IH especially on the face have a higher risk of **PHACES syndrome** (Posterior fossa malformations, Hemangioma, Arterial anomalies, Cardiac anomalies and aortic coarctation, Eye abnormalities, Sternal clefting and Supraumbilical raphe).

- IH located in the midline lumbosacral area are a marker of occult spinal dysraphism and large IH on the lower body have a risk of **LUMBAR syndrome** (Lower body/lumbosacral hemangioma and Lipomas, Urogenital anomalies and Ulceration of hemangioma, Myelopathy, Bony deformities, Anorectal and arterial anomalies, and Renal anomalies).

- IH that occur in a "beard" distribution over the mandible, chin and neck have a risk of airway involvement.
- Patients with multifocal (>5) IH are at risk of having visceral hemangiomas, most commonly in the liver.

How is it treated?

Since many will regress spontaneously, not all infantile hemangioma require treatment and active non-intervention with close follow-up may be appropriate for small, non-ulcerated IH on the trunk or extremities. Small, superficial IH in more cosmetically sensitive areas may be treated with topical betablockers such timolol. Standard therapy for complex IH is oral propranolol. Propranolol works quickly to halt growth, but in some instances oral corticosteroids may be required. More information on timolol and propranolol can be found in Chapter 16.

Indications for treatment include:

1. Location in cosmetically sensitive areas and may result in deformity (such as on the face, and especially the nose, lip and ear),
2. Potential to interfere with function (such as periorbital interfering with vision, around the mouth that impacts feeding, or airway), and
3. Large, deep or ulcerated IH.

Patients at risk of PHACES or LUMBAR syndrome should be referred for multidisciplinary care including general pediatrics, dermatology, neurology and cardiology. Infants with beard IH should be referred to ENT to rule out airway involvement. Infants with multiple (>5) IH should be have an abdominal ultrasound to rule out visceral hemangiomas.

Though hemanigiomas do typically regress, they may not disappear. Sometimes the residual skin changes are treated with laser or surgery when children reach school age.

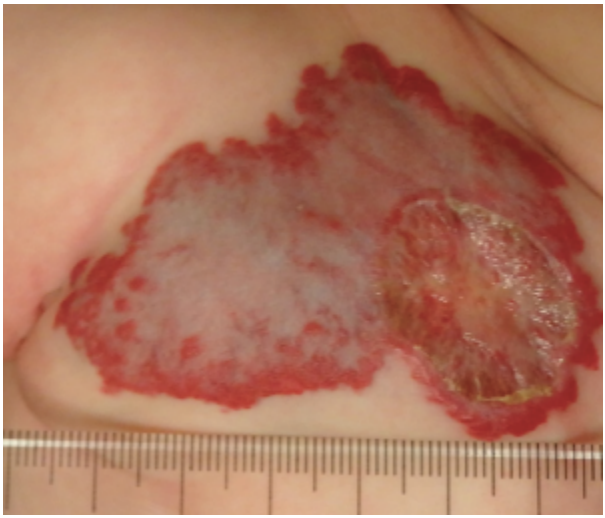

Image 7.6: Infantile hemangioma with dusky and ulcerated center

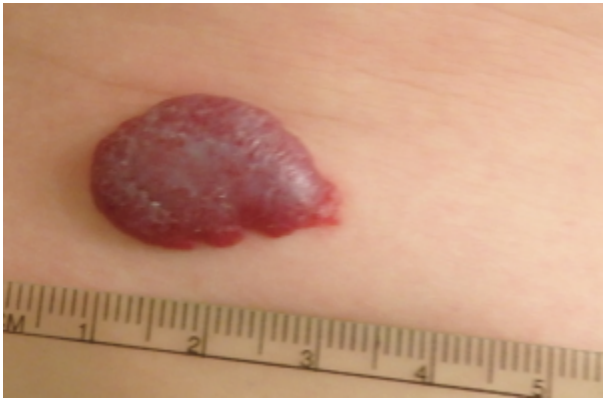

Image 7.7: Infantile hemangioma on abdomen

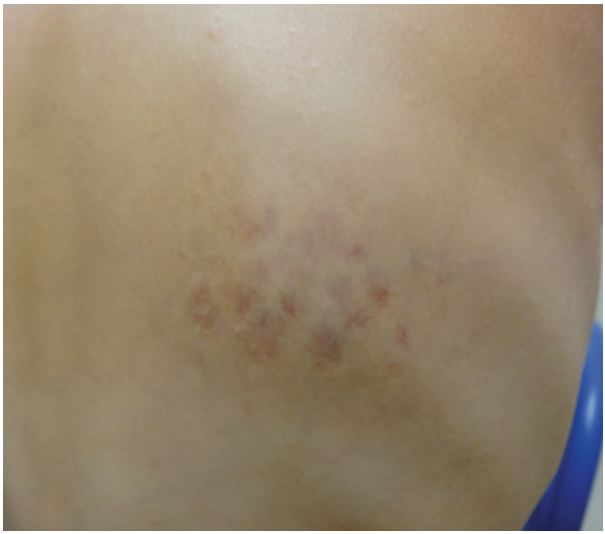

Image 7.8: Residual texture and vascular changes on the back at the site of a regressed infantile hemangioma

Vascular Tumors: Congenital Hemangioma

What is it?

Congenital hemangioma are present and fully formed at birth. They generally either rapidly involute (Rapidly Involuting Congenital Hemangioma, RICH) or they persist unchanged (Non-involuting Congenital Hemangioma, NICH). Congenital hemangiomas are equally present in males and females.

What does it look like?

When compared with an infantile hemangioma, congenital hemangiomas tend to present as deeper nodules with overlying prominent telangiectasia and peripheral pallor. RICH can completely involute by 12-15 months old and can leave residual atrophy. Possible complications include necrosis, ulceration and bleeding.

How is it treated?

Congenital hemangiomas do not respond to beta-blocker therapy. RICH do not require treatment since they generally involute. Surgical excision is usually the treatment of choice for NICH.

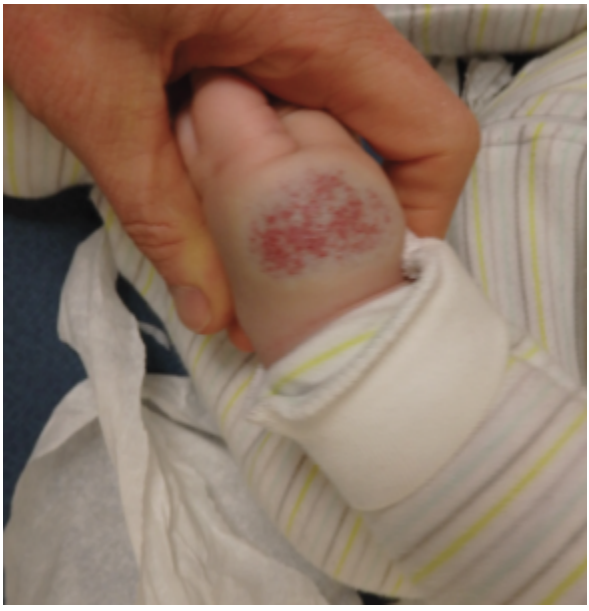

Image 7.9: Congential hemangioma

Vascular Tumors: Pyogenic Granuloma

What is it?

Pyogenic granulomas are common, acquired benign vascular lesions. They can develop at any age but are common in children and young adults. The exact cause is unknown but they are commonly associated with trauma.

What does it look like?

Pyogenic granuloma presents as a rapidly growing bright red, or sometime red-brown, slightly pedunculated papule. They have a friable appearing surface and are prone to superficial ulceration and bleeding. Pyogenic granuloma can present on the skin or mucus membranes but are especially common in areas of trauma and on the face.

How is it treated?

Some pyogenic granulomas may decrease in size spontaneously but they generally persist. Given their propensity to bleed and ulcerate, they are usually treated. A shave excision or curettage followed by electrocautery is usually sufficient. Non-surgical options in younger children include pulsed dye laser for smaller lesion, topical timolol or topical imiquimod. Pyogenic granuloma can recur even after excision. Any tissue removed by shave or curettage should be submitted to pathology to confirm the diagnosis, because worrisome lesions such as melanoma can mimic a pyogenic granuloma.

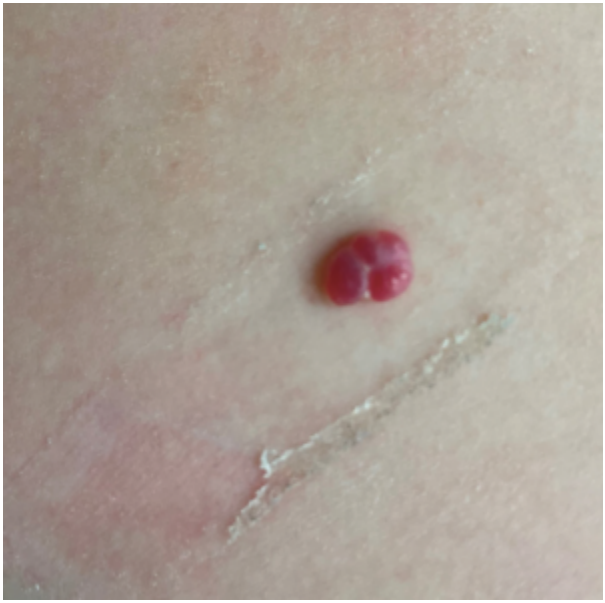

Image 7.10: Pyogenic granuloma: Lobulated and pedunculated vascular papule with evidence of bandaid use

Quiz

1. What type of laser is commonly used to treat capillary malformations?

a. CO2 laser
b. Pulsed dye laser
c. Excimer laser
d. Nd:Yag laser

2. Facial salmon patches tend to fade with age.

a. True
b. False

3. Pyogenic granulomas are often removed due to frequent bleeding.

a. True
b. False

4. Which of the following are possible associations with a port wine stain?

a. Sturge-Weber syndrome
b. CLOVES syndrome
c. Klippel-trenaunay syndrome
d. All of the above

5. What type of malformation is a port wine stain?

a. Capillary
b. Venous
c. Arterial
d. Lympathic

6. Lymphatic malformations can enlarge acutely in which circumstance?

a. With age
b. If the patient is given an NSAID
c. If the patient is sick
d. Spontaneously

7. In which gender do infantile hemangiomas occur most commonly?

a. Male
b. Female

8. Which of the following are treatment options for infantile hemangioma?

a. Watchful waiting
b. Topical beta blockers
c. Oral beta blockers
d. Any of the above depending on the circumstance

9. Which of the following is not an indication for treating infantile hemangiomas?

a. All hemangiomas require treatment
b. Potentially disfiguring mucus membranes
c. Head and neck (threatening vision)
d. Distal extremities (ulceration)

10. Parents of children with hemangiomas can be reassured that the birthmark will go away by kindergarten.

a. True
b. False

Answers: 1. B 2. A 3.A 4.D 5.A 6.C 7.B 8.D 9.A 10B*

*10: False, some hemangiomas require treatment to prevent complications, other hemangiomas will regress by kindergarten age, but often leave residual skin changes

CHAPTER 8: LUMPS AND BUMPS

Melanocytic Lesions: Acquired Melanocytic Nevi

What is it?

Melanocytic nevi are most commonly referred to as moles. The number of moles in any patient is related to their skin type, age, genetics, and sun exposure. Acquired nevi first appear in early childhood, increase in size and number into the third or fourth decade, and then slowly decrease in number with age. In childhood, fair skin colour, sun exposure and sunburns are associated with a higher number of moles. The biggest concern about moles from patients and their parents is the risk of melanoma and some are of cosmetic concern. The vast majority of nevi are benign. but acquired nevi may be a marker of an increased risk and very rarely a mole can be a precursor lesion to a melanoma.

What does it look like?

Acquired melanocytic nevi are classified by the location of the nevus cells in the skin. This classification system mirrors the natural history of a nevus from a junctional nevus early in life which develops into a compound nevi and then an intradermal nevus in later adulthood.

Junctional nevi: light to dark brown, hairless macules measuring 1mm-1cm diameter.

Compound nevi: skin-coloured to brown papules with smooth or rough surface – may have coarse hairs.

Intradermal nevi: soft, dome-shaped papules varying from skin coloured to brown – may also contain hairs.

How is it treated?

Acquired melanocytic nevi should be observed routinely by the child or parent for any new or concerning features. Any nevi with sudden unusual growth or bleeding, should be referred to dermatology for evaluation.

Special considerations:

- Nevi on the palms, soles and genitalia tend to retain a flat, junctional appearance throughout life.
- Scalp nevi are often larger than other acquired nevi, present at the part-line, and may have a fried egg or eclipse pattern
- Halo nevi are common in children and young adults. They appear as a central (usually pigmented) melanocytic nevus

with a peripheral halo between 1-5mm of hypo- or de-pigmented skin. Patients with halo nevi have an increased incidence of vitiligo.

- Blue nevi can be congenital or acquired. There are two subtypes – common and cellular blue nevi. They appear as blue-grey/black smooth papules or plaques with uniform color. They should be monitored for change, but are generally benign.
- Atypical or dysplastic nevi often appear in puberty, are somewhat larger than other acquired nevi, and have some pigment variability. Having multiple atypical nevi is thought to suggest elevated risk of developing melanoma.
- Familial atypical multiple mole-melanoma (FAMMM) syndrome is an autosomal dominant genetic syndrome characterized by multiple atypical nevi and an increased risk of melanoma and pancreatic cancer. Children with a family history of FAMMM syndrome should be seen regularly for a full cutaneous exam.
- Spitz nevi are a subtype of melanocytic nevi that occur primarily in children. On histology they share features of malignant melanoma but are a benign process. They appear as a solitary smooth, red-brown or dark brown to black dome-shaped papule. Occasionally a child will have multiple lesions, referred to as agminated spitz nevi. Because of their histologic similarity to melanoma, the diagnosis may be difficult or the lesion concerning. If Spitz nevi are not excised, they should be monitored.

Image 8.1: Acquired nevus: Tan macule with regular pigmentation

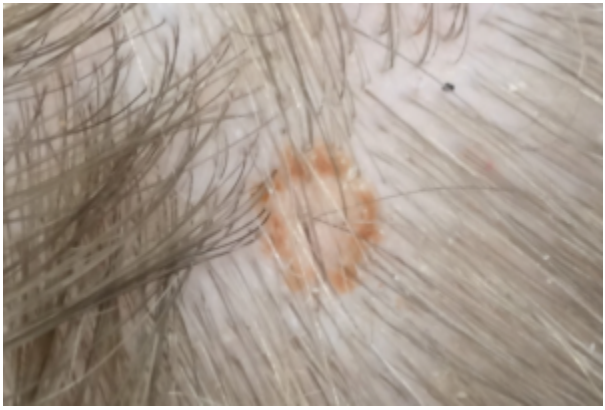

Image 8.2: Scalp nevus demonstrating eclipse pattern

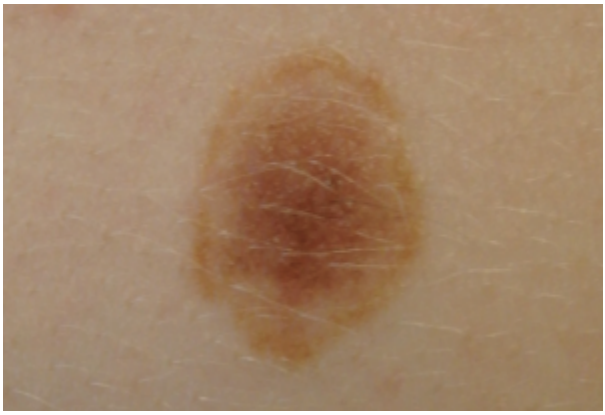

Image 8.3: Cockade nevus

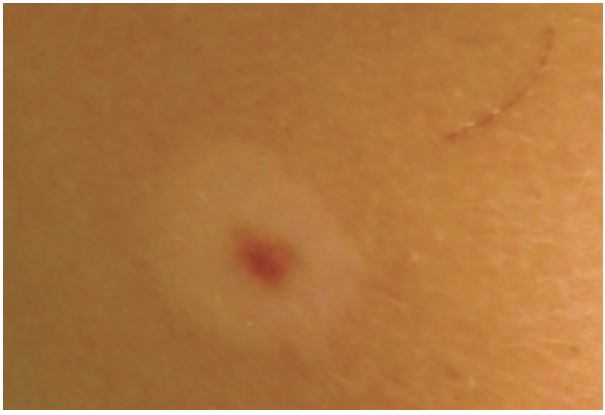

Image 8.4: Halo nevus depigmentation surrounding central benign nevus

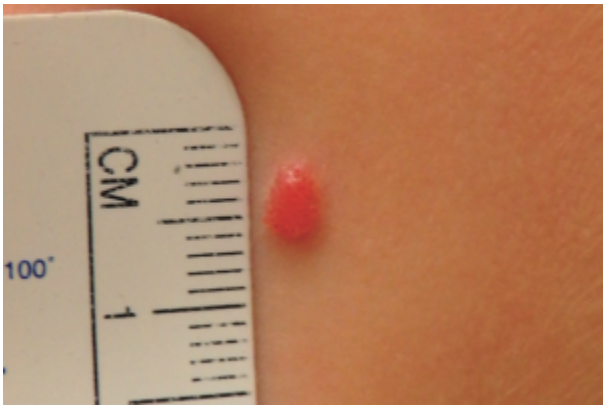

Image 8.5: Spitz nevus: Pink dome shaped papule on cheek of a young child

Melanocytic Lesions: Congenital Melanocytic Nevi

What is it?

Congenital melanocytic nevi (CMN) are present at birth although are sometimes first noticed later in the first year of life. CMN are classified by size according to their projected adult diameter. Small and medium CMN are significantly more common than large or giant.

Congenital melanocytic nevi classification:

Small: < 1.5cm

Medium: 1.5-20cm

Large: 20-40cm

Giant: >40cm

What does it look like?

CMN appear as light to dark brown papules or plaques that over time become thicker and develop dark, coarse hair within them. They may be speckled or have colour variation. Large and giant CMN can be associated with presence of smaller "satellite" nevi elsewhere on the body.

What are the possible complications?

The risk of melanoma in small and medium sized CMN is thought to be similar to the general population risk, and melanoma generally arises in adulthood. Large and giant CMN are thought to have an associated increased risk of melanoma, which is estimated at roughly 2-5%, and most often develops before the age of five. Many of these melanomas are deep or extracutaneous, such as in the CNS.

How is it treated?

The management of CMN is individualized for each patient. In any patient with a CMN a full cutaneous exam should be done to look for other nevi. Small and medium CMN do not need to be removed unless there is significant atypia. If they are of cosmetic concern, they can be excised. Patients with large and giant CMN should be referred for evaluation by dermatology, often in conjunction with plastic surgery, and need close follow-up. The decision to excise or debulk the lesion depends in individual factors. Children with multiple lesions are often referred for baseline MRI of the head due to the potential risk of CNS melanoma and neurocutaneous melanosis.

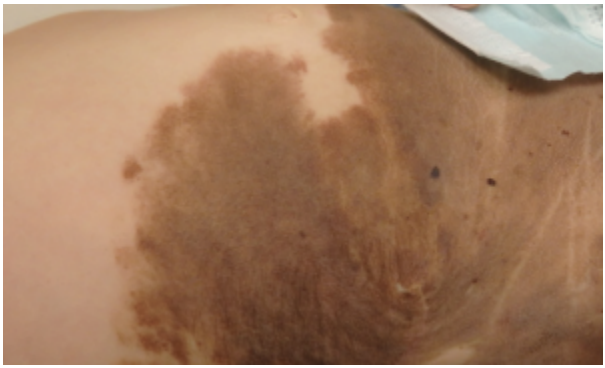

Image 8.6: Congenital melanocytic nevus with a few associated speckles

Melanocytic Lesions: Melanoma

What is it?

Melanoma is the most dangerous form of skin cancer. Fortunately, it is rare in children, with only 1-3% of all melanoma cases occurring under 20 years of age. The cause of melanoma is multifactorial with both genetics and environmental exposure (most importantly UV light) playing a role. Malignant melanoma is most common in light skinned patients. When melanoma occurs in patients with skin of colour it is usually on acral surfaces or the nail beds.

What does it look like?

In adults and older children, melanoma follows the ABCDE criteria. In children under 12, amelanotic melanoma is more common. These generally present with a new and growing pink papule that bleeds. They are often misdiagnosed and so any such lesion that is removed should be submitted to pathology.

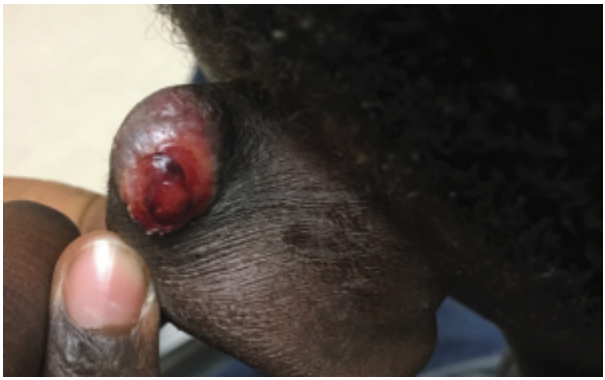

Image 8.7: Melanoma presenting as a bleeding papule

The ABCDE's of Melanoma in Adults:

Asymmetry: The colour is not uniform across the mole and the shape is not symmetric

Border irregularity: The mole has variable edges including scalloping or notches and may have projections growing off to the side.

Colour variability: The mole has multiple colours or shades of colour within it. It may be brown, black, red, white, or even blue.

Diameter: Mole is >6mm in width.

Evolving: Growing and changing in size or shape.

How is it treated?

A suspected melanoma should be biopsied with a 1-2mm clinical margin, but is likely to require re-excision of the pathology confirms melanoma. Fortunately, the prognosis for children with melanoma is generally better than that for adults. A complete work-up with by dermatology in conjunction with medical oncology is generally recommended to determine the need for systemic therapy and direct therapy choices. Close follow-up is indicated for patients with previous melanoma who are at highest risk of developing a second melanoma in the first 2-3 years after diagnosis.

Other Birthmarks

Nevus Sebaceus

Nevus sebaceous are common congenital lesions that mainly occur on the face and scalp. It appears as hairless, yellow or tan plaques with a verrucous or rough surface. They are usually present at birth and grow proportionately with the child until puberty when they may become much thicker and more verrucous. Nevus sebaceous generally does not require treatment but if the patient is bothered by the appearance or the lesion develops a localized growth within it, surgical excision is recommended.

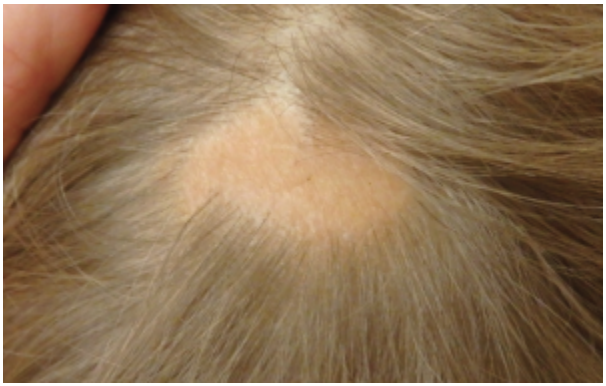

Image 8.8: Nevus Sebaceus: Linear hairless plaque with yellow hue

Epidermal Nevus

Epidermal nevi are benign congenital lesions that arise from hyperplasia (overgrowth) of the epidermis and superficial dermis. They present as tan to brown, velvety to verrucous papules or plaques. They are most commonly a single linear lesion that follows the lines of Blashko. Treatment of epidermal nevi is for cosmetic reasons and can be challenging. Complete surgical excision is effective but should be reserved for small, localized lesions. Superficial destructive therapies, such as cryotherapy, laser or electrodessication, are commonly followed by recurrence, but are helpful to debulk larger lesions. Epidermal nevus syndrome is the association of large or widespread epidermal nevi with non-skin changes.

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Becker's Nevus

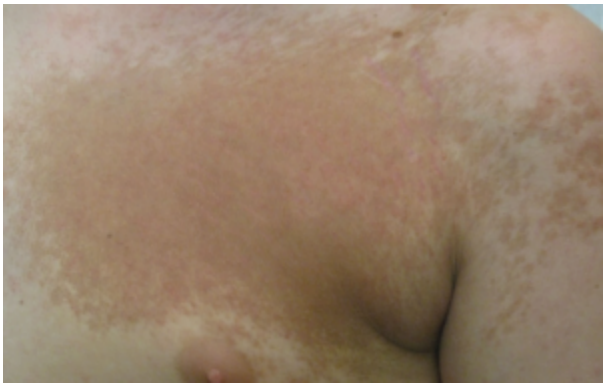

Image 8.9: Becker's nevus on the chest of a teenage body

A Becker's nevus, presents as an irregular, well-defined unilateral brown patch, characteristically on the upper trunk of teenage males. They can measure up to 15cm in diameter. The development of the hyperpigmented patch is followed by hypertrichosis (excess hair growth). After the initial appearance they may enlarge slowly for a 1-2 years but then generally remain stable in size. Becker's nevi are up to six times more frequent in males than in females.

Nevus Spilus

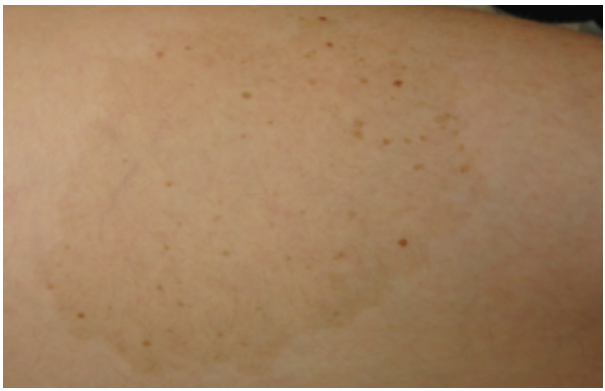

Image 8.10: Nevus spilus: Tan patch with overlying hyperpigmented macules

A nevus spilus appears at birth or early infancy as a tan to brown patch, similar to a café-au-lait macule, with eventual development of darker brown to black macules within it giving it a speckled appearance. They are usually a solitary lesion and range from 1cm up to 20cm in diameter. Nevus spilus does not require routine excision and can be observed. If any areas develop atypical features these should be excised.

Nevus Comedonicus

Nevus comedonicus is a birthmark that presents as a cluster of open and closed comedones on the skin, most commonly on the face, neck, trunk and upper extremities. These do not require treatment but if of cosmetic concern topical retinoids can be tried, or they can be surgically excised.

Flat Pigmented Lesions

Ephelides

Ephelides are more commonly known as freckles and are a marker of UV exposure. They are small 1-3mm light brown macules and occur only on sun-exposed skin in light skinned patients. During times of the year with low UV exposure such as winter ephelides tend to become lighter in colour.

Café-au-lait Macules

Café-au-lait macules (CALM) are named for their light brown colour resembling coffee with milk. They are round to oval light brown patches that vary from ~1.5cm up to 15cm in diameter. CALM are a common finding occurring in up to 1/3 of normal children. An isolated CALM is therefore a benign finding and no further workup or treatment is necessary. The finding of 5 or more CALM can be associated with a syndrome, such as neurofibromatosis, and suggests the need for further evaluation. Referral to opthalmology for evaluation of Lisch nodules is recommended in these cases. Referral to pediatrics, genetics and neurology should be considered for patients with confirmed NF1.

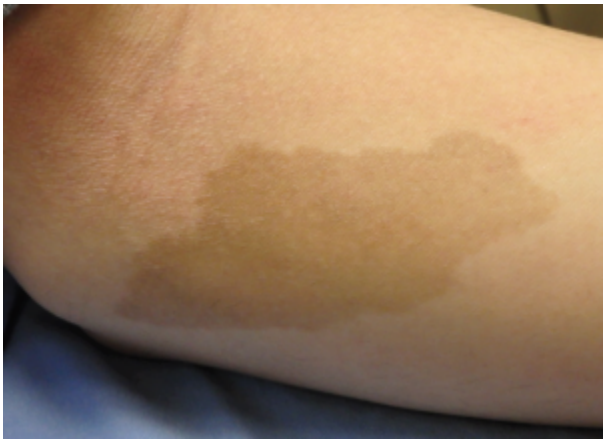

Image 8.11: Cafe au lait macule with uniformtan colour and sharp margins

Lentigines

Lentigines are small, sharply circumscribed macules or patches that can vary from tan, dark brown in colour. They usually begin to appear in childhood and increase in number into adulthood. They can occur anywhere on the skin or mucus membranes. Lentigines can in the mouth or on the lips, termed oral and labial melanotic macules respectively. Lentigines are generally darker than both ephelides and CALM. Lentigines are generally benign but can be associated with several syndromes including:

LEOPARD syndrome: (multiple lentigines, EKG abnormalities, ocular hypertelorism, pulmonary stenosis, abnormal genitalia, retardation of growth, and sensorineural deafness)

Carney complex: (multiple lentigines, blue nevi, and endocrine abnormalities and tumors)

Peutz-Jeghers syndrome: (localized mucocutaneous lentigines and intestinal polyps)

Juvenile Xanthogranuloma

Juvenile xanthogranulomas (JXG) is a non-langerhans cell histiocytosis seen most often in infants and young children. JXG tends to occur on the upper body and head and neck region as red-brown, dome-shaped papules that become more yellow in colour with time. When occurring as a solitary lesion these are regarded as benign and no further work-up or treatment is necessary.

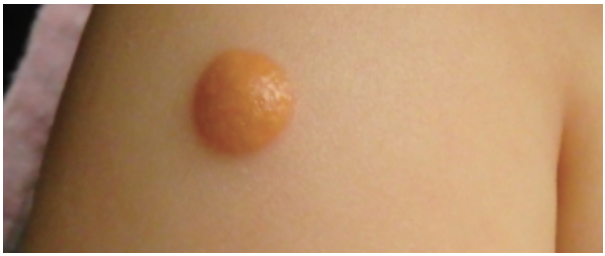

Image 8.12: JXG: Dome shaped nodule with yellow/orange hue on the arm

Mastocytosis

Solitary Mastocytomas

Solitary Mastocytomas are common in childhood. They present as pink to tan colored plaques with a peau-d'orange surface. They represent a collection of mast cells in the skin that will release histamine when triggered. They often develop surrounding erythema and an urticarial wheal, or even blister, with mechanical irritation (Darier sign). Mastocytomas usually resolve spontaneously over several years.

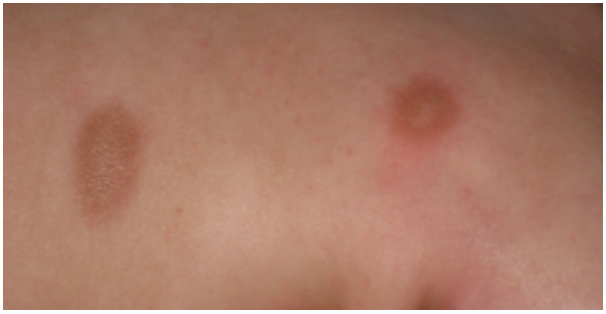

Image 8.13: Mastocytoma: Tan plaque with peau d'orange surface and positive Darier sign

Urticaria Pigmentosa

Urticaria Pigmentosa is another form of mastocytosis in childhood, which presents with multiple (sometimes hundreds) of pink to red-brown macules and papules. They tend to spare the palms and soles. Urticaria pigmentosa tends to develop in the first few years of life, but generally the lesions will spontaneously resolve by or during adolescence. Due to the increased number of mast cells in the skin, children with urticaria pigmentosa may develop systemic symptoms of histamine release, including pruritus, flushing and GI upset. It is important to avoid triggers of mast cell degranulation such as aspirin, NSAIDs, morphine and alcohol. In addition, exercise, heat and emotional stress can also be a trigger in some patients. Routine therapy with antihistamines and/or epipen might be recommended.

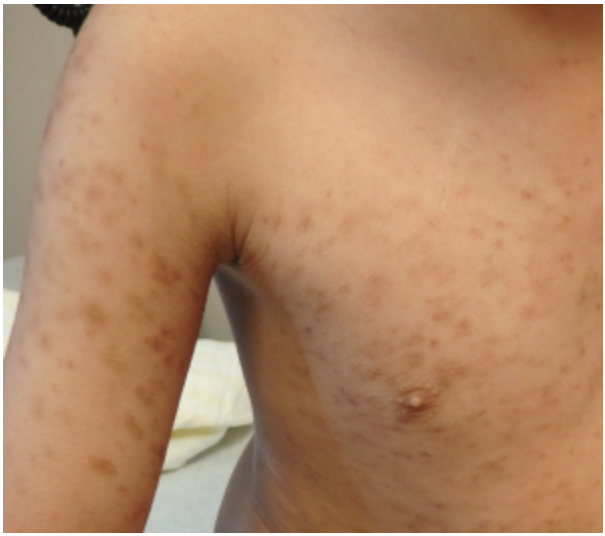

Image 8.14: UP: Hyperpigmented macules with soft edges that urticate when rubbed

Cysts

Epidermoid Cyst

Epidermoid (epidermal inclusion) cysts are the most common cutaneous cysts. They present as skin-coloured to yellow dermal nodules often with a visible punctum. They can be up to several centimeters in size and are most common on the face and upper trunk. Non-inflamed cysts are generally asymptomatic. If the cyst wall ruptures this can cause an intense, painful inflammatory reaction. Inflamed cysts can be treated with intralesional corticosteroid injection or may require incision and drainage. If superimposed infection develops oral antibiotics may be required. Surgical excision of an epidermoid cyst is curative but it is important that the entire cyst wall is removed or else the cyst can recur.

Dermoid Cyst

Dermoid cysts occur in infancy along embryonic fusion planes on the face. Most commonly these occur around the eyes along the orbital ridge. They present as a discrete, subcutaneous nodule measuring up to 4cm in diameter. These can be treated with excision after imaging to rule out underlying connection to the CNS.

Pilomatricoma

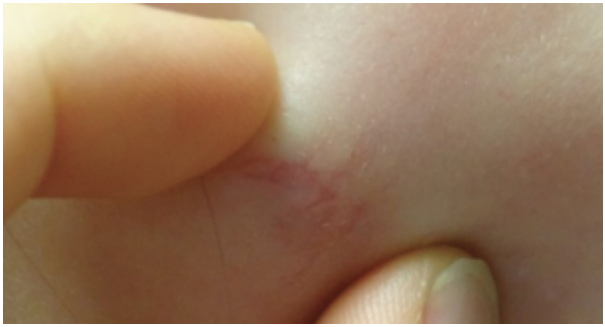

Image 8.15: Pilomatricoma: Hard dermal nodule with overlying telangiectasia

Pilomatricoma develops from hair matrix cells. It presents as a dermal nodule that is quite hard, due to calcium with the lesion. Pilomatricomas are usually solitary but can be multilobular and rarely people can have multiple. They often have a bluish hue over the surface. They are most often seen in young children especially on the head and neck. They are generally removed surgically as they do not go away by themselves.

Milia

Milia are very common superficial cysts that occur in patients of all ages. Milia present as 1-2mm, white to yellow papules, most commonly on the face. Milia may occur as a primary processes or as a secondary phenomena in response to trauma, cosmetic procedures, or blistering disorders. They are common in infancy and are seen in increasing frequency in people with Trisomy 21. Milia can be removed by incision with a needle or scalpel and expressing the milium, or by electrodessication and laser ablation. For multiple or recurrent milia topical retinoids may be helpful. (see Ch. 9 for a photo)

Dermatofibroma

Dermatofibromas are a benign growth thought to possibly occur in response to trauma but the exact cause is unknown. They appear as firm, rubbery skin coloured to hyperpigmented nodules with a characteristic “dimple sign” when the lesion is gently squeezed. No treatment is required.

Acrochordon (Skin Tags)

Acrochordon, more commonly known as skin tags, are soft, skin-coloured, fleshy papules with a narrow base. They are more common in middle-aged adults and have a higher frequency in obese patients or in association with acanthosis nigricans. They are often found at sites of friction such as the axilla, neck and groin. Skin tags can be removed with liquid nitrogen or by scissor excision (though slight bleeding may occur due to a feeder arteriole at the center).

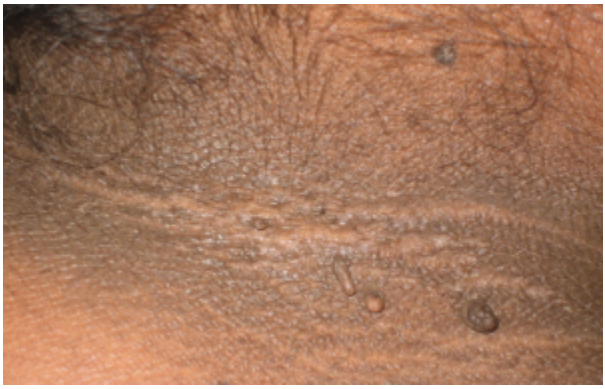

Image 8.16: Skin tags in an area of acanthosis nigricans

Idiopathic Facial Aseptic Granuloma

Idiopathic facial aseptic granuloma (IFAG) presents as a large, painless nodule on the cheek of children. Usually these are solitary lesions. It has been suggested that IFAG may be a childhood form of rosacea as there is an association with relapsing chalazions, facial telangiectasia, erythema, flushing and conjunctivitis.

Quiz

1. What skin condition is often associated with the development of skin tags?

a. Acanthosis nigricans
b. Atopic dermatitis
c. Herpes Zoster
d. Psoriasis

2. What feature does not influence the number of moles an individual develops?

a. Skin type
b. Sun exposure
c. Obesity
d. Genetics

3. Which of the following is the earliest stage of an acquired melanocytic nevus?

a. Compound
b. Junctional
c. Intradermal
d. Dermal

4. What is the risk of melanoma developing in association with a large or giant congenital melanocytic nevus?

a. <1%
b. 2-5%
c. 5-10%
d. 15%

5. How does melanoma most often present in children?

a. An existing mole becomes dark in colour and irregular in size.
b. A rapidly spreading brown patch
c. A new bleeding, skin coloured bump.
d. A rapidly growing black nodule.

6. What condition is associated with a halo nevus in children?

a. Vitiligo
b. Melanoma
c. Hypothyroidism
d. Iron deficiency anemia

7. A tan to brown patch with smaller black macules within describes a _____?

a. Nevus comedonicus
b. Becker's nevus
c. Cockade nevus
d. Nevus spilus

8. Lentigines fade during the winter months when there is less UV exposure.

a. True
b. False

9. Darier sign is a characterstic feature in which of the following?

a. Mastocytoma
b. Melanoma

c. Urticaria
d. Dermatofibroma

10. Where is the most common location of dermoid cysts?

a. The lumbar spine
b. Preauricular
c. Periorbital
d. Scalp

Answers: 1.A, 2. C, 3.B, 4.B, 5.C, 6.A, 7.D, 8. B, 9.A, 10.C

CHAPTER 9: GENODERMATOSES

Genodermatoses

Genetic mutations can lead to a wide variety of skin changes that reflect the nature and timing of the genetic mutation. Given the nature of skin embryology, the skin provides a remarkable window into the source of timing of such mutations. Some genetic skin alterations lead to only minor or cosmetic skin changes while others are severe and life-limiting. A complete review of genodermatoses is clearly beyond the scope of this manual and fortunately, the vast majority of such conditions are rare. Nonetheless, a few key points and conditions are worth mentioning.

Genodermatoses: Epidermolysis Bullosa

Epidermolysis bullosa (EB) is a family of blistering skin diseases in which the components of skin adhesion are not able to function properly due to genetic alterations.

EB is roughly broken into 3 classifications based on location of the resultant blister within the skin:

EB Simplex (EBS): Blisters form within the epidermis, most commonly due to mutations in keratins. Most cases of EBS are inherited in an autosomal dominant fashion or represent new mutations. Symptoms range from mild blistering on the hands and feet to much more widespread, but superficial blistering. Blisters tend to be worse in warm conditions. The most severe types can also be associated with significant itching.

Junctional EB: Blisters form with the dermal-epidermal junction due to alterations in the structural proteins in the basement membrane. Junctional EB is further divided into lethal and non-lethal forms, with lethal junctional EB having a life-expectancy of only about 1 year.

Dystrophic EB (DEB): Blisters for beneath the dermal-epidermal junction due to mutations in Collagen 7. Both dominant (DDEB) and recessive (RDEB) forms of dystrophic epidermolysis bullosa exist. Due to depth of blisters, these often heal with milia formation and scarring. Patients with RDEB have quite severe blistering that requires protection and frequent dressing changes. They are at risk of infection, severe pain, scarring, and eventually squamous cell carcinoma formation. Due to blistering of mucosa, they often have oral sores, challenges with dentition and the need for periodic esophageal dilation due to stricture formation.

Wound care is crucial for patients with EB and the appropriate plan for wound care depends on the phenotypic presentation of the disease. A multidisciplinary team, which might include

general pediatrics, dermatology, gastroenterology, dental, pain control and plastic surgery, is often helpful in providing the necessary care to affected children.

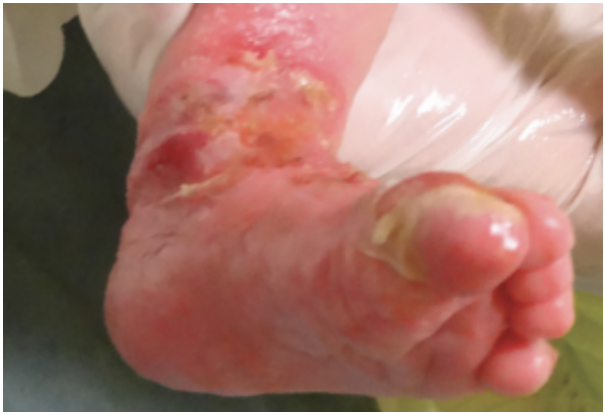

Image 9.1: Epidermolysis bullosa with blistering on the foot of a newborn

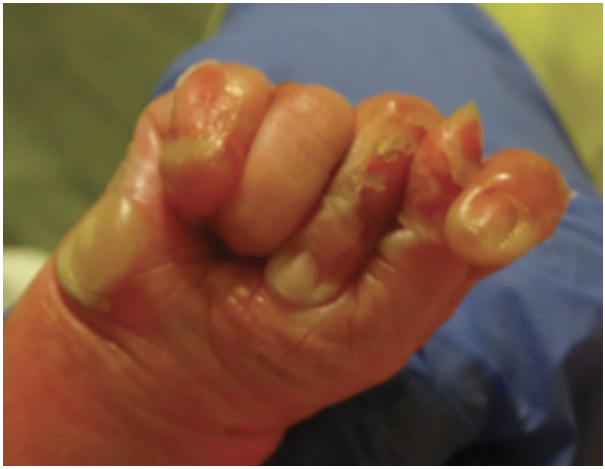

Image 9.2: Epidermolysis bullosa with blistering on the hand of a newborn

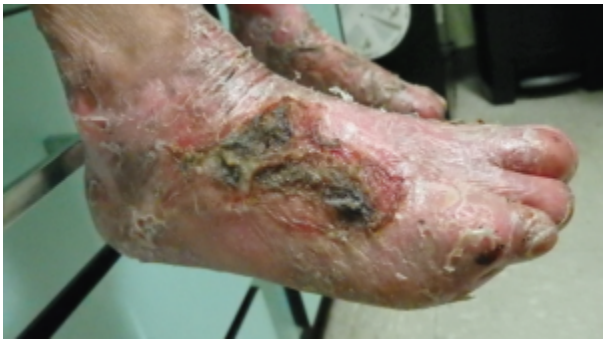

Image 9.3: Epidermolysis bullosa with chronic blistering and ulceration on the feet

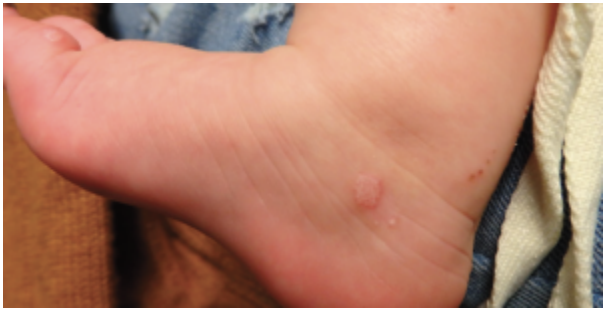

Image 9.4: Dystrophic epidermolysis healing with milia

Genodermatoses: Ichthyosis

Ichthyosis is a family of diseases that lead to dry, scaly skin. As with epidermolysis bullosa, there is a wide range of presentations for mild involvement to severe and life-threatening involvement. The mildest forms of ichthyosis are so common and mild, that they are usually only identified based on clinical examination and no further investigation is warranted. More severe ichthyosis presents at birth and rapid intervention is needed.

Types of ichthyosis include:

Ichthyosis vulgaris: Ichthyosis vulgaris is quite common and is caused by mutations in the Fillagrin gene. Patients have increased risk of atopy and present with dry skin especially over the shins. They may have associated hyperlinearity of the palms.

X-linked ichthyosis (XLI): XLI is seen only in boys and mothers are carriers. They may be born after prolonged labor, can have undescended testes, and may be found to have corneal opacities that do not affect vision. Skin changes lead to appearance of dirty brown skin with accentuation on extremities but sparing of the antecubital and popliteal fossa.

Autosomal Recessive Congenital Ichthyosis (ARCI): ARCI is uncommon and often presents with collodion membrane at birth. One form of ARCI presents with widespread erythema and fine scale. Another form of ARCI, commonly called lamellar ichthyosis, presents with large plate-like scale and may be associated with alopecia, ectropion and eclabium.

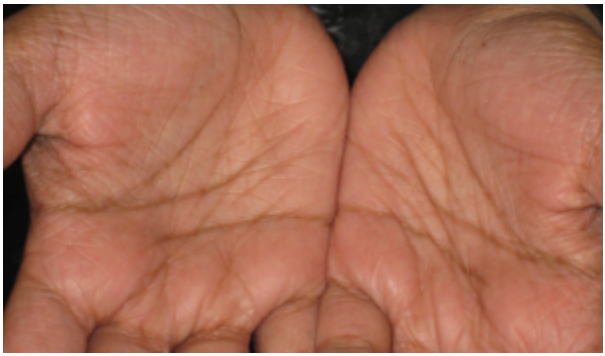

Image 9.5: Hyperlinear palms seen in ichthyosis

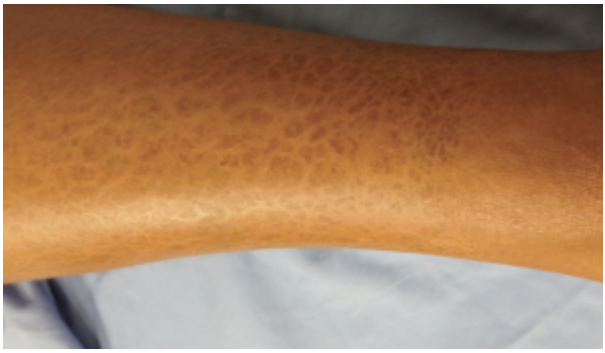

Image 9.6: Ichthyosis vulgaris with diffuse dry skin and brown scale

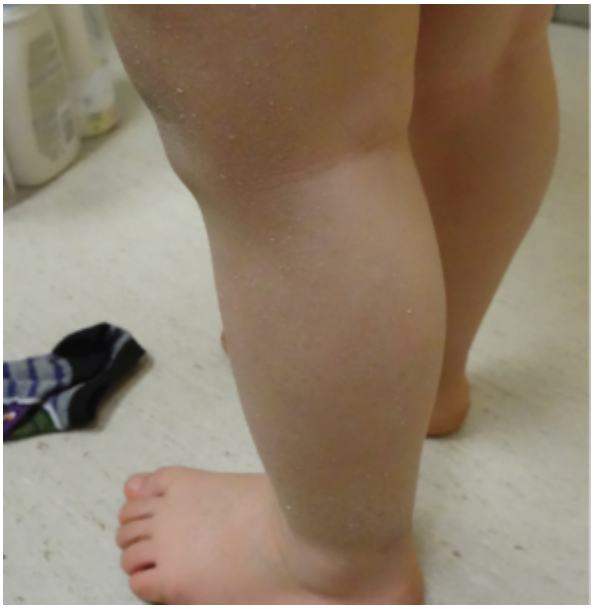

Image 9.7: XLI with diffuse scale
Note the relative sparing of the popliteal fossa

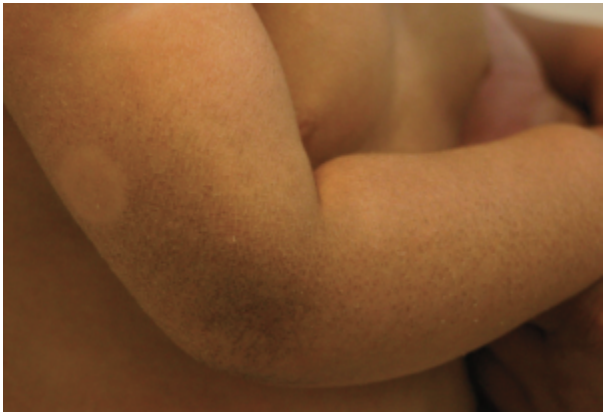

Image 9.8: X-linked ichthyosis with light brown scale

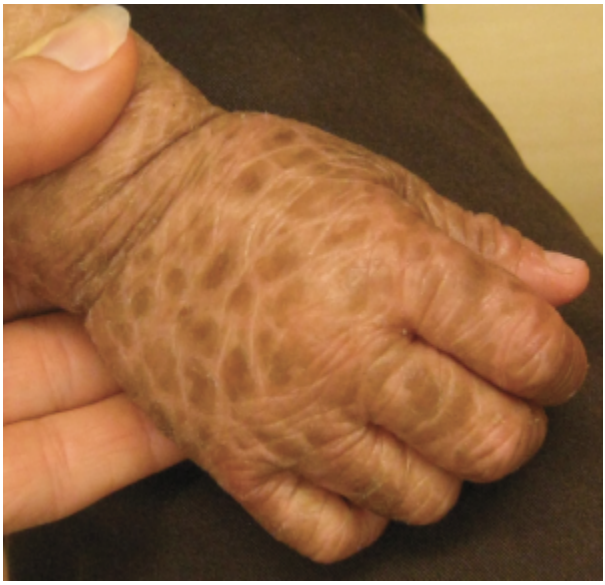

Image 9.9: Autosomal Recessive Congenital Ichthyosis, Lamellar type showing plate-like scale

Genodermatoses: Neurofibromatosis

Neurofibromatosis

Neurofibromatosis is caused by a mutation in the NF-1 gene that leads to changes in a number of body systems. Several dermatologic findings are among the disease criteria and patients with skin findings concerning for NF should be referred to ophthalmology for examination for Lisch nodules and optic glioma. Referrals to other specialties such as neurology and genetics depend on the presentation and symptoms.

Criteria for NF1 include:

- ≥≥6 café au lait macules >5mm in size in children and >15 mm in size in adults
- ≥2 neurofibromas or any plexiform neurofibroma
- Axillary or inguinal freckling
- Optic glioma
- Lisch nodules

A similar, but distinct, condition called Legius syndrome is caused by mutations in the SPRED-1 gene and leads to development of café au lait macules, but not neurofibromas.

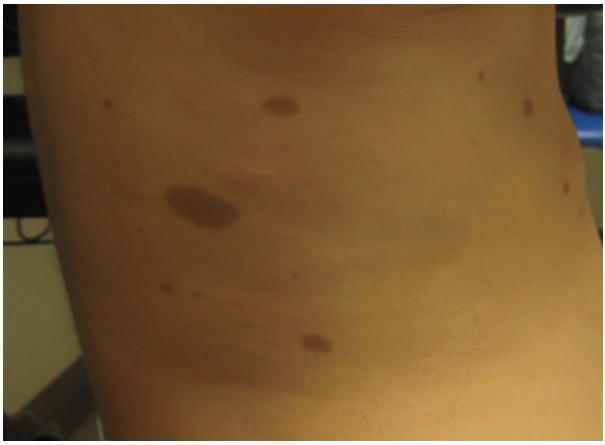

Image 9.10: Cluster of cafe au lait macules seen in segmental neurofibromatosis

Genodermatoses: Tuberous Sclerosis

Tuberous Sclerosis

Similar to NF-1, tuberous sclerosis presents with skin findings in addition to a constellation of other changes. It is caused by mutation in the TSC gene.

Selected criteria for TSC1 include:

Major criteria

- Hypomelanotic macules at least 5 mm in diameter
- Facial angiofibromas or fibrous cephalic plaque
- Periungual fibromas
- Shagreen patch (connective tissue nevus)
- Multiple retinal hamartomas
- Cortical dysplasias
- Subependymal nodules
- Subependymal giant cell astrocytoma
- Cardiac rhabdomyosma
- Lymphangioleiomyomatosis
- Angiomyolipomas

Minor features

- Hypopigmented macules (“confetti” macules)
- Dental enamel pits

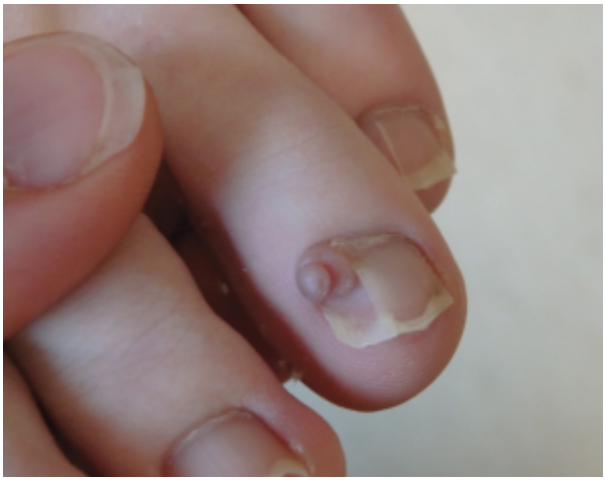

Image 9.11: Tuberous sclerosis: Periungual fibroma

Genodermatoses: X-linked Dominant Disorders

A few conditions present in X-linked dominant form. These conditions are generally only seen in girls as the mutations are usually lethal in developing boys who have only the affected copy of the X gene. Boys can rarely be affected if they have a post-zygotic mutation or have an XXY genotype. In girls, the skin findings often present with lines/swirls that represent lyonization, the process by which one X chromosome is activated in any given cell.

Incontinentia pigmenti is caused by mutations in the NEMO gene, which helps to regular apoptosis. There are 4 phases of IP that occur in the skin in roughly sequential order, though the path is not entirely linear and conditions such as illness may shift the skin toward an "earlier" phase.

1. Blistering
2. Verrucous plaques
3. Hyperpigmentation
4. Hypopigmentation

Children with IP may have ophthalmologic, neurologic and dental changes, so referral to these specialties is recommended.

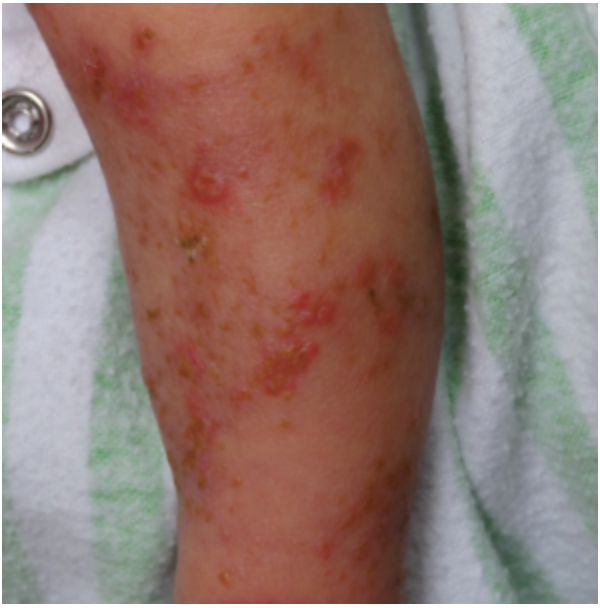

Image 9.12: Incontinential pigmenti: Vesicles in swirling pattern on the leg of a newborn

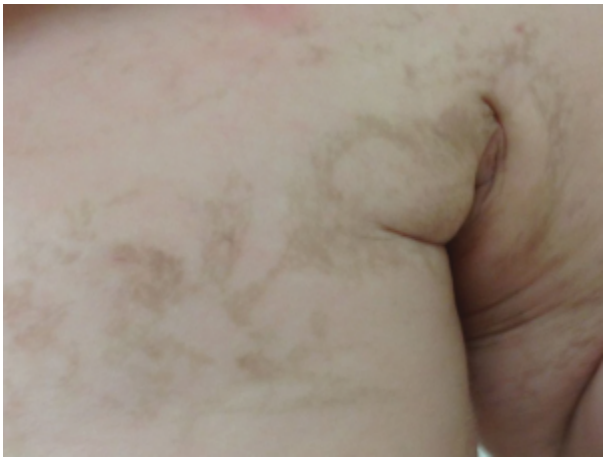

Image 9.13: Hyperpigmentation in swirling Blaschkoid pattern in the 3rd stage

Genodermatoses: Post-zygotic Mutations

Post-zygotic mutations occur after conception and lead to genetic change in only a subset of cells. Such changes are often referred to as mosaic conditions and often present on the skin with changes in a segmental pattern or in a Blashkoid pattern with lines and swirls. Examples of post-zygotic mutations include segmental NF, pigmentary mosaicism, and birthmarks such as port wine stains and epidermal nevi.

Quiz

1. Dystrophic Epidermolysis bullosa (EB) is due to alteration in which gene?

a. Keratin 1
b. Lamina densa
c. Collagen 7
d. Transglutaminase

2. What kind of skin does Ichthyosis lead to?

a. Blistering skin
b. Itchy skin
c. Dry, scaly skin
d. None of the above, skin is fairly normal

3. X-linked ichthyosis (XLI) is found in females.

a. True
b. False

4. Autosomal Recessive Congenital Ichthyosis (ARCI) is associated with which of the following?

a. Alopecia
b. Ectropion
c. Eclabium
d. All of the above can be associated with it.

Answers: 1. C, 2.C, 3.B, 4.D

CHAPTER 10: INFLAMMATORY SKIN CONDITIONS

Vasculitis

What is it?

Vasculitis is an inflammatory process involving the blood vessels. It is classified according to the size of the affected vessels and the type of inflammatory process causing the problem.

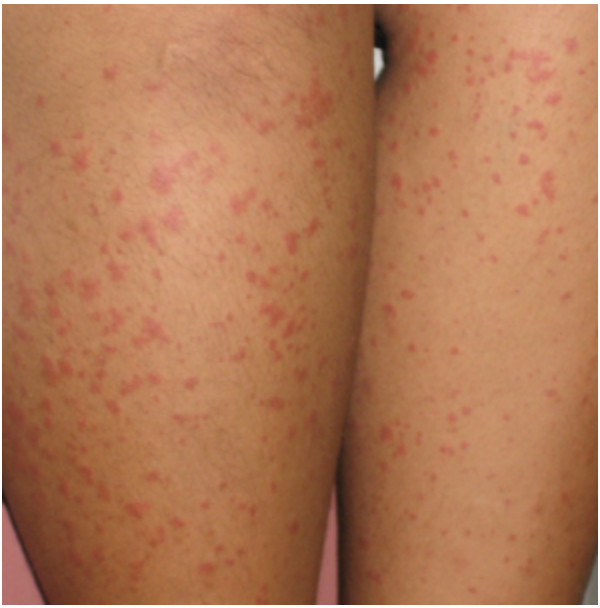

Image 10.1: Nonblanchable macules seen in small vessel vasculitis

What causes it?

The most common cause of cutaneous small vessel vasculitis (leukocytoclastic vascultitis) is a hypersensitivity reaction following an infection or exposure to a new medication. It may also occur due to an underlying malignancy or autoimmune condition such as lupus or rheumatoid arthritis. Often, a trigger is not identified. An inflammatory response targets the blood vessels and causes leakage of blood into the skin.

What does it look like?

Cutaneous vasculitis is most commonly seen on the lower extremities due to gravity. It presents with non-blanchable violaceous macules and papules often described as palpable purpura. They range from pinpoint to several millimeters in diameter and may be associated with mild edema of the ankles.

How is it diagnosed?

The diagnosis of vasculitis is often made clinically, but may be confirmed with skin biopsy of an early purpuric lesion (ideally one present for >24-48 hours). Direct immunofluorescence can be performed on a second biopsy to identify the type of inflammatory process causing the vasculitis. Underlying trigger can be identified by history and laboratory investigations such as CBC, throat swab, HBV/HCV/HIV serologies and ANA/ANCA. Urinalysis and creatinine should be checked to assess whether the kidneys are also affected.

How is it treated?

Treating the underlying infection or discontinuing any drug(s) suspected of eliciting the response is an important component of treatment. Rest and elevation of the legs is helpful. Systemic corticosteroids may be necessary for patients with ulcerations, diffuse involvement, or significant pain.

Other forms of vasculitis:

Henoch-Schönlein purpura (HSP) is a form of small vessel vasculitis that commonly occurs in children (<10 years old) and rarely in adults. It involves deposition of IgA immune complexes, which can be seen on direct immunofluorescence. HSP presents with a classic tetrad of palpable purpura (usually on the legs), abdominal pain, arthritis and hematuria. Renal involvement is particularly common (40-50%) but does not usually progress to chronic renal failure.

Polyarteritis nodosa, Takayasu arteritis and **temporal arteritis** affect medium or large vessels tend to present with different skin findings, such as subcutaneous nodules and ulcers.

Urticarial vasculitis is a form of small vessel vasculitis which presents with urticarial appearing lesions, but are painful/tender as opposed to itchy, leave behind bruise-like or hyperpigmented marks and last longer than 24 hours. Patients may also have systemic symptoms such as fever and joint pain.

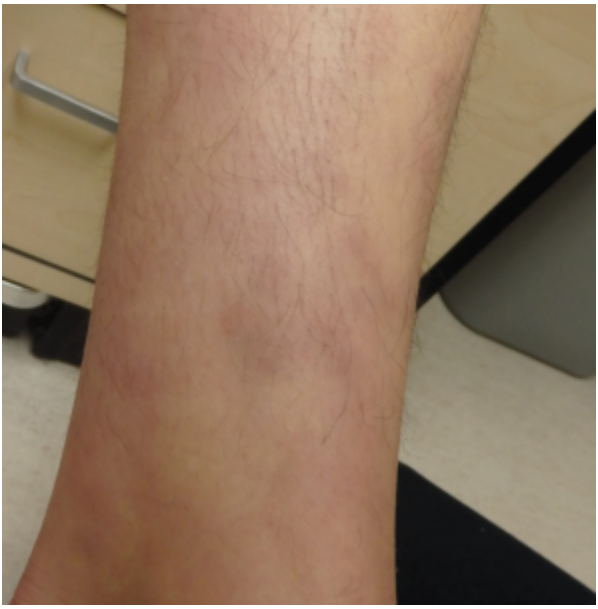

Image 10.2: Polyarteritis nodosa presenting with deep violaceous nodules on lower legs

Erythema Nodosum

What is it?

Erythema nodosum is the most common form of panniculitis – a term referring to inflammatory conditions affecting the subcutaneous fat layer.

What causes it?

Erythema nodosum is a hypersensitivity reaction which can develop due to a number of causes including infections, drugs, autoimmune disorders, pregnancy or malignancy. Streptococcal infections are the most common cause among children. In at least one third of cases, no cause is identified.

What does it look like?

Erythema nodosum presents with bilateral tender red nodules between 2-5cm. These are almost always on the shins but may rarely involve other areas such as the thighs and forearms. Due to their depth, the nodules may be difficult to see and are best appreciated by palpation. There may also be arthritis, fevers and malaise.

How is it diagnosed?

While the diagnosis can be made clinically in straightforward cases, a deep biopsy that includes fat may be necessary when the presentation is less obvious. Investigations to identify any underlying trigger may include: antistreptolysin O titer/throat swab, chest x–ray, CBC, beta-hCG and tuberculosis testing.

How is it managed?

The mainstay of treatment is identifying and treating any underlying cause if possible. Management is supportive and focused on alleviating pain through rest, elevation and compression. NSAIDs are first line treatment, but potassium iodide may be used. The lesions usually improve within 2 weeks but pigment change may last months.

What is the differential diagnosis?

Other forms of panniculitis should be suspected in cases of painful deep nodules which last longer than 6 weeks, are in a location other than shins, ulcerate, and etc. In these cases, the patient should be assessed by a dermatologist and will likely require a biopsy to clarify the diagnosis.

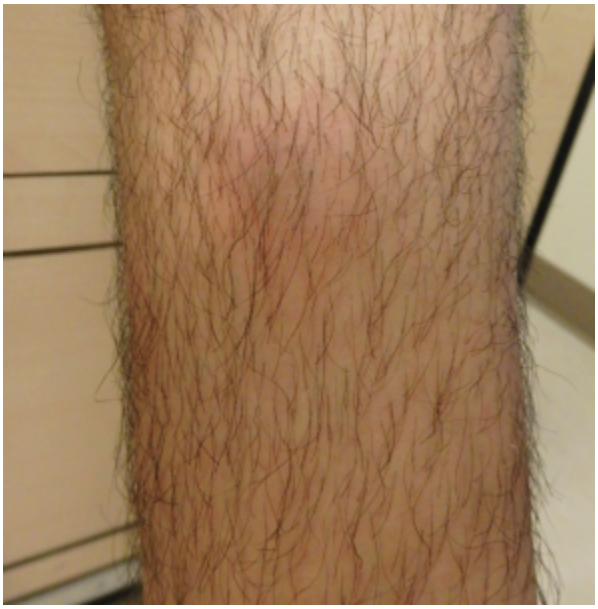

Image 10.3: Painful subcutaneous erythematous nodule as seen in EN

Cutaneous Lupus Erythematosus

What is it?

Cutaneous lupus describes a wide range of skin findings that may or may not be seen in association with systemic lupus. The frequency with which patients have or go on to develop systemic lupus varies widely depending on which type of cutaneous lupus they have:

Acute cutaneous lupus is almost always accompanied by systemic lupus (more than 90% of cases) and includes the classic malar ("butterfly") rash that most people associate with lupus. However, it may also manifest as a more widespread rash of red macules and papules on the trunk and limbs.

Subacute cutaneous lupus presents in a photodistribution (areas exposed to sunlight such as the face, neck and outer arms) and may be scaly and red (similar to psoriasis) or annular (lesions with a red rim with central clearing). Approximately 50% of patients with this form of cutaneous lupus will meet criteria for systemic lupus at some point in their life. Approximately 20-30% of cases are drug-induced, and may be caused by widely prescribed medications such as terbinafine, minocycline and hydrochlorothiazide.

Discoid lupus is a form of chronic cutaneous lupus. Only around 10% of these patients will have systemic lupus. It presents as scaly red plaques on the head and neck, which may scar leaving dyspigmentation and permanent hair loss. Commonly affected areas include inside the ear and on the nose and cheeks.

Neonatal lupus is seen in newborns due to placental transmission of maternal auto-antibodies against Ro, La and/or U1RNP. It is usually present at birth or shortly thereafter. It presents as round, red, scaly plaques typically located on the forehead and around the eyes. It can be associated with internal manifestations including heart block, liver disease and low platelets.

Non-specific skin findings such as photosensitivity, diffuse non-

scarring alopecia, Raynaud phenomenon and dilated blood vessels around the nails may all be seen with lupus but are also frequently seen in other connective tissue diseases such as dermatomyositis and systemic sclerosis.

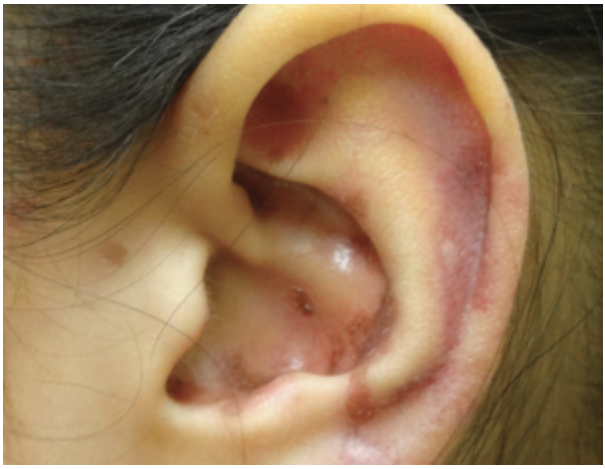

Image 10.4: Systemic lupus causing violaceous and atrophic plaques on the ear

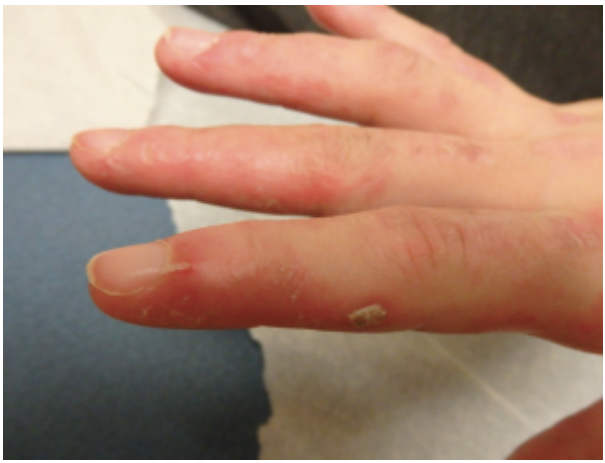

Image 10.5: Systemic lupus causing chronic changes on the fingers

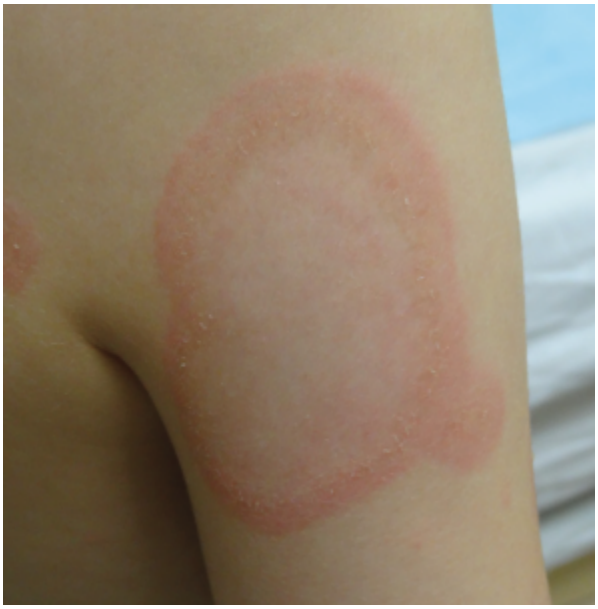

Image 10.6: SCLE: Annular plaque presenting in sun exposed area

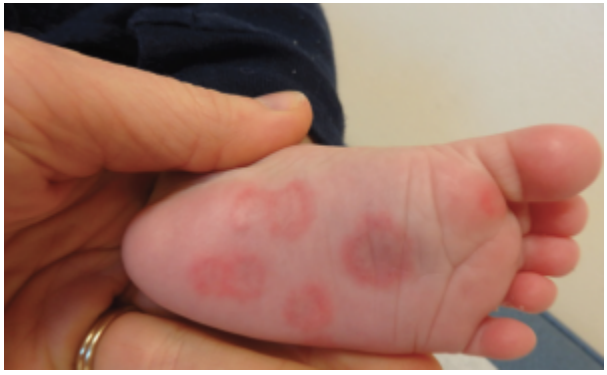

Image 10.7: Neonatal lupus with annular erythematous plaques on the foot of a newborn

How is it managed?

A history and physical (focusing on the signs and symptoms of connective tissue disease such as fevers, joint pain, oral ulcers, Raynaud phenomenon, hair loss, photosensitivity, neurologic symptoms) and laboratory work up (such as CBC, renal function, ANA/ENA, double-stranded DNA, complement levels, and skin biopsy) should be performed to investigate for systemic lupus or other autoimmune conditions.

Patients with all forms of cutaneous lupus are photosensitive and need adequate sun protection. Patients with **acute cutaneous lupus** are usually systemically unwell and should be managed in consultation with a rheumatologist.

Subacute cutaneous lupus is often treated with topical corticosteroids and/or calcineurin inhibitors. Hydroxycloroquine might be added as a systemic treatment.

Localized and mild forms of **discoid lupus** can often be managed with sun avoidance and topical or intralesional steroids. Topical calcineurin inhibitors may also be used to avoid prolonged use of topical steroids on the face. Extensive, severe or resistant cases can be treated with systemic agents such as hydroxychloroquine and corticosteroids.

What is in the differential diagnosis?

Subacute cutaneous lupus and discoid lupus might be confused with fungal infections or nummular eczema. The malar rash in SLE can be confused with rosacea or seborrheic dermatitis. **Dermatomyositis** is an autoimmune disease targeting the skin and/or muscle. There is a wide range of potential skin manifestations, many of which are non-specific and overlap with lupus. However, there are several findings which are more specific: the heliotrope sign describes purple discolouration of the eyelids sometimes accompanied by swelling; Gottron's papules are red to purple flat-topped papules affecting the dorsal hands, especially the skin over the knuckles (MCPs, PIPs and DIPs); capillary loops and drop-out can be seen at the cuticles. In adults, dermatomyositis is often associated with underlying malignancy but not in juvenile dermatomyositis, a distinct variant of this condition peaking at 8 years of age. The juvenile form also differs in that it frequently presents with calcinosis cutis: hard irregular nodules that form on the elbows and knees and may drain chalky material.

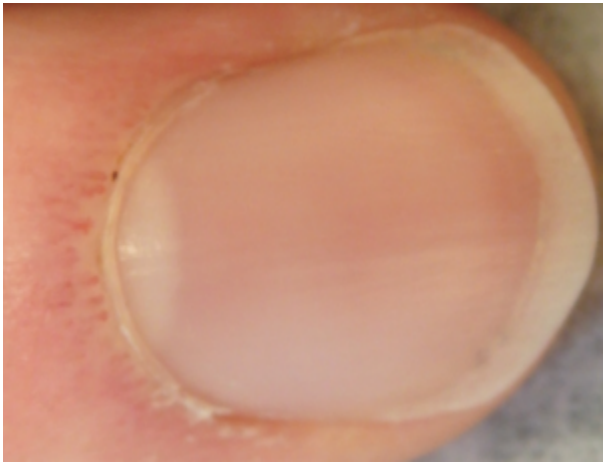

Image 10.8: Dermatomyositis with capillary loop changes

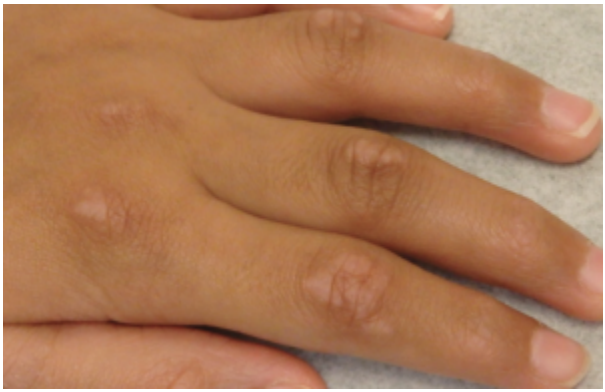

Image 10.9: Dermatomyositis: Pink papules over MCP, DIP and PIP joints

Morphea

What is it?

Morphea is an inflammatory condition that leads to hardening of the skin.

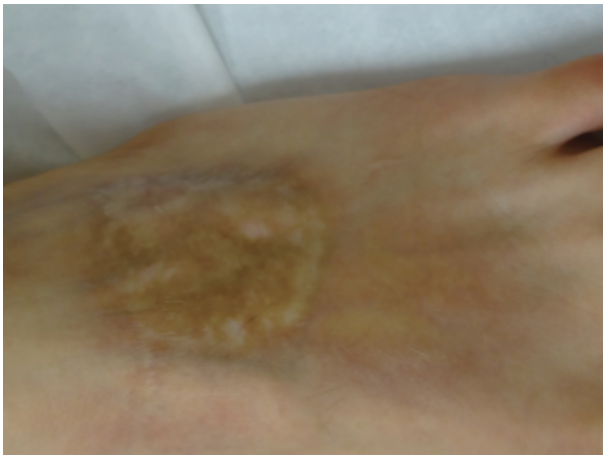

Image 10.10: Morphea: Hyper and hypopigmented plaque with atrophy and erythema in surrounding skin

What does it look like?

Morphea presents with indurated (hardened), atrophic (indented) plaques with pigmentary change. Active lesions are often red with a purple border, but older lesions tend to by hyper- or hypo-pigmented. Lesions may be oval (plaque type) or form a straight line (linear type) and the distribution varies from a single plaque to widespread disease. A common childhood variant is en coup de sabre in which linear morphea affects the forehead and/or frontal scalp.

How is it managed?

Localized morphea might be treated with potent topical steroids, but most patients are treated with systemic agents such as prednisone or methotrexate, or with a specialized form of phototherapy, UVA1.

Does it cause problems?

Morphea can lead to complications. If it crosses joints it may cause contractures. Significant facial involvement can lead to complications in the eyes, mouth and/or brain. There may be cosmetic considerations as atrophy can create noticeable asymmetry.

What is the differential diagnosis?

Systemic sclerosis is the systemic form of scleroderma. In this condition, hardening of the skin begins with the fingers (sclerodactyly), and spreads proximally. It is accompanied by other skin

findings such as Raynaud phenomenon, telangiectasia and calcinosis cutis. The lungs, heart, blood vessels and esophagus may also be affected.

Lichen sclerosus is another skin condition in which there is hardening of the skin. It most often affects the genital and perianal skin and is shiny white in colour (described as "porcelain" appearance) due to atrophy. If left untreated complications include fusion of the labia in women and phimosis in men. Treatment is with high-potency topical steroids and calcineurin inhibitors.

Urticaria (Hives)

What is it?

Urticaria (hives) is a vascular reaction that is caused by the release of histamine from mast cells. The histamine results in raised, red lesion with significant edema, usually causing significant pruritus. Most lesions resolve within 12 hours but new ones continue to appear. Urticaria is classified as acute if it lasts less than 6 weeks and chronic if it lasts more than 6 weeks after it is initially triggered.

What causes it?

The most common causes of acute urticaria are drugs (especially antibiotics), and infections (especially streptococcal and viral respiratory illnesses). Foods (especially eggs, milk, shellfish, nuts, and chocolate) may also be a trigger but only account for ~1% of acute urticaria. In the majority of cases of chronic urticaria, no trigger is identified, but a careful history and physical exam should look for signs of thyroid disease, connective tissue disease, infection, and chronic drug or food exposure.

What does it look like?

The classic lesion of urticaria is wheals: itchy, edematous, skin-coloured to pink lesions with a rim of pallor which come and go within 24 hours. Their size and distribution is variable. In children they may be annular and may have slightly dusky center. Wheals may be accompanied by **angioedema**, which is deeper swelling that typically affects the lips, tongue and skin around the eyes. This tends to be painful or tender as opposed to itchy and lasts 24-48 hours.

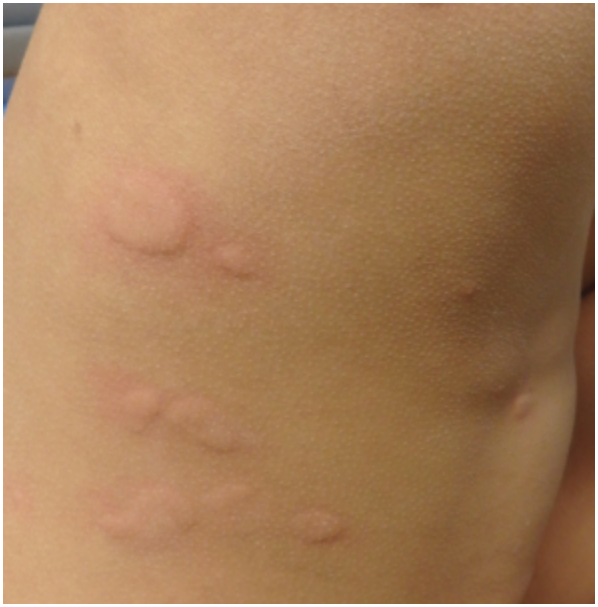

Image 10.11: Urticaria: Erythematous papules and plaques showing wheal and flare

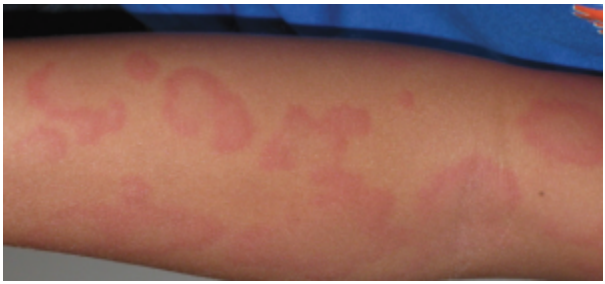

Image 10.12: Urticaria presenting in annular pattern

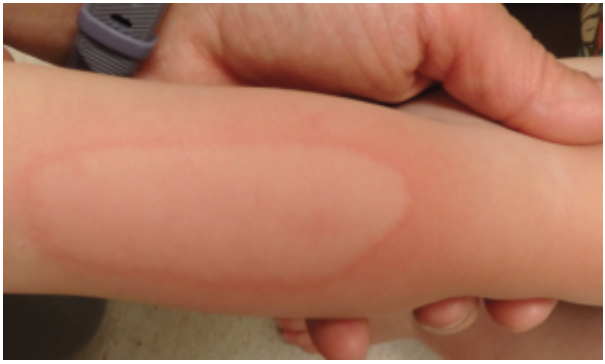

Image 10.13: Cold urticaria: Hive presenting after application of ice cube

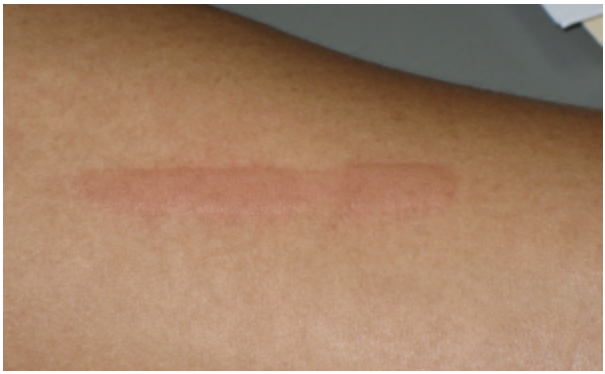

Image 10.14: Dermatographism

How is it treated?

The first step is trigger identification (if possible) and avoidance. In some cases this may be sufficient, but treatment with antihistamines is often required. For chronic urticaria, these are best taken daily for several weeks.

Physical urticarias (also known as inducible urticarias) are a distinct subgroup of chronic urticaria caused by an external stimulus. These are much less common than idiopathic or spontaneous chronic urticaria, and can usually be screened for on history and physical quite easily:

Dermatographism is a type of urticaria in which wheals appear after scratching or rubbing of the skin.

In **delayed pressure urticaria**, wheals appear 30 mins-12 hours after there is pressure on the skin such as from tight socks, shoes or waistbands.

Cholinergic urticaria is a condition in which wheals appear within 15 minutes of a sweat-inducing episode such as exercise, hot bath, or stress. It is usually seen on the upper trunk.

With **cold urticaria**, wheals appear after exposure to cold and can be eluted with the ice cube test. People with cold urticaria should be counseled not to jump into cold water.

Solar urticaria is rare and occurs within minutes of exposure to the sun (sometimes even through clothes). Headache and fainting may occur if the reaction is severe enough.

Aquagenic urticaria occurs after exposure to water of any temperature.

Granuloma Annulare

What is it?

Granuloma annulare is an inflammatory condition of unknown cause. It most frequently affects patient younger than 30 and is twice as common in women.

What does it look like?

Granuloma annulare may be localized or generalized. It presents as annular red to brown plaques with central clearing and no scale. It often occurs on the extremities, especially the hands, feet, elbows and ankles. It may be itchy or asymptomatic.

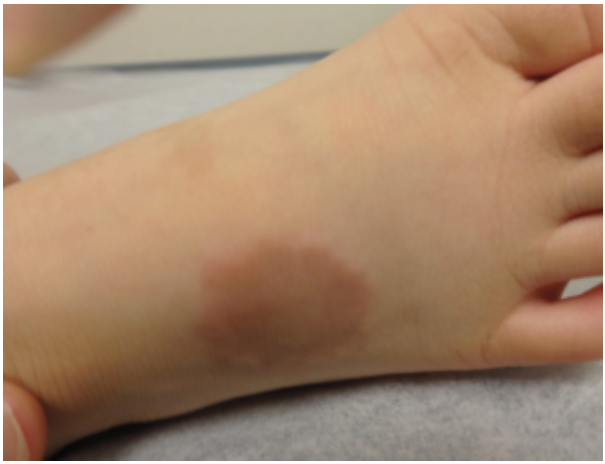

Image 10.15: Granuloma annulare: Erythematous annular dermal plaque with no surface change

How is it managed?

Granuloma annulare is a benign condition and resolves without treatment within a few months to years. Topical or intralesional steroids can be used. Other options include topical calcineurin inhibitors, phototherapy, systemic retinoids, dapsone and hydroxychloroquine.

What is the differential diagnosis?

Granuloma annulare is frequently misdiagnosed as tinea corporis (ringworm). The key distinguishing feature is the lack of scale in granuloma annulare. If needed, KOH prep can be attempted to look for the presence of fungi.

Pyoderma Gangrenosum

What is it?

Pyoderma gangrenosum is an inflammatory skin disorder presenting with ulcers.

What causes it?

The exact cause of pyoderma gangrenosum is not known. However, around half of cases are seen in association with systemic conditions, most commonly inflammatory bowel disease (Crohn disease and ulcerative colitis). Lesions are triggered by local trauma, which is known as pathergy.

What does it look like?

Pyoderma gangrenosum often starts at a site of minor injury such as a cut or scrape. It begins as a pustule which eventually ulcerates and is very painful. The ulcers of pyoderma gangrenosum have a classic appearance with a well-defined border with a rolled, deep-purple rim. It resolves with characteristic cribriform scarring (a lattice-like pattern).

How is it diagnosed?

Pyoderma gangrenosum is a diagnosis of exclusion. Other causes of ulcers such as infections, venous/arterial insufficiency, vasculitis, panniculitis, pressure sores and malignancy should be ruled out, which may require wound swabs and/or biopsy.

How is it managed?

Aggressive debridement should be avoided as this can trigger new ulcers to form. Smaller lesions can be treated with topical or intralesional corticosteroids and wound care. More severe cases may require systemic anti-inflammatory agents such as corticosteroids, cyclosporine or anti-TNF biologics.

Other Inflammatory Conditions

Erythema multiforme is a self-limiting hypersensitivity reaction that is most often triggered by herpes simplex virus. It classically presents with "target" lesions consisting of 3 concentric zones: a dusky centre that blisters or crusts, a pale pink middle layer and a red outer ring. Lesions commonly begin on the hands and feet but may become widespread and involve the lips and mouth.

Sweet syndrome is an inflammatory disorder that presents with painful red, edematous papules and nodules. Patients often have fevers malaise and elevated white blood cells. Sweet syndrome may be caused by a wide variety of triggers including infections, autoimmune conditions, drugs and malignancies (especially hematologic).

Quiz

1. How is vasculitis classified?

a. Size of affected vessels
b. Colour
c. Type of inflammatory process
d. None of the above
e. More than one of the above is correct

2. Where is vasculitis commonly seen?

a. Face
b. Hands
c. Lower extremities
d. Chest

3. Which of the following is a trigger for erythema nodosum?

a. Autoimmune disorder
b. Pregnancy
c. Infection
d. Drugs
e. All of the above are causes

4. Potassium iodide is the first line treatment or erythema nodosum.

a. True
b. False

5. Which of the following forms of lupus require necessary sun protection?

a. All kinds of lupus require sun protection
b. Neonatal lupus
c. Discoid lupus
d. Subacute cutaneous lupus
e. Acute cutaneous lupus

6. Morphea is an inflammatory condition that leads to:

a. Patchy skin
b. Blistering skin
c. Hardening skin
d. None of the above

7. What is the cause of granuloma annulare?

a. Infections
b. Foods
c. Drugs
d. It is unknown

8. Where are erythema multiforme lesions found?

a. Hands
b. Feet
c. Lips
d. Mouth
e. All of the above

9. Which of the following is not a trigger for acute urticaria?
a. Food

b. Infections
c. Drugs
d. Trauma

10. Granuloma annulare is twice as common in women.

a. True
b. False

Answers: 1. E (A &C), 2.C, 3.E, 4.B, 5.A, 6.C, 7.D, 8.E, 9.D, 10.A

CHAPTER 11: DRUG REACTIONS

Medication Reactions

Medications can cause a host of dermatologic problems ranging from contact dermatitis to life threatening hypersensitivity reactions. Medications may also be implicated in exacerbating underlying skin condition such as acne or psoriasis. A careful history of all medications and supplements taken in the past 6 weeks and physical exam can be helpful in determining the cause and severity of the reaction. When possible, discontinuation of the culprit medication is the first step in management. Sometimes this is not possible and the severity of the reaction must be balanced with the patient's need for the medication.

Morbilliform Drug Reaction

What is it?

Morbilliform drug eruptions, also known as maculopapular or exanthematous drug eruptions, are the most common form of drug eruption and do not cause any serious harm to the patient.

What causes it?

Antibiotics are the most common causes (especially aminopenicillins, cephalosporins and sulfonamides), but almost any medication can be responsible.

What does it look like?

The rash consists of widespread small blanchable erythematous macules and papules appearing 5-14 days after starting the medication. Pruritus is common.

How is it diagnosed?

Morbilliform drug rash is diagnosed clinically. Drug history should include any topical, over-the-counter, and natural health products the patient has been using. It might not be possible to definitively differentiate a morbilliform drug reaction from a viral exanthem or to know with certainty which medication was the culprit.

How is it treated?

Once the responsible medication is stopped, the rash typically resolves in 7-14 days. However, if a medication is considered necessary, you can "treat through" the rash as morbilliform drug eruptions are not life threatening. Topical corticosteroids and/or oral antihistamines are helpful for control of associated itch.

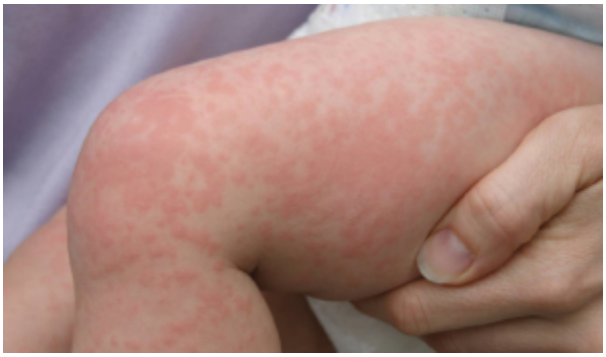

Image 11.1: Morbilliform eruption demonstrating diffuse blachable macules and papules

Severe Cutaneous Adverse Reactions (SCAR)

Concerning features that should prompt further investigation and consideration of a more serious drug eruption include fevers, swelling of the face and/or lymph glands, involvement of mucous membranes (such as the eyes, mouth and urogenital tract), blistering and skin pain (as opposed to itch).

Stevens-Johnson syndrome/toxic epidermal necrolysis is an uncommon but severe drug reaction with blistering. It often begins with "target" lesions similar to erythema multiforme, which then blisters as the epidemis detaches from the dermis. When this epidermal detachment affects more than 30% of the body surface area the condition is referred to as **toxic epidermal necrolysis**. Fever and flu-like symptoms may precede the eruption, which begins anywhere from 4-21 days after starting the drug. Mucosa (mouth, eyes, urethra, and/or vulva) are almost always involved. Sulfa drugs and aniepileptics are the most common culprits. Other causes include anticonvulsants, NSAIDs, and allopurinol. Management is as with burns: supportive care, nutritional/fluid support, and protection from infections due to loss of the skin barrier. Prompt discontinuation of the culprit medication is required. Treatments TNF alfa inhibitors seem most beneficial, but cyclosporin, IVIG and corticosteroids have also been used.

Drug reaction with eosinophilia and systemic symptoms (DRESS), also known as drug-induced hypersensitivity syndrome (DIHS), presents with fever and a rash. It also generally has a delayed onset – at least two weeks after initiating the medication. Laboratory abnormalities may include elevation of eosinophils, presence of atypical lymphocytes, elevated creatinine, and transaminitis. The rash itself looks similar to a morbilliform drug eruption but may be accompanied by facial edema, lymphadenopathy and lip cracking. Medications that commonly cause DRESS include antibiotics (trimethoprim-sulfamethoaxole, vancomycin, etc.), anticonvulsants (carbamazepine, lamotrigine, phenytoin, etc.) and allopurinol. In addition to discontinuing the responsible medication, patients should be seen by any relevant specialists if there is evidence of organ involvement, as they may require treatment with systemic corticosteroids.

Acute Generalized Exanthematous Pustulosis (AGEP) has a relatively rapid onset within 5 days after the culprit medication is started. The rash is distinct and consists of numerous small pustules on a background of erythema. Fever and elevated neutrophils and/or eosinophils are common. Beta-lactam and macrolide antibiotics are the most common cause. Management is similar to a morbilliform drug eruption: withdrawal of the medication and symptomatic treatment of itch. Most patients with AGEP improve quickly once the culprit medication is discontinued.

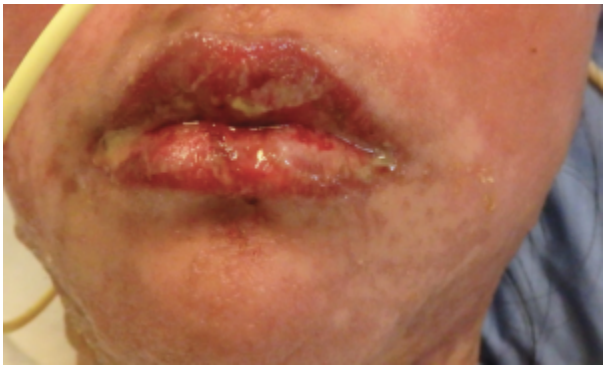

Image 11.2: SJS/TEN: Mucositis with widespread erythema and blistering on skin

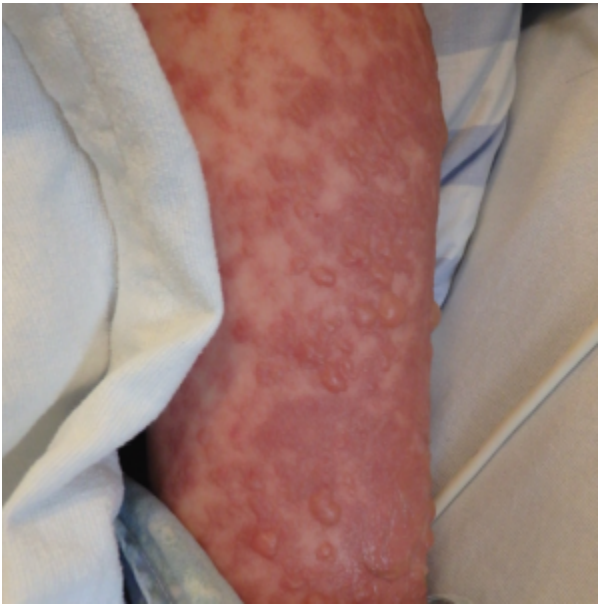

Image 11.3: Widespread erythema with overlying bullae due to sulfasalazine

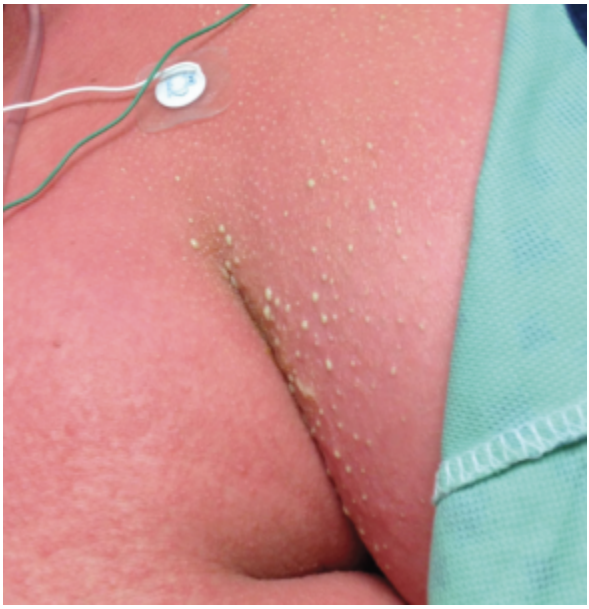

Image 11.4: AGEP: Non-follicular pustules overlying a background of erythema

Clinical features distinguishing morbilliform and more serious drug eruptions

	Onset	**Appearance of rash**	**Mucosal involvement**	**Systemic signs**	**Lab findings**	**Other clues**
Morbilliform drug eruption	5-14 days	Blanchable red macules/ papules	Absent	Mild fever	Mild eosinophilia	Overall, patient appears well
SJS/TEN	4-21 days	Target lesions: vesicles/blisters	Almost always (mouth, eyes common)	Prodrome of fever & sore throat	Lymphopenia	Skin pain/ tenderness as opposed to itch
DRESS	2-6 weeks	Blanchable red macules/ papules	Infrequent	Fever >38	Eosinophilia, abnormal liver and renal function tests	Facial edema, lymphadenopathy
AGEP	<5 days	Small pustules on background of erythema	Infrequent	Fever >38	Neutrophilia	Prominent in skin folds

Other Drug Eruptions

Fixed drug eruption is a localized drug reaction that occurs in the same spot every time a patient is exposed to the responsible medication. It presents as a painful red plaque, that can blister and often leaves behind darkened skin. With subsequent re-exposures to the medication, more sites may become involved. In addition to antibiotics such as tetracyclines and trimethoprim-sulfamethoxazole, common causes include NSAIDs and acetaminophen.

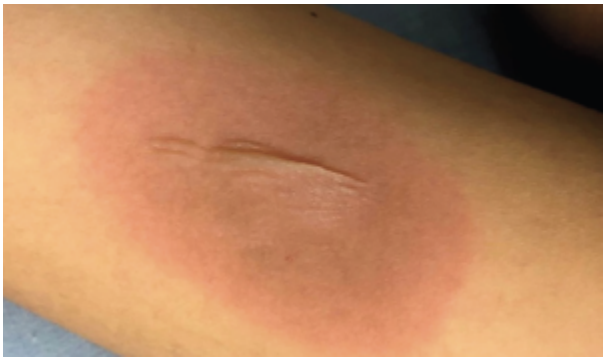

Image 11.5: Fixed drug eruption: Erythema, dusky center, and bulla formation

Drug-induced hyperpigmentation is the development of darkened skin due to medication use. Several medications can cause this including antimalarials (e.g. hydroxychloroquine), minocycline and certain types of chemotherapy. The colour changes range from brown to shades of blue and gray. The distribution might be widespread, occur in site of previous rash or scars, or favour sun-exposed areas. The nails may also be affected (melanonychia, see Ch. 14 for a photo). It typically resolves slowly after discontinuation of the medication. Luckily, the discolouration is only of cosmetic concern and does not cause any harm.

Drug-induced acne. Any topical ointment may lead to worsening acne by clogging pores. Inappropriate use of topical corticosteroids may also cause acne or rosacea on the face. Retinoinds can lead to a flare of acne when they are first started, so patients should be counselled that things might get "worse before they get better". Systemic corticosteroids, lithium, phenytoin and iodides (found in contrast media) are common causes of drug-induced acne in addition to those listed below. Anabolic steroids may also worsen acne. Drug-induced acne often presents with acute flare and monomorphous (all similar to each other) skin lesions. Sometimes systemic steroids lead to Malassezia folliculitis that resembles acne, but has no associated comedones. Psoriasis may also be triggered or worsened by certain medications including lithium, beta-blockers and antimalarials (e.g. hydroxychloroquine). Though TNF inhibitors are often used to treat psoriasis, in some cases, they may paradoxically cause it to flare when used to treat other conditions, such as inflammatory bowel disease.

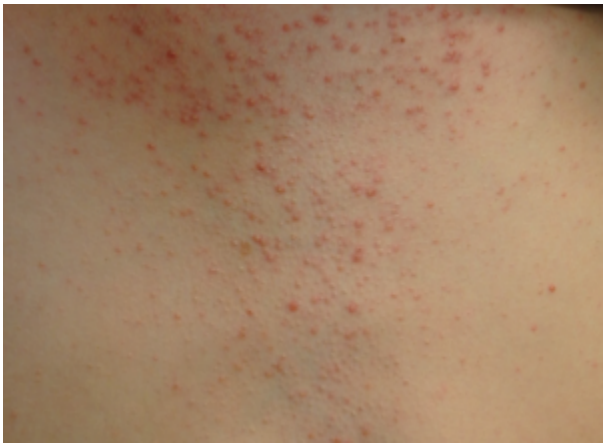

Image 11.6: Steroid induced acne with monomorphous inflammatory papules on the chest

Quiz

1. Which is the most common form of drug eruption?

a. Morbilliform
b. Toxic epidermal necrolysis
c. Drug reaction with eosinophilia
d. Acute generalized exanthemous pustulosis

2. How long after stopping medication does morbilliform rash resolve?

a. 5-10 days
b. 3-5 days
c. 7-14 days
d. 20-30 days

3. Mucosa rarely occurs in toxic epidermal necrolysis.

a. True
b. False

4. How does drug reaction with eosinophilia and systemic symptoms (DRESS) present?

a. Fever
b. Blister
c. Rash
d. None of the above
e. More than one of the above

5. What is a common cause of acute generalized exanthemous pustulosis (AGEP)?

a. Beta-lactams
b. Lithium
c. Iodides
d. Phenytoin
e. None of the above

6. What is the onset of Stevens-Johnson syndrome/toxic epidermal necrolysis (SJN/TEN)?

a. 4-21 days
b. 1-5 days
c. 20-30 days
d. 1-2 days

7. Morbilliform drug eruptions do not include mucosa.

a. True
b. False

8. Which of the following drug eruptions have systemic signs of a fever > 38 degrees Celcius?

a. DRESS
b. AGEP
c. SJN/TEN
d. Morbilliform
e. More than one of the above

9. Nails are never affected by drug-induced hyperpigmentation.

a. True
b. False

10. Morbilliform drug eruptions are life threatening.

a. True
b. False

Answers: 1.A, 2.C, 3.B, 4.E (A &C are true), 5.A, 6.A, 7.A, 8.E (A,B, and C can have fever), 9.B, 10.B

CHAPTER 12: SKIN PROBLEMS CAUSED BY THE ENVIRONMENT

Sun-Induced Conditions: Sunburn

What is it?

Sunburn is an acute cell injury caused by exposure to UV radiation. Exposure to UVB rays causes erythema beginning 6 hours after exposure and peaking 12-24 hours after exposure. The amount of skin damage is proportional to the amount of UV exposure received. Patients experience pain and/or pruritus and, in severe cases may develop blisters. Peeling after a sunburn is common even in patients who did not experience blistering. Natural pigments are protective against sunburn. Skin types are determined according to the ability of the skin to withstand UV radiation, and loosely correspond to colour, but there is significant variability and skin type is not a proxy for skin colour or race.

Type 1: Always burns, never tans, often freckles

Type 2: Often burns, can tan with long slow exposure to sunlight

Type 3: Tans after initial burn

Type 4: Tans easily but might burn

Type 5: Tans easily rarely burns

Type 6: Becomes darker after sun exposure and very rarely burns

How is it treated?

Prevention is key. Sun protection includes wearing hats, long sleeves and pants, and seeking shade; this with the use of sunblock is the ideal way to protect from sun damage. This is particularly important for lighter skin types, but since all skin types can burn and burn increases risk of developing melanoma, following these precautions is wise for anyone who anticipates significant sun exposure.

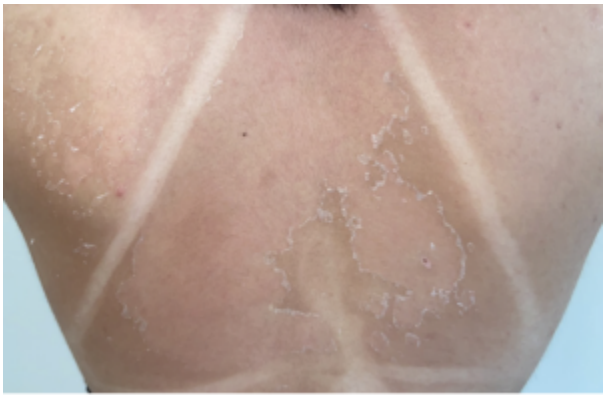

Image 12.1: Well demarcated erythema and spared skin in acute sunburn

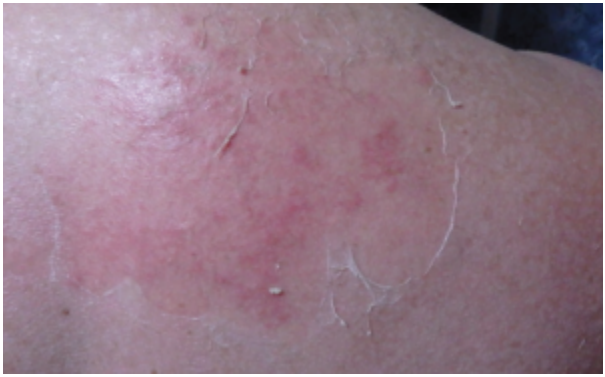

Image 12.2: Diffuse peeling after sunburn

Sun-Induced Conditons: Polymorphous light eruption (PMLE)

What is it?

Polymorphous light eruption (PMLE) is the most common form of light sensitivity. It is a delayed hypersensitivity reaction triggered by UV light and presents hours and even days after sun exposure on sun exposed areas. It is most frequently seen in teenage girls and improves with age. It is seasonal and usually occurs after exposure to the first strong sun in spring or early summer.

What does it look like?

PMLE is called polymorphous because it can have different appearances in different people. Most typically, it presents with papules and papulovesicles on sun-exposed areas such as the dorsal hands, forearms, neck and face. It is associated with stinging and itching. A variant of PMLE called "juvenile spring eruption" is most frequently seen on the ears of schoolage boys.

Can it be treated?

It is prevented by protection from exposure to UVA radiation through the use of sunscreens, long clothing, and shade seeking. Symptomatic treatment with topical corticosteroids can be helpful. It tends to improve over the course of summer due to "hardening" of the skin, so some patients choose to undergo light therapy to prevent flares. In severe cases, hydroxychloroquine might be helpful.

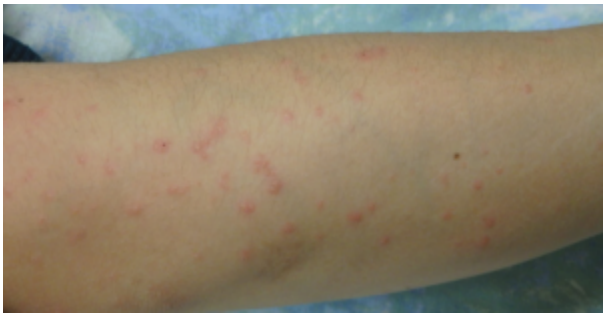

Image 12.3: PMLE: Erythematous papules in sun exposed areas

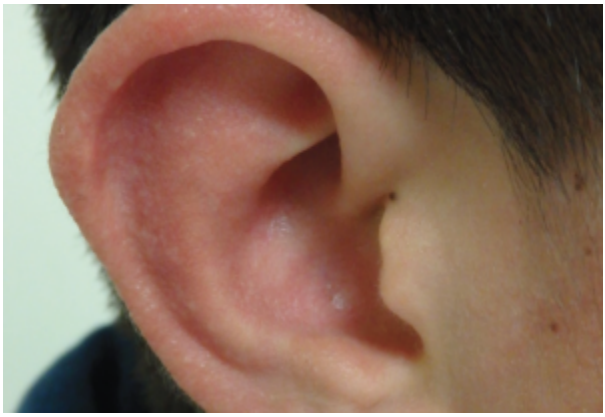

Image 12.4: Juvenile skin eruption: Erythema and small vesicles on the helix

Sun-Induced Conditions: Phytophotodermatitis

Phytophotodermatitis is a reaction that occurs after exposure to plants and ultraviolet radiation.

What does it look like?

Phytophotodermatitis appears as red vesicles and even blisters that leave profound postinflammatory hyperpigmentation in bizarre shapes where the skin touched the offending plant. Linear lesions and swirls are quite common. Common triggers are lemon, lime, hogweed and fig.

How is it treated?

No treatment is necessary. Over time, the hyperpigmentation will fade.

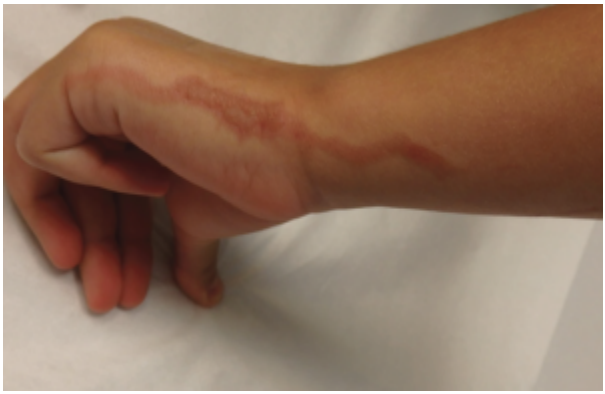

Image 12.5: Phytophotodermatitis: Hyperpigmented and blistering line in bizzare pattern at site of contact with lime juice and sun

Cold-Induced Conditions: Raynaud's Disease

What is it?

Raynaud's is caused by vasoconstriction of small arteries in the fingertips and toes on exposure to cold. It can be primary or secondary to underlying rheumatologic problem.

How is it treated?

Prevention is through keeping the hands and feet warm with gloves, socks and appropriate footwear. Keeping the core body warm is also helpful. For those with significant symptoms, vasodilators such as nifedipine can be helpful.

What does it look like?

Affected fingertips and toes turn white and/or blue in the cold and rewarm with erythema. The fingers may feel numb at the time.

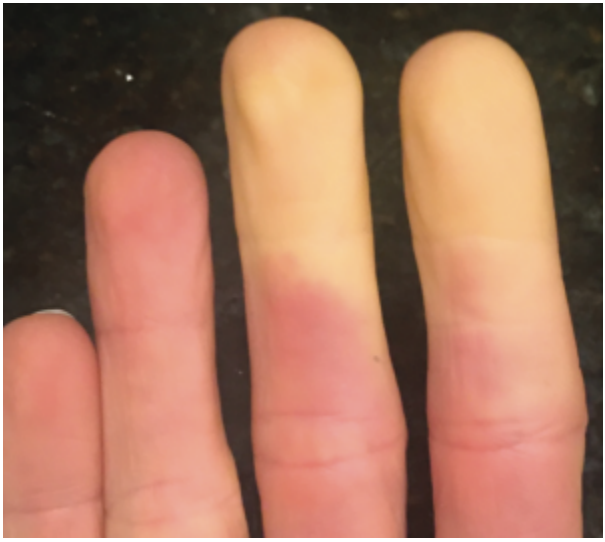

Image 12.6: Raynauds: White discolouration of finger tips due to cold-induced vasoconstriction

Cold-Induced Conditions: Chilblains/ Pernio

Chilblains or pernio is term to describe an abnormal inflammatory response in acral skin (fingers and toes) in response to cold exposure. It mainly occurs in areas that have a cold and damp weather.

What does it look like?

The lesions present on the fingers and toes with multiple red-blue papules, nodules and erosions. It is often initially painful then becomes very pruritic.

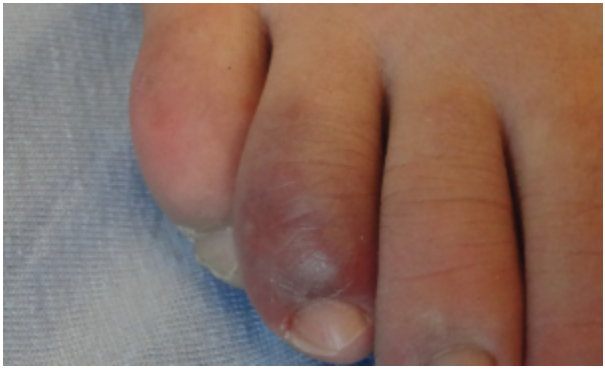

Image 12.7: Chilblains: Tender violaceous plaque on distal toe

How is it treated?

Prevention is through avoidance of exposure to cool damp weather and wearing proper clothing. Treatment of active lesions is mid-high potency topical corticosteroids. Those who suffer recurrent episodes may be treated with calcium-channel blockers such as nifedipine.

Cold-Induced Conditions: Frostbite

Frostbites result from exposure to sub-freezing temperatures can lead to cell death. It mainly involves acral areas including the fingers, toes, ears and the nasal tip. Drug and alcohol use may predispose to developing frostbite as they may decrease sensation of affected areas and lower heat-seeking behavior.

The severity of frostbites is classified like burns:

- first-degree frostbite is called frost nip and resolves completely with no scarring;
- second-degree causes blistering and neurological sequelae may persist;
- third-degree and fourth-degree carry poor prognosis.

Gentle rewarming with warm water is the intervention of choice for frostbite, but should not be carried out until the skin can be maintained warm to prevent worse damage from freeze-thaw cycle. Wound care management and pain control are other aspects of management for frostbite.

Cold-Induced Conditions: Cold Panniculitis

Cold panniculitis is a form of injury to the lobules of fat that occurs after exposure to extreme cold. It is seen in several forms including subcutaneous fat necrosis of the newborn, popsicle panniculitis, and panniculitis after use of cold-packs for injuries. The tendency of fat to develop cold panniculitis is utilized therapeutically in the fat reduction technique of cryolipolysis.

Bites and Stings: Arthropod bites

What are they?

Arthropods are a phylum that includes arachnids and insects. Many arthropods such as mosquitoes, fleas, ticks and flies bite humans as a source of nutrition or as a part of their life cycle. Arthropod bites themselves can also cause significant morbidity, particularly in children, where they can lead to exaggerated reactions with formation of blisters and intense itching. Scratched lesions may become secondarily infected leading to impetigo, abscesses, or cellulitis. It is often difficult to tell the source of the bite based on clinical morphology, but there are a few clues. Bed bug bites typically occur overnight on exposed skin and are clustered. Flea bites are commonly seen on lower extremities, but more widespread on toddlers who are closer to the ground. Mosquito bites are most prevalent on exposed skin, and chigger (larval mites) lesions are most prevalent on covered skin. In endemic areas, arthropods may also carry diseases such as malaria, zika, and dengue that are transmitted by mosquitos, as well as rickettsial illnesses including **Rocky Mountain spotted fever** (caused by Rickettsia rickettsii) and **Lyme disease** (caused by Borrelia burgdorferi) that are transmitted by ticks.

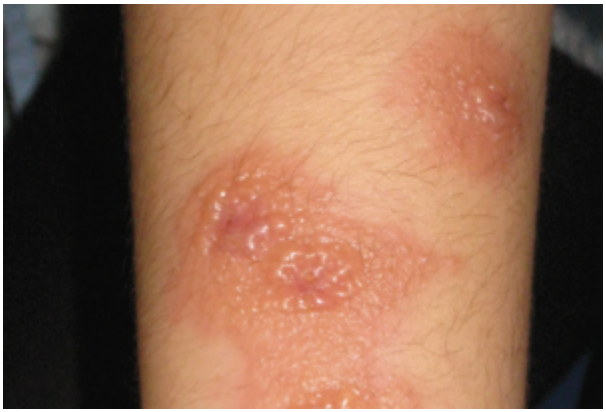

Image 12.8: Bullous arthropod bites demonstrating individual pruritic lesions with central blister and surrounding erythema

How can bites be managed?

Prevention of bites is an important part of management. The approach is multifaceted and can include: use of repellants containing DEET (with concentration less than 10% for use in pediatrics), avoidance of areas where it is clear bites are occurring and treatment of pets for fleas and ticks. Thorough inspection of the household and/or use of professional exterminators should be considered in cases where the bites are suspected to be occurring at home. In terms of the bites themselves, oral antihistamines can lessen the itching and application of a topical corticosteroid mixed with antibiotic ointment can decrease the reaction while preventing secondary infection that may occur after scratching.

Bites and Stings: Papular Urticaria

What is it?

Papular urticaria is a name for the reaction pattern seen with multiple insect bites, but also can occur due to contact with caterpillars, carpet beetles, and mites for whom humans are not a usual host.

What does it look like?

Papular urticaria presents with widespread small 1-2mm individual erythematous pruritic papules, many of which might be excoriated.

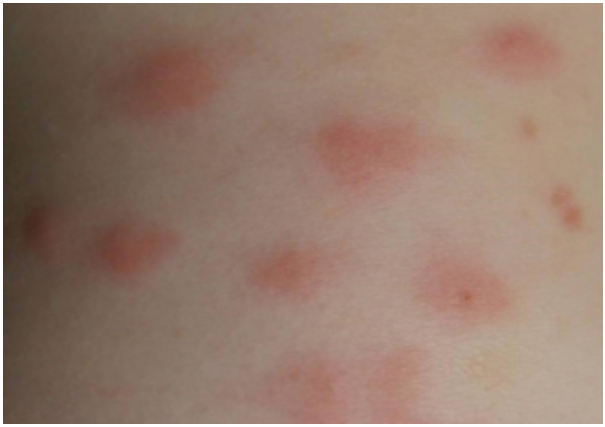

Image 12.9: Urticarial papules seen after exposure to caterpillars

Bites and Stings: Sea-jelly stings

Sea-jelly stings

Sea-jellies contain stinging cell that fire on contact with other animals. The stinging mechanism (nematocyst) is at the end of the tentacle and contact with the tentacle causes pain, redness and blistering in skin. Treatment is with removal of any attached tentacles, rinsing with sea-water and soaking in 5% acetic acid to prevent further firing of the nematocyst. Larvae of sea jellies can also become trapped under swim clothing and fire into the skin leading to sea bather's eruption. Rapid removal of clothing followed by rinsing can help minimize symptoms. Rinsing with fresh water or towelling vigorously can worsen them.

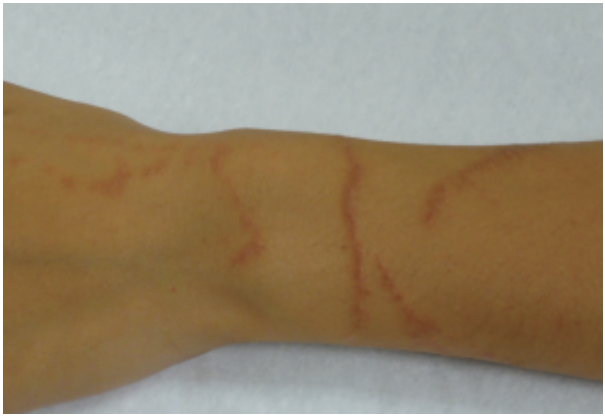

Image 12.10: Sea jelly stings causing linear vesicles in whip-like pattern

Bites and Stings: Swimmers Itch

Swimmers Itch

Swimmers itch is caused by contact with larva of schistosomes that burrow into the skin and then die because humans are not the intended host for the lifecycle. It occurs on exposed skin (as opposed to sea-bathers eruption that is on covered skin) after swimming in water containing the larva. It is seen most commonly after freshwater exposure where birds and snails are plentiful as they are important in the lifecycle of the schistosome. Erythematous papules appear within a few days of exposure and will clear spontaneously within a few weeks. Topical steroids may be helpful in lowering itch. Rinse with fresh water and vigorously towelling off after swimming to help decrease severity.

Other Externally Induced Skin Changes

Erythema Ab Igne

Erythema ab igne occurs after long exposure of the skin to low-level localized heat such as hot water bottle, space heater, hand warmer, and/or laptop computer. It presents with reticulated purple-brown patches of discolouration with occasional erosions. The skin changes resolve once the contact with the head source is discontinued.

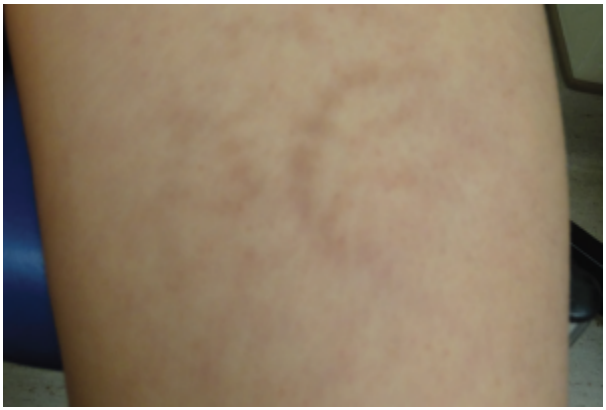

Image 12.11: Erythema ab igne: Reticulate red-brown discolouration due to prolonged use of a laptop on the thighs

Alternative healing practices

Some cultural practices, such as cupping, can lead to skin changes. Cupping is a traditional medical therapy in which a mild vacuum is applied to the skin, generally over the back to help with pain, inflammation and relaxation. The vacuum causes ecchymosis at the site of the cups that heal within 10 days to 2 weeks. **Coining** is a process by which a smooth surface, such as the edge of a coin, is rubbed over oiled skin in a linear pattern until a mark becomes visible.

Tattoos

Tattoos are intentional changes in the skin, but may be associated with unwanted consequences. Acute inflammatory reaction is immediate and improves after 2-3 weeks. It is expected in the course of tattooing. Wound care during this phase includes prevention of infection through keeping the area clean and covered.

Infections can occur if the technique was not hygienic or aftercare was inadequate. Skin infections can also include superficial bacterial infections, folliculitis, abscesses, warts, and atypical mycobacterial infections. Blood borne infections such as hepatitis and HIV can theoretically be transmitted through tattooing.

Allergic reactions that present with eczema, can develop in response to allergens in the ink. Red dyes are the most common to cause allergic reactions. Yellow dye (cadmium sulfate) can also react to sunlight and cause photosensitive reactions. Henna temporary tattoos may also result in allergic contact dermatitis at the site of application.

Quiz

1. Skin type can be used as a proxy for race.

a. True
b. False

2. What Fritzpatrick skin type tans easily and rarely burns?

a. Type 2
b. Type 3
c. Type 4
d. Type 5

3. Where does Polymorphous light eruption (PMLE) most often occur?

a. Dorsal hands
b. Forearms
c. Neck
d. Face
e. All of the above

4. Topical corticosteroids can help with PMLE.

a. True
b. False

5. What are some common triggers of phytophotodermatitis?

a. Lemon
b. Lime
c. Hogweed
d. Fig
e. None of the above
f. All of the above

6. What medication helps with Raynauds?

a. Vasodilators
b. Antibiotics
c. Corticosteroids
d. Stimulants

7. Keeping the body cool helps with Raynauds.

a. True
b. False

8. Which degree of frostbite causes blistering?

a. 1st degree
b. 2nd degree
c. 3rd degree
d. Blistering is not caused by frostbite

9. Where are flea bites commonly found?

a. Face
b. Upper chest
c. Legs
d. Neck

10. How long does it take for ecchymosis from cupping to heal?

a. 1-5 days
b. 5-10 days
c. 10-28 days
d. 30-40 days
Answers: 1.B, 2.D, 3.B, 4.A, 5.F, 6.A, 7.B, 8.C, 9.C, 10.C

CHAPTER 13: DISORDERS OF PIGMENTATION

Hypopigmented and Depigmented Lesions: Pityriasis Alba

What is it?

Pityriasis alba is a common condition seen in children, most commonly in children with darker coloured skin. It is thought to be a result of low-grade inflammation from a mild dermatitis. It often coexists with dry skin and atopic dermatitis.

What does it look like?

Pityriasis alba is characterized by hypopigmented round to oval patches on the cheeks, neck, upper trunk and proximal extremities. They may be slightly scaly. The lesions are well-circumscribed but may not have very sharp edges and might show follicular prominence at the border. Usually it is asymptomatic but in some patients it might be slightly itchy. Pityriasis alba often appears worse after sun exposure due to the contrast caused by tanning of the surrounding skin.

How is it treated?

Pityriasis alba can be treated with a mild topical corticosteroid and the use of a moisturizer to affected areas several times daily.

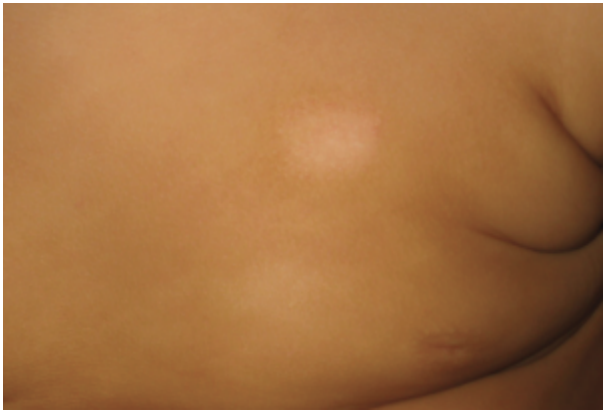

Image 13.1: Ill-defined hypopigmentation with associated follicular prominence

Hypopigmented and Depigmented Lesions: Vitiligo

What is it?

Vitiligo is an autoimmune skin condition involving the loss of melanocytes that results in depigmented, or white areas of skin. The cause of vitiligo is not well understood but it can run in families. In the end the loss of melanocytes leaves the skin completely white. Most patients develop vitiligo before age twenty. Vitiligo is generally asymptomatic but is often a significant cosmetic concern to patients and their families.

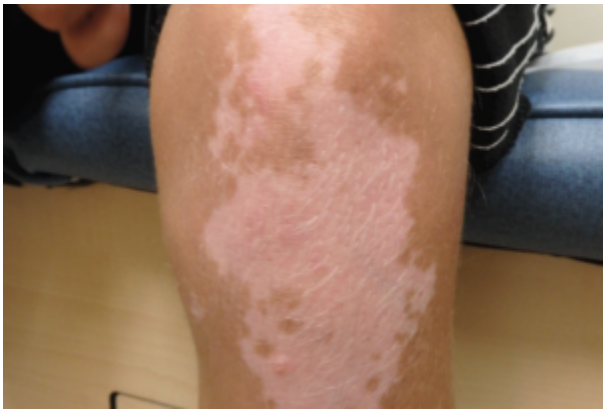

Image 13.2: Vitiligo: Well demarcated depigmentation with associated white hairs and islands of repigmentation

What does it look like?

Vitiligo is characterized by depigmented patches that often appear in a symmetric distribution. They are well defined and may have islands of residual pigmentation visible around hair follicles within the lesions. White hairs might be visible within the patches, which is a poor prognostic sign. It can be localized or segmental in nature, or it can present with more generalized lesions, which are commonly seen bilaterally on dorsal hands and feet, over bony prominences, on the face, and in the genital area. If needed, a Wood's lamp can be used to differentiate depigmented skin (which appears fluorescent white) from hypopigmented skin (which may appear slightly lighter but is not white). Generally vitiligo is a clinical diagnosis and a biopsy is not required.

How is it treated?

Vitiligo is difficult, but not impossible, to treat. Treatment begins with the highest potency steroid appropriate for the site. Steroids are used daily, but with occasional breaks such as 4 weeks on and 2 weeks off, for several months to see if any response will be noted. Topical calcineurin inhibitors, usually tacrolimus 0.1% ointment can be utilized on areas that cannot be safely treated with potent topical steroids and can be rotated with topical steroids for other locations. If there is no improvement within a few months, continued therapy is not likely to be effective. Phototherapy with narrow-band UVB, PUVA, or excimer laser can be helpful

where it is available. Ambient sun exposure can be helpful in stimulating repigmentation, but is often not recommended as a treatment due to an increased risk of sunburn in depigmented areas and increased prominence of skin changes when normal skin becomes more tan. There is an association with vitiligo and other autoimmune conditions, especially autoimmune thyroid disease. Laboratory investigations are not routinely required but may be needed if patients have systemic symptoms that suggest an underlying autoimmune condition. If indicated a CBC, fasting blood glucose and TSH can be checked.

Hypopigmented and Depigmented Lesions: Nevus depigmentosus and Nevus anemicus

Nevus depigmentosus and Nevus anemicus

Nevus depigmentosus is a birthmark that presents as a hypopigmented macule or patch, measuring a few centimeters in diameter and with well-defined but irregular borders. It is most common on the trunk but can appear anywhere. Nevus depigmentosus might not be noticed at birth and become apparent at several years of age.

Nevus anemicus is an uncommon capillary malformation consisting of a localized area of vasoconstricted vessels. It appears as a hypopigmented patch with well defined but irregular borders.

Nevus depigmentosus and nevus anemicus can be differentiated clinically by applying pressure to the skin with a glass slide. A nevus anemicus will characteristically “disappear” into the surrounding skin. Another technique is to rub the skin overlying the area, a nevus anemicus will stay hypopigmented but a nevus depigmentosus will become pink like the surrounding skin. There is no treatment necessary for either of these lesions. If patients are bothered by the appearance make-up to camouflage the area can be helpful.

Hyperpigmented Lesions

Acanthosis nigricans

Acanthosis nigricans is not a skin disease but is a cutaneous sign of an underlying condition. It is characterized by hyperpigmentation and thickening of the skin, often with a velvety texture. This most commonly occurs on the posterior neck and axilla but can involve any skin folds. Most often this is associated with insulin resistance and can be seen in obesity or with poorly controlled diabetes mellitus. Treatment of acanthosis nigricans should be directed at correcting the insulin resistance including weight loss or change in diabetes treatment. To improve the appearance of existing plaques topical retinoids can be tried, but are often irritating.

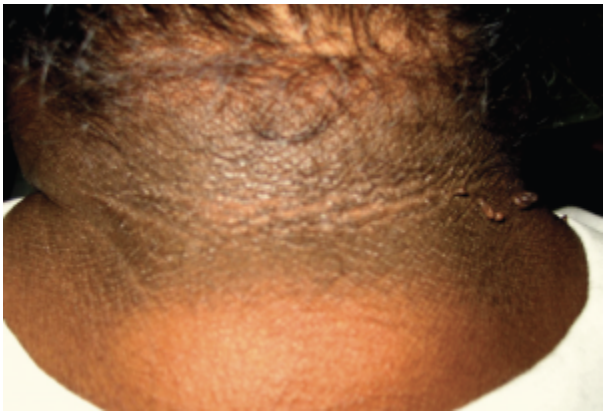

Image 13.3: Acanthosis nigricans: Velvety brown hyperpigmentation on posterior neck with associated skin tag formation

Melasma

Melasma is often called the "mask of pregnancy." It is most commonly seen in adult women and is very uncommon in males. The cause is not fully understood but is associated with hormonal changes such as pregnancy and oral contraceptives. It presents as asymptomatic hyperpigmented macules and patches with irregular borders on the forehead, cheeks, and upper lip. Melasma is commonly a cosmetic concern to patients but does otherwise not need to be treated. Sun protection is the most important component of treatment. Combination therapy with tretinoin, hydroquinone and hydrocortisone is often utilized. Other treatment options include azelaic acid, glycolic acid, kojic acid, and transexamic acid. Prolonged use of hydroquinone is not recommended as it can cause other forms of hyperpigmentation.

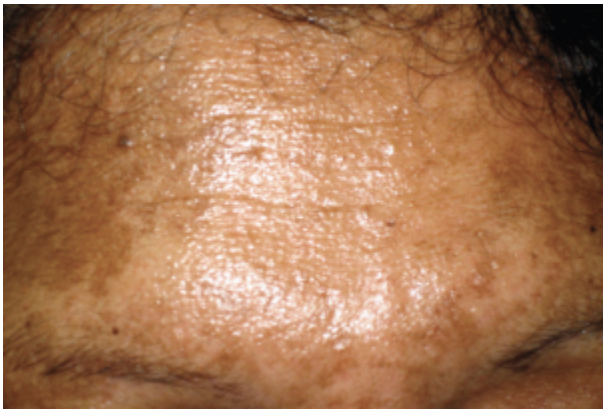

Image 13.4: Melasma: Patchy hyperpigmentation in sun exposed areas with flash artifact causing central brightness

Retention Hyperkeratosis

Retention hyperkeratosis (terra firma-forme dermatosis) presents with reticulate hyperpigmentation that has a slightly velvety appearance. It has the appearance of dirty skin, but cannot be washed off with soap and water. It is caused by changes in keratinization and is not "dirty" skin. The hyperkeratosis can be removed with an alcohol wipe, which makes the diagnosis. Treatment is with use of an alcohol wipe, cream containing salicylic acid or lactic acid, or with gentle scrubbing using a washcloth or loofa pad.

Hyper- or Hypo-pigmented Lesions

Post-inflammatory pigment changes

Post-inflammatory pigment changes are commonly seen after resolution of a rash or skin injury. This is very common with atopic dermatitis, psoriasis or acne vulgaris. Resolution of inflammation can leave either hyper-pigmentation, from deposition of pigment in the dermis, or hypo-pigmentation, from a temporary halt in production of pigment. These pigment changes can occur in any patient but are especially common in darker skin of colour. These pigment changes usually last for months after the skin eruption has resolved and do not need to be treated. Sun protection is helpful as tanning of normal skin makes hypopigmented areas more prominent, and hyperpigmented areas will become even darker.

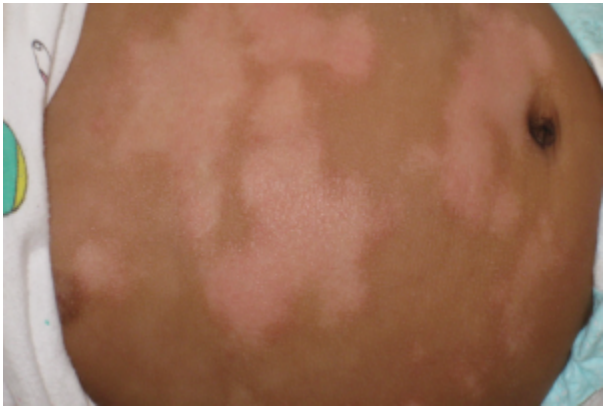

Image 13.5: Post inflammatory hypopigmentation secondary to atopic dermatitis

Pityriasis Versicolor

Pityriasis versicolor, as its name suggests, can present as a variety of colour changes on the skin, including areas that are hyper- or hypo-pigmented. For a full description of Pityriasis versicolor see the Infection and Infestations section.

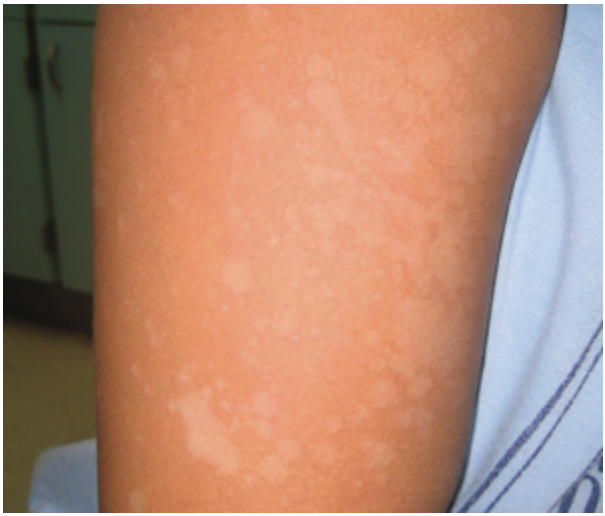

Image 13.6: Pityriasis versicolor: Hypopigmented macules with subtle shade

Pigmentary Mosaicism

Pigmentary mosaicism occurs when patients have two different skin tones in a pattern that corresponds to migration pattern of skin cells during embyogenesis (Lines of Blaschko). These pigment changes arise from genetic mosaicism in the skin cells. Most patients do not have any associated symptoms or syndromic features associated with the pigmentary mosaicism, and no further evaluation or treatment is necessary. Occasionally, widespread pigmentary mosaicism can be seen in conjunction with neurologic changes, such as has been reported in hypomelanosis of Ito, linear and whorled nevoid hypomelanosis, and Blaschkoid dyspigmentation.

Quiz

1. Pityriasis alba tends to have very sharp borders.

a. True
b. False

2. Post-inflammatory hyperpigmentation can be seen following which of these skin conditions?

a. Atopica dermatitis
b. Psoriasis
c. Acne vulgaris
d. All of the above

3. Once melasma is treated it is gone for good and patients cannot get it again.

a. True
b. False

4. A patient with a velvety darkening and thickening around the neck should have what lab test performed?

a. CBC
b. Fasting glucose
c. Urinalysis
d. Liver enzymes

5. What colour does vitiligo show under a woods lamp?

a. Black
b. Fluorescent green
c. Fluorescent white
d. Fluorescent yellow

6. Applying pressure with a glass slide to a nevus anemicus makes it "disappear" into the surrounding skin?

a. True
b. False

7. In general, how long does it take post-inflammatory hyper- or hypo-pigmentation to resolve?

a. Weeks
b. Months
c. Years
d. It is permanent

8. What do Blaschko lines describe?

a. The relaxed skin tension lines
b. They follow the edge of dermatomes
c. The migration of skin cells during embryogenesis
d. They follow the major arteries of the body

9. When a vitiligo lesion repigments, where does the pigmentation usually first develop?

a. Perifollicular
b. Uniformly through the lesion
c. At the center
d. At the most proximal border

Answers: 1.B, 2. D, 3. B, 4. B, 5.C, 6.A, 7.B, 8.C, 9.A

CHAPTER 14: HAIR AND NAILS

Alopecia

Alopecia is the medical term for hair loss, which may be due to a wide variety of causes. It is not a specific diagnosis but may be due to a wide variety of causes. In general, hair loss can be categorized by 2 criteria: localized vs. diffuse, and scarring vs. non-scarring. In localized hair loss, the thinning occurs in isolated areas or patches, while in diffuse hair loss it is seen as thinning over a larger area of the scalp. In non-scarring hair loss, the follicles remain unharmed though they are not making hairs, while in scarring hair loss, the follicles are lost and cannot regrow hair even after the underlying problem has been treated. Clinically, scarring appears as loss of the follicular openings, creating a smooth, shiny, white, scar-like appearance. Using these criteria can help to quickly narrow the differential diagnosis as well as to prioritize which cases are more urgent – cases of scarring alopecia require more urgent dermatologic assessment as they can result in permanent hair loss.

Differential diagnosis for alopecia (hair loss):

	Scarring	**Non-Scarring**
Localized	Discoid lupus Kerion (advanced tinea capitis) Acne keloidalis nuchae Folliculitis decalvans Aplasia cutis congenita	Alopecia areata Tinea capitis Traction alopecia Trichotillomania Triangular temporal alopecia Androgenetic alopecia Secondary syphilis
Diffuse	Dissecting cellulitis of the scalp	Alopecia totalis/universalis Anagen effluvium Telogen effluvium Loose anagen syndrome Androgenetic alopecia Alopecia assoc with systemic disease/nutritional deficiency

Localized, Non-Scarring Alopecia: Alopecia Areata

What is it?

Alopecia areata is an autoimmune disease caused by T-cells that cluster around the bulb of the hair follicle and cause the hair to fall out. On biopsy, the T lymphocytes look like a "swarm of bees" around the hair follicle. Alopecia areata can be associated with several other autoimmune diseases, such as thyroid disease, vitiligo and inflammatory bowel disease.

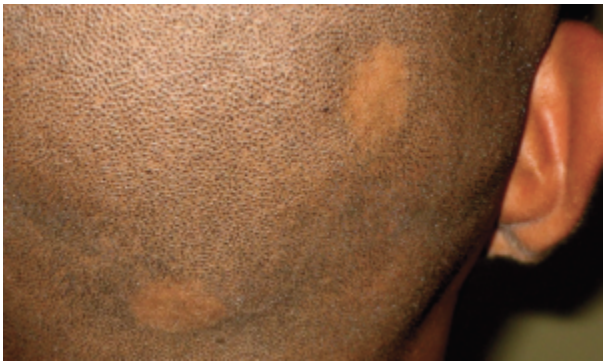

Image 14.1: Alopecia areata: Patchy non-scarring hair loss with no associated redness or scale

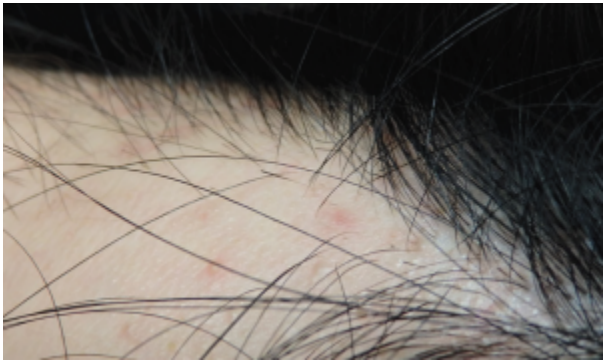

Image 14.2: AA with exclamation point hairs

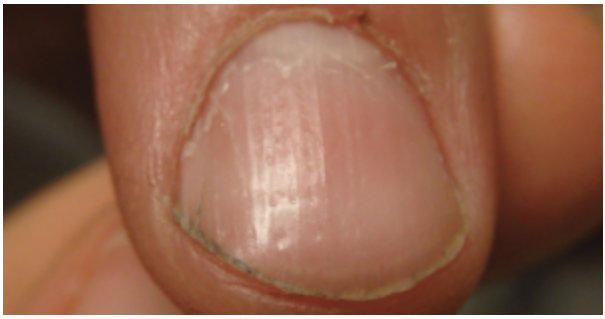

Image 14.3: Geometric pitting of nails associated with alopecia areata

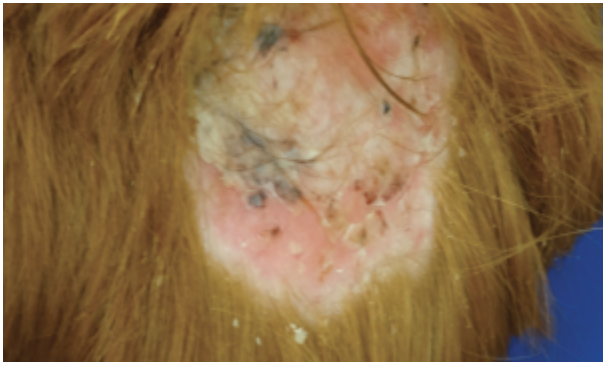

Image 14.4: Tinea capitis: Localized alopecia due to severe tinea infection, which regrew entirely after treatment

What does it look like?

The hair loss in alopecia areata is non-scarring and localized. The patches are typically round or oval in shape and well circumscribed with complete loss of hair. It may affect the scalp or other areas such as eyebrows, eyelashes and facial hair. Exclamation point hairs are a classic finding and are best seen with magnification. These are hairs, which taper closer to the scalp, resembling an exclamation point. Nail pitting can be seen. There are several variants of alopecia areata that are particularly difficult to treat. **Alopecia totalis** is complete loss of hair on the head and **alopecia universalis** is loss of hair on the entire body. **Ophiasis** is hair loss around the occiput (hair line on the back of the scalp) and is seen mostly in children.

How is it diagnosed?

The diagnosis is usually clinical and typically does not require a biopsy. A thorough history and physical should be done to assess for associated disorders, particularly thyroid disease. Bloodwork can be ordered if there is any concerning signs or symptoms but does not need to be performed routinely.

How is it treated?

Hair will often regrow on its own within affected patches. However, a new patch somewhere else is likely to appear in the future, and the overall course of the disease is unpredictable. If patients are motivated and can tolerate needles, treatment is usually with local intradermal injections of steroids (triamcinolone 2.5 mg/cc). In young patients and those who cannot tolerate intralesional therapy, Potent topical steroids can be utilized. Topical minoxidil has been helpful in some patients, particularly once new growth begins. Application of irritants and allergens, such as anthralin or DPCP/squaric acid, can be performed, but are not always well tolerated. For patients with severe and widespread disease, systemic medications such as pulse steroids, methotrexate, and JAK inhibitors can be considered.

What is the differential diagnosis?

Tinea capitis is a superficial fungal infection of the scalp. Usually, it can be distinguished from alopecia areata by the presence of scale and redness. The hairs may also be broken off near the scalp creating a "black dot" appearance. A scraping for KOH prep can confirm the diagnosis. Treatment is with oral antifungals (See Ch. 6).

Trichotillomania is a self-induced condition, wherein hair loss is cause by pulling or twirling of the hairs. It is often associated with anxiety, stress or behavioral conditions. Clinical clues include patches of hair loss with sharp, angular borders and twisted and broken hairs of varying lengths. Consultation with psychology can be useful to address the underlying cause.

Traction alopecia is hair loss due to frequent or prolonged mechanical strain on hairs. It is most commonly seen in children who wear their hair in tight braids, pony tails, or whose hair is tied back under a turban. The hair loss is usually noted wherever hair has the highest degree of strain. Change in hair-care practices can help reverse the condition.

Secondary syphilis is sometimes associated with a "moth-eaten" alopecia. Usually patients also have a diffuse rash and other symptoms such as low-grade fever and fatigue. Syphilis is increasingly common in North America, so a sexual history may be relevant in adolescent patients to decide if this condition is on the differential.

Localized Scarring Alopecia: Discoid Lupus

What is it?

Discoid lupus is a cutaneous form of lupus; if it affects hair-bearing sites such as the scalp it can cause scarring (permanent) hair loss. Around 10-20% of patients with discoid lupus will meet criteria for systemic lupus at some point in their life.

What does it look like?

Plaques of discoid lupus typically affect the head and neck, and less often other areas of the body. They start as scaly red plaques which eventually leave scar-like white areas centrally and hyperpigmented rims.

How is it treated?

Referral to a dermatologist is indicated for diagnostic confirmation and treatment. Mild disease can be treated topically with potent corticosteroids and/or calcineurin inhibitors. Injection of corticosteroids can help treat localized resistant lesions. The first line treatment for more severe disease is hydroxychloroquine. Conservative measures such as sun protection are also very important.

What other forms of localized scarring alopecia are there?

Acne keloidalis is a relatively common form of localized scarring alopecia. It is a variant of acne that causes bumps on the back of the scalp that scar and result in hair loss in this region. Keloid scars may form as the acne heals. It is treated with a combination of antibiotics, such as doxycycline, and intralesional steroid injections with triamcinolone acetonide (typically using concentrations of 20-40 mg/mL).

Aplasia cutis congenita is a congenital form of localized hair loss. It most commonly occurs on the scalp and is typically an isolated anomaly, although may rarely be associated with certain genetic syndromes or other congenital abnormalities. The skin is usually absent with an erosion or ulcer at birth that heals with scarring. There is sometimes a rim of thick/coarse hair around the patch of hair loss called the “hair collar sign”. If hair collar sign is present or the area of aplasia is quite large, the area should be imaged to ensure closure of the skull below the lesion.

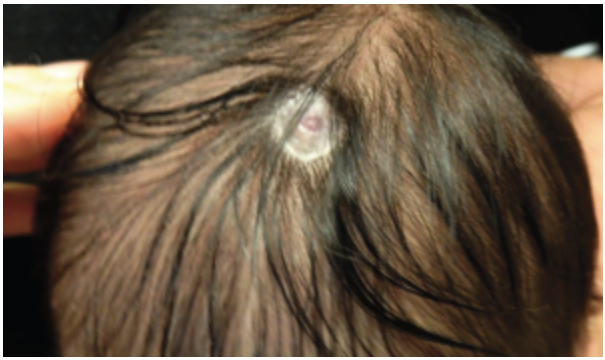

Image 14.5: Aplasia cutis congenita: Round hairless plaque from birth

Kerion is a severe, inflammatory form of tinea capitis and present with inflamed, boggy skin often with pustules. While most tinea capitis is non-scarring, due to the severity of inflammation, kerion can result in permanent scarring. A short course of systemic corticosteroids can be considered in addition to routing oral antifungal therapy.

Morphea, En Coup de Sabre is an autoimmune condition that can present in a linear atrophic band on the forehead and scalp. Alopecia associated with this condition is often scarring and is associated with significant atrophy. If headaches are present, patients should be referred to neurology and imaging with MRI considered,

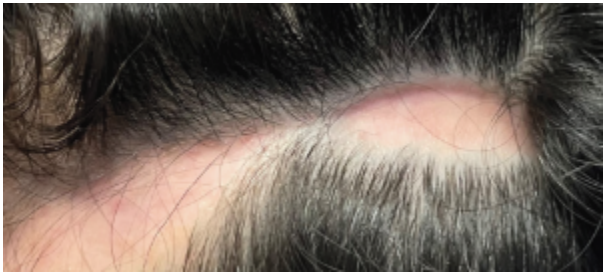

Image 14.6: Morphea en coup de sabre causing linear plaque of scarring alopecia

Diffuse Non-Scarring Alopecia

Telogen Effluvium: Telogen effluvium occurs when a significant portion of the hair simultaneously enters resting phase (telogen) due to an acute stress, illness, or rapid dietary change such as crash diet. Several months later, this hair is shed and thinning is noticed. After a few months, the hair will begin to regrow normally and no treatment is needed.

Anagen Effluvium: Cytotoxic chemotherapy medications cause arrest in hair growth and subsequent hair loss. Hair is shed during the growth (anagen) phase and will regrow after completion of chemotherapy.

Androgenetic alopecia: Patterned hair loss occurs in both men and women but is generally more pronounced in males. It can begin in adolescence, though usually does not appear until adulthood. Thinning occurs over the crown as well as frontal scalp. In men, it often presents with receding frontal hair line and vertex of the scalp and can progress to complete hair loss. In women, widening of central part is more common. Treatment is with minoxidil topically or finasteride depending on clinical context and severity.

Loose anagen syndrome: An uncommon form of hair loss noted in young children. It most typically presents in young girls who present with the history of never needing a haircut. In this condition, the hairs are not well attached to the scalp during growth phase and so fall out before they reach full length. It tends to improve with age.

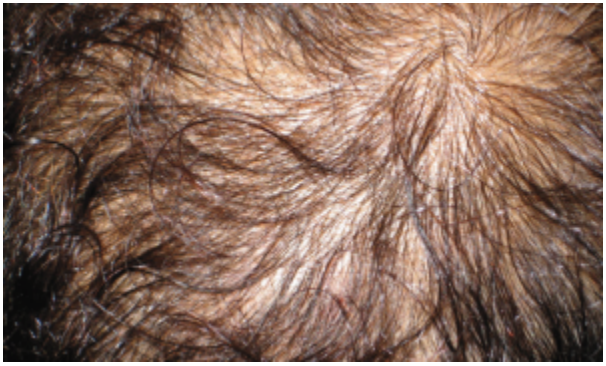

Image 14.7: Telogen effluvium: Diffuse hair thinning with no background skin change

Nails

In addition to disorders that primarily affect the nails, abnormalities in the nail may be markers of systemic conditions and overall health status. They can also provide useful clues towards certain skin conditions when the appearance of the rash is not diagnostic.

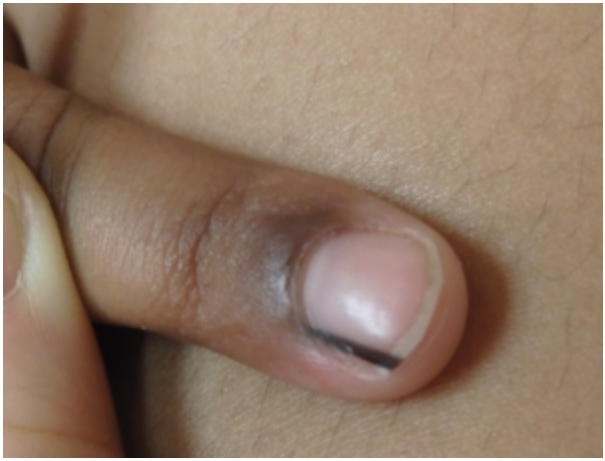

Image 14.8: Melanonychia striata: Dark but uniform band of hyperpigmentation in nail plate

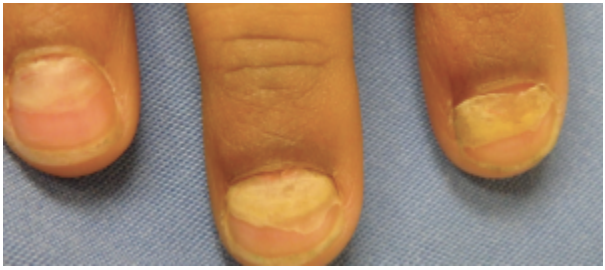

Image 14.9: Onychomadesis: Peeling of nails from poximal edge after hand foot and mouth

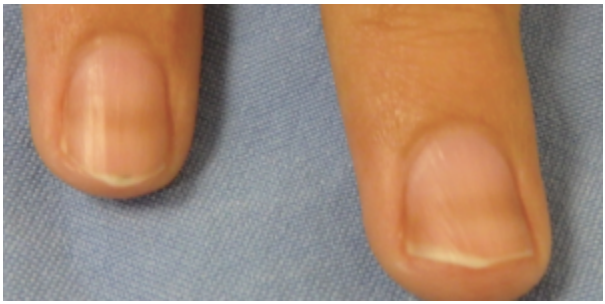

Image 14.10: Horizontal bands of hyperpigmentation in the nail due to chemotherapy

Image 14.11: Physiologic melanonychia causing tan band with the nail

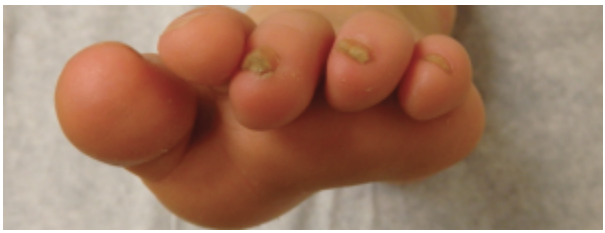

Image 14.12: Onychomycosis with thick, crumbly nails

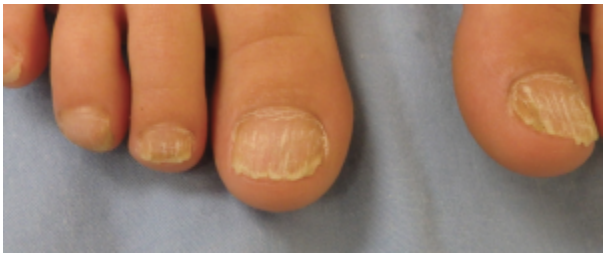

Image 14.13: Trachyontchia: Dull lack-luster nails with increased longitudinal ridging

Terminology for nail findings and their clinical significance

Name	Description	Clinical Significance
Onycholysis	Distal nail plate detaches from nail bed causing white appearance distally	Psoriasis and onychomycosis most common; trauma, drugs (commonly tetracyclines), tumors under nailbed
Beau's lines	Transverse depressions of the nail plate	Most often trauma; eczema around nail; involvement of multiple digits at same level suggests systemic cause
Onychomadesis	Detachment of nail plate from proximal nail fold (a depressed groove replaces proximal nail plate)	Single nail – most often trauma Multiple – systemic cause such as HFMD
Trachyonychia	Diffuse homogenous roughness, loss of translucency	Isolated finding Alopecia areata, lichen planus, psoriasis, eczema
Pitting	Punctate depressions of nail plate surface	Psoriasis, alopecia areata, eczema
Splinter hemorrhages	Red to purple thin longitudinal lines in the nail plate	Trauma (most common), psoriasis, onychomycosis; proximal splinters are rare and suggest systemic disease (e.g. endocarditis, vasculitis)

Longitudinal melanonychia	Longitudinal brown to black band(s)	Multiple: physiologic, trauma, drugs, systemic cause Single: nevus, melanoma
Subungual hyperkeratosis	Thickened nail due to build-up of scale under the nail plate	Onychomycosis, psoriasis, eczema
Koilonychia	Spoon-shaped nails	Normal in 2nd-4th toes in children aged 1-4 years; Adults: severe iron deficiency

Longitudinal Melanonychia

What is it?

Longitudinal melanonychia describes brown to black streaks/bands that run longitudinally in the nail. It may be present in just one nail or may affect multiple nails. A single nail may have multiple streaks. It is often a source of concern for patients and physicians due to the possibility of melanoma.

What causes it?

Longitudinal melanonychia can occur due to numerous causes. If multiple nails are involved, this is suggestive of normal physiologic pigmentation (commonly occurs in darker-skinned individuals). Trauma, endocrine diseases, nutritional deficiency or drug/pregnancy-induced pigmentation can also lead to multiple bands of melanonychia. If only one nail is involved the differential is more limited and suggests presence of a melanocytic lesion. In children, these are usually benign lentigo or nevi, but new melanonychia in adults raises concern for melanoma. Features concerning for nail melanoma include width of >3mm, extension of the pigment onto the cuticle/proximal nail fold (Hutchinson sign), blurry or irregular borders, nail dystrophy (distortion of the normal nail structures), and triangular shape of band suggesting growth of the lesion; however, in children even benign lesions can share some of these features.

How is it managed?

The management addresses any underlying cause that is identified. In many cases, this simply involves reassurance that the pigmentation is a normal change. If there is concern about a melanoma of the nail, a biopsy of the nail matrix should only be undertaken. Due to risk of permanent nail dystrophy and rarity of nail melanoma in children, it is typically recommended that pediatric lesions be monitored, and biopsy only performed if they undergo rapid expansion and/or darkening. In adults, however, new isolated melanonychia generally warrants biopsy.

Quiz

1. Alopecia can be categorized by which of the two categories?

a. Localized vs. Diffuse
b. Scarring vs. Non-scarring
c. Blistering vs. Non-blistering
d. Scale vs. No scale

2. In non-scarring hair loss, follicles are destroyed, which is why they are not making hairs.

a. True
b. False

3. Which of the following is an example of localized scarring alopecia?

a. Tinea capitis
b. Telogen effluvium
c. Discoid lupus
d. Secondary syphilis

4. Alopecia areata is an autoimmune disease caused by which kind of cells?

a. B-cells
b. T-cells
c. None of the above
d. Both of the above

5. Ophasis is mostly seen in children.

a. True
b. False

6. How is tinea capitis distinguished from alopecia areata?

a. Redness
b. Scale
c. Scarring
d. Blistering
e. More than one of the above
f. All of the above

7. Pitting is significant in which of the following?

a. Eczema
b. Alopecia areata
c. Psoriasis
d. All of the above
e. None of the above

8. Why would physicians find brown/black streaks that run longitudinally in the nail concerning?

a. There is never a concern, it is always pigmentation
b. It may be melanoma
c. It could damage the nail
d. There is never a concern, it is normal to occur

Answers: 1. A &B, 2.B, 3.C, 4.B, 5.A, 6.E (Redness and scale), 7.D, (Though most prevalent in AA and psoriasis), 8.B

CHAPTER 15: OTHER DERMATOLOGIC CONDITIONS

Scars

Conventional wound healing occurs in three phases: inflammatory, proliferative, and remodeling. The inflammatory phase controls the injury and prevents infection. In the proliferative phase there is formation of granulation tissue. Remodelling is the longest phase, lasting up to years, in which the scar matures.

Scars: Hypertrophic Scar

Hypertrophic scars are raised and thickened scars that are confined to the wound margin. They develop immediately after an injury within a few weeks to months. Occasionally they may gradually improve spontaneously. They may be itchy or painful. For management of hypertrophic scars, the same techniques as discussed below for keloid scars can be used. Hypertrophic scars generally respond well to treatment.

Scars: Keloid Scar

A keloid scar extends beyond the wound margins into the adjacent normal skin. The onset is delayed, and they are not always preceded by a significant injury. Keloid scars are often painful or itchy.

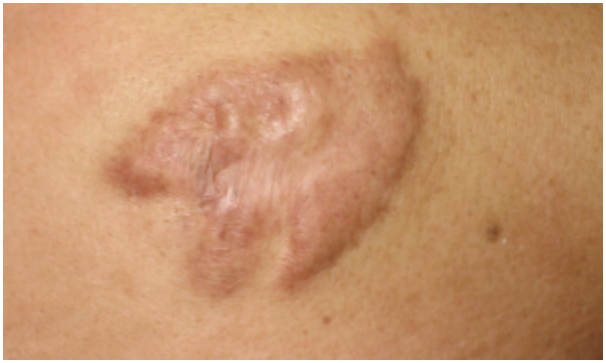

Image 15.1: Keloid scar: Pink brown keloid scar growing beyond boundaries of original scar

Keloid scars are more common in younger patients, patients with skin of colour, those with a prior history of a hypertrophic or keloid scar. Shoulder, chest, upper back or ear are common sites.

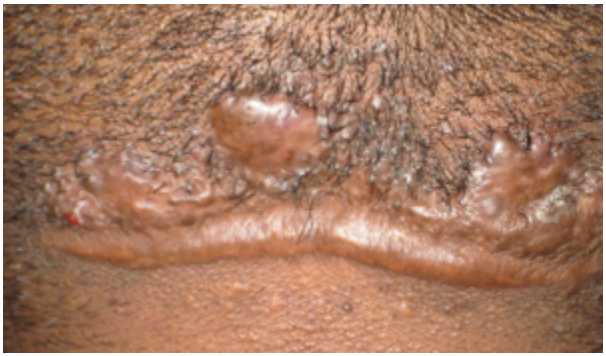

Image 15.2: Keloid scar: Shiny linear plaque of scar at site of excision of previous scars due to acne keloidalis

Prevention of keloid scars is important and unnecessary procedures should be avoided in high risk patients. Topical silicone sheets or gels and massage may help to prevent formation of hypertrophic and keloid scars, but there is insufficient evidence to recommend this routinely

and these products can be expensive. If there is very high risk of keloid formation, intralesional triamcinolone might be injected post-operatively.

Intralesional corticosteroid injections with triamcinolone acetonide (TAC 20-40 mg/cc) is most commonly used in treatment. Of note, keloid scar injections are painful and only small volumes can be injected each session due to the tight nature of the scar. Some keloid scars are excised, and careful wound care put in place to prevent recurrence.

Scars: Keratosis Pilaris

What is it?

Keratosis pilaris (KP) is a common condition to due keratin plugging of hair follicles. It runs in families as an autosomal dominant trait. KP is most prominent during childhood and teenage years and usually spontaneously resolves by adulthood.

What does it look like?

Patients have tiny rough, red bumps in a follicular distribution, most commonly over the posterior upper arms and lateral thighs. Variants include: keratosis pilaris rubra facei that has rough bumps on a background of erythema; ulerythema ophryogenes associated with loss of lateral eyebrows, and keratosis pilaris atrophicans that leaves pitted scars. In patients with darker skin of colour there can be hyperpigmentation at each hair follicle. KP is generally asymptomatic but some patients experience itch.

How is it treated?

There is no cure for keratosis pilaris since it is a genetic trait. However, as mentioned above it does improve with age. Treatment with emollients is helpful, especially those with a keratolytic, such as salicylic acid or urea. Any treatment however is a temporary fix as the bumps do reappear after moisturization is stopped. If itch is a concern, then a mild topical corticosteroid is usually sufficient.

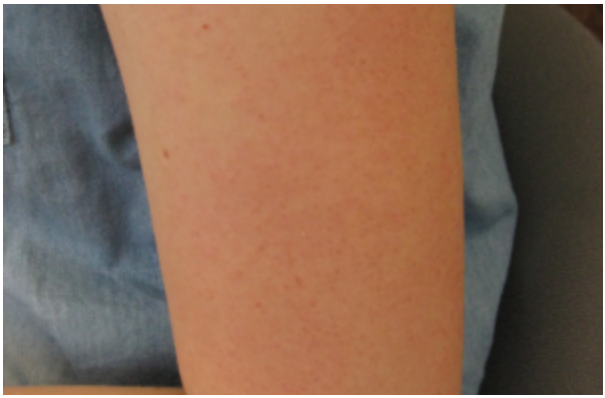

Image 15.3: Keratosis pilaris causing perifollicular papules on posterior upper extremity

Hyperhidrosis

What is it?

Hyperhidrosis refers to excessive and uncontrollable sweating. Primary hyperhidrosis usually starts in childhood or adolescence and there may be a family history of hyperhidrosis.

Secondary hyperhidrosis is much less common and can occur from damage to the nervous system or from endocrine disorders including diabetes and hyperthyroidism. There are several medications that can cause hyperhidrosis including, tricyclic antidepressants, selective serotonin reuptake inhibitors (SSRI), nicotinamide, opioids. Alcohol and caffeine can also cause excessive sweating. Hyperhidrosis can be very distressing to patients and have a significant psychosocial impact.

What does it look like?

Hyperhidrosis can be localized or generalized. Localized hyperhidrosis most commonly affects the axilla, palms, and/or soles. Primary hyperhidrosis is generally localized and symmetric. It can be made worse by hot weather, exercise, anxiety and spicy food. Secondary hyperhidrosis is more likely to be unilateral, asymmetric or generalized.

How is it treated?

General measures that can help decrease sweating or the impact of it include wearing loose-fitting and stain-resistant clothing, changing clothing and footwear when it is damp, avoiding caffeinated beverages and alcohol and discontinuing any medications that may be contributing.

Topical antiperspirants such as aluminum chloride in 12-20% solutions are first line in treatment. These are applied nightly to the affected areas until sweating is decreased and then several times weekly for maintenance. The most common side effect is skin irritation, which is worsened by application to damp skin.

If topicals are not sufficient, oral medications such as beta blockers and oral anticholinergic drugs can be trialed. Beta blockers can be used in situations where a patient anticipates having anxiety such as during a presentation. Oral anticholinergics that can be used include oxybutynin or glycopyrrolate, although these may lead to the side effects of dry mouth and eyes, blurry vision, dizziness and constipation.

Iontophoresis devices involve submersion of the affected area in water and application of an electrical current for ~15 minutes. These can be purchased by the patient for home use for hyperhidrosis of the palm or soles and some devices have special pads for axillary use.

Injection of botulinum toxin is often used in the axilla and is very effective, although it is expensive and needs to be repeated approximately every 6 months. It can be used on the palms or soles, but the injections are very painful, and there is a risk of muscle weakness which may interfere with dexterity.

More invasive measures reserved for severe, refractory cases include removal of axillary sweat glands, and sympathectomy.

Langerhans Cell Histiocytosis

What is it?

Langerhans cell histiocytosis (LCH) is an inflammatory neoplasm of dendritic cells primarily in the skin and bone. LCH is most common in children, especially those younger than 3 years old. It has a higher incidence in Caucasian patients than in those of Asian or African descent. LCH most commonly involves the skin and bone, but it may also affect other internal organs, including the liver, spleen, bone marrow, pituitary gland and lungs. There can be a single organ system involved, or multiple. If there is skin involvement usually there is at least one other organ system involved.

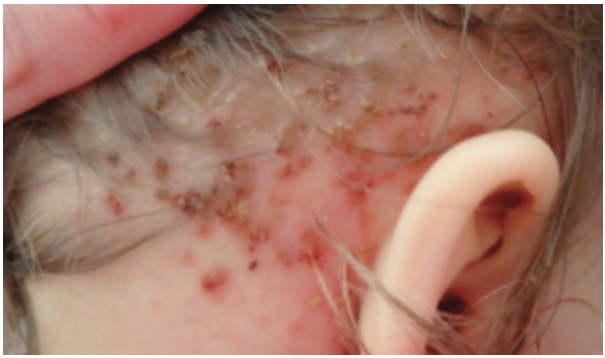

Image 15.4: LCH: Crusted and petechial papules in a post-auricular distribution

What does it look like?

It presents most commonly with erythematous papules, petechiae, and plaques on the trunk, scalp, axilla and groin. These lesions are often crusted and may be itchy.

Consider a diagnosis of LCH if a child has a persistent eruption on the scalp or groin that is not responding to standard treatment, and especially if the child has any systemic symptoms. Rapid referral to dermatology and general pediatrics for biopsy and further assessment is recommended.

How is it treated?

If a diagnosis of LCH is made with systemic involvement these patients are usually primarily managed by the pediatric hemato-oncology team.

The prognosis of LCH depends on several factors, including whether there is single or multi-system disease, and if there is involvement of high-risk organs (liver, spleen, or bone marrow). Single system disease, and multi-system disease without risk organ involvement have a favourable prognosis with a greater than 98% five-year survival.

Aphthous Stomatitis

Aphthous stomatitis or aphthous ulcers (also called canker sores) are a common occurrence in the general population and usually first appear in childhood or adolescence. Aphthous ulcers are well demarcated, round or oval, superficial ulcers with a white to yellow base and surrounding erythema. Triggers include stress, lack of sleep, trauma, irritation from food or toothpaste, or viral infection. A family history of aphthous ulcers may exist in those who experience recurrent lesions. Most aphthous ulcers heal within 1-2 weeks without any treatment. However, they are often painful and patients may benefit from protective pastes, with or without a corticosteroid, or topical anesthetics. Patients should also avoid any known triggers.

The differential diagnosis of an oral ulcer should include a herpes simplex infection, erythema multiforme, oral lichen planus, and Behçet disease. The manifestations of herpes simplex and erythema multiforme are discussed in the Infections & Infestations and Inflammatory Skin Conditions sections respectively.

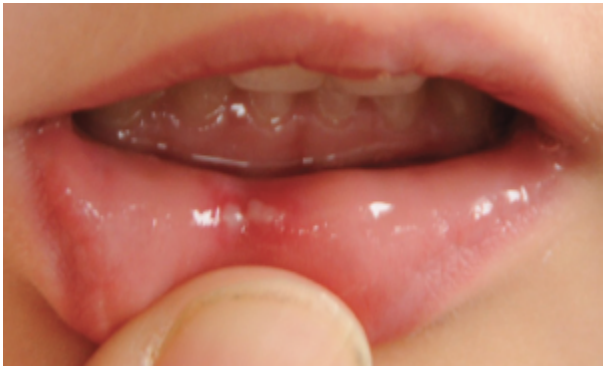

Image 15.5: Oral aphthous ulcers with white center and erythematous rim on mucosal surface

Reactive Infectious Mucosal-predominant Eruption (RIME)

RIME (sometimes known as Mycoplasma-Induced Rash and Mucositis (MIRM)) is a relatively newly described entity, which is a reactive process that occurs in the setting of infection with *Mycoplasma pneumoniae* (a common bacterial cause of community-acquired pneumonia) or other infections. It usually affects children. It is characterized by severe mucositis (inflammation of mucosa, such as the mouth and eyes) with generally mild/limited skin involvement. Virtually all patients have oral involvement presenting as hemorrhagic crusts and erosions on the lips, tongue and buccal mucosa. The majority of cases will also have bilateral conjuctivitis and ~60% have urogenital involvement. The skin is usually less involved and the appearance of the rash is variable, with the most common presentation being vesicles and blisters. Since the clinical picture can be very similar to that of Stevens-Johnson syndrome/toxic epidermal necrolysis (see Ch. 11), patients are usually best assessed in an acute care setting to rule this out. Treatment of severe cases includes systemic steroids and consideration of medication with anti TNF activity such as etanercept or cyclosporine for a few doses. Oral care can involve use of "magic mouthwash" (combination of topical anesthetic, corticosteroid, antibiotic and antacid) and saline soaks followed by petroleum jelly to crusts/erosions. Patients often require referral to ophthalmology and/or urology or gynaecology. Antibiotics covering *M.pneumoniae* (e.g. macrolides such as azithromycin) may be used but it is unclear if this shortens the course of mucositis and rash. RIME can recur with future infections.

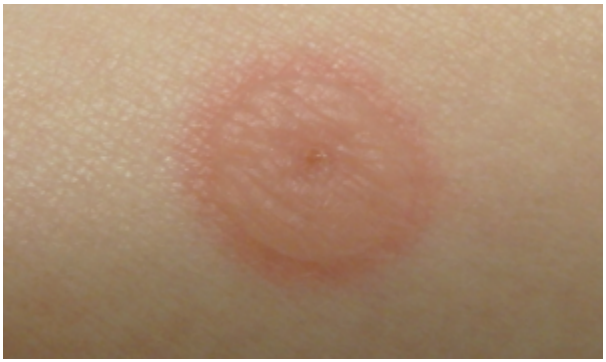

Image 15.6: RIME: A targeted bulla on the arm

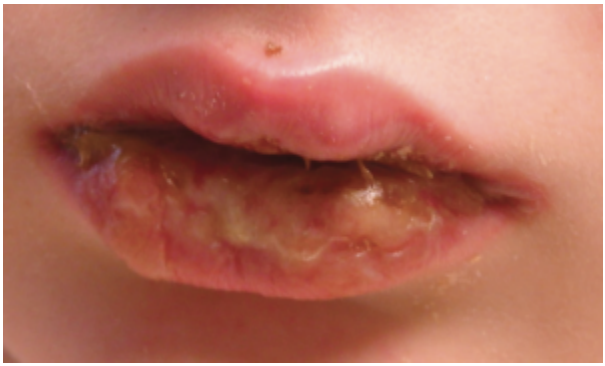

Image 15.7: RIME: Significant mucositis associated with conjuctivitis and scattered targetoid bulla on extremities

Prurigo Nodularis

What is it?

Prurigo nodularis is a pattern that can occur following any process that is initially itchy, such as arthropod bites and eczema. The involved areas are scratched repeatedly and then thicken in response to the injury. This leads to the development of papules and nodules that are themselves itchy, which brings more scratching and more thickening. Potent topical steroids and avoidance of scratching are the mainstays of treatment.

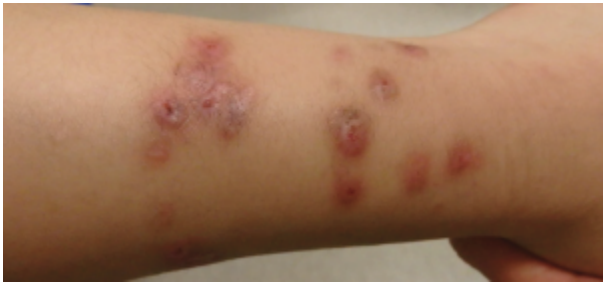

Image 15.8: Prurigo: Violaceous papules and nodules with excoriation

Quiz

1. Hypertrophic scars are confined to the wound margin.

a. True
b. False

2. Which of the following is not a risk factor for developing a keloid scar?

a. Darker skin colour
b. Occuring on the shoulder or ear
c. Thermal burns
d. Prior history of keloid scar

3. What is the first line treatment for a keloid scar?

a. Vitamin E oil
b. Intralesional triamcinolone acetonide
c. Thermal burns
d. Prior history of keloid scar

4. Which of the following is not a common location for keratosis pilaris?

a. Shins
b. Face
c. Upper arms
d. Thighs

5. Successful treatment of keratosis pilaris with a keratolytic prevents further recurrences.

a. True
b. False

6. Possible triggers of hyperhidrosis include:

a. Emotional stress
b. Caffeine
c. SSRI's
d. All of the above

7. Applying topical aluminum chloride to the skin when it is damp with sweat will increase the penetration and result in a faster response to treatment.

a. True
b. False

8. Orofacial granulomatosis may be triggered by:

a. Cinnamon and benzoates
b. Malassezia furfur overgrowth
c. Chronic topical corticosteroid use
d. Persistent lip licking

9. RIME is always associated with mycobacterial infection.

a. True
b. False

Answers: 1.A, 2.C, 3.B, 4.A, 5.B, 6.D, 7.B, 8.A, 9.B

CHAPTER 16: COMMON DERMATOLOGIC THERAPIES

Routine Skin-Care Measures: Sun Protection

Sun protection requires a multi-pronged approach including sunglasses, a wide-brimmed hat, protective clothing and sunscreen. Prolonged sun exposure should be avoided by seeking shade and avoiding time in the mid-day sun in spring and summer months. Sunscreen needs to be reapplied frequently on exposed skin especially after swimming or excessive sweating.

Sunscreens are barriers applied to the skin that either absorb or reflect the sun's ultraviolet (UV) rays.

UVB protection is measured by the Sun Protection Factor (SPF) of the sunscreen. SPF is a ratio of the amount of time it takes skin with sunscreen applied to burn compared to unprotected skin. Sunscreens with a "Broad Spectrum" label contain ingredients that have been shown to protect from UVA such as titanium dioxide, zinc oxide and avobenzone. A broad spectrum sunscreen with at least SPF 30 is recommended.

Routine Skin-Care Measures: Emollients/ Moisturizers

In general, thicker moisturizers in an ointment or cream base are the most moisturizing. Patients should look for products without fragrance. For those with dry skin or eczema, moisturizer is best applied every day and especially after bathing. It is extremely important to consider the patient's preference as they are unlikely to consistently use a product that they do not like. In addition, expensive does not necessarily mean a product will work better.

Some moisturizers have added ingredients such as keratolytics which include salicylic acid, glycolic acid, lactic acid or urea. These break down surface scale and can be useful in conditions with thickening of the skin or hyperkeratosis such as keratosis pilaris or palmoplantar keratodermas.

For patients with sensitive skin who are worried about irritation from products they can perform a Repeat Open Application Test (ROAT). This is done by applying a small amount of the product to the inner forearm (making sure this area is free of eczema or other skin disease) twice daily over a week. If no irritation develops it should be safe to use this product more widely on the body.

Routine Skin-Care Measures: Soap and Cleansers

Pure soap can be irritating to the skin. Syndets (synthetically produced detergents) are widely available and are more gentle on the skin. For patients with atopic dermatitis or with sensitive skin we recommend using non-soap cleansers for face washing and in the shower.

Routine Skin-Care Measures: Hand Sanitizers

Hand sanitizing gels are alcohol-based and an effective alternative for hand washing where clean water and soap are not available. However, they can be drying and can sting if there are any abrasions on the skin. For people who need to wash their hands frequently they may be less drying than washing multiple times with soap and water.

Routine Skin-Care Measures: Dilute Bleach Baths

Bleach baths are commonly used in patients with atopic dermatitis. Household bleach of any brand can be used. Of course, care should be taken to avoid direct skin contact with bleach and to prevent unsafe storage that could lead to accidental exposure.

To prepare the bleach bath in a full-sized bathtub:

- Add ¼ cup of household bleach to a ¼ full bathtub
- Add ½ cup of household bleach to a ½ full bathtub

If the child is being bathed in an infant tub:

- Add 2 tsp of household bleach to 2L of water

Patients soak in the prepared tub for 10-15 minutes, with care not to get water in the eyes, and then rinse off before patting dry. Moisturizer should be applied all over after bathing. Bleach baths can be repeated several times per week.

Routine Skin-Care Measures: Topical Therapies

Topical products, either prescribed or over the counter, are commonly used for both treatment and prevention of many dermatologic conditions. All topical medications are formulated into a base, which has an impact on the delivery of the medication. The potency of the same medication is highest when formulated into an ointment, followed by creams and lotions.

Ointment: Thick, little water added, clear, greasy feel. Least likely to sting.

Cream: Thick, cannot be poured, white, primarily oil with water added, moisturizing, easier to apply than ointment.

Lotion: Thin, can be poured, white, non-greasy feel, easy to apply.

Oil: Runny, no water added, easy to apply, best applied to slightly wet skin.

Solution: Water or alcohol based with dissolved medication. Liquid, easy to apply to scalp, might sting.

Routine Skin-Care Measures: Anti-inflammatories

Corticosteroids

Topical corticosteroids (TCS) are classified by their ability to cause vasoconstriction, which roughly parallels their anti-inflammatory ability. Class I are the strongest steroids, and Class VII are the weakest.

Practical Tips

- A general guide to steroid concentration by body site:
- Hands and feet: Class I & II
- Trunk, limbs: Class III-V
- Face and body folds (groin, axilla): Class VI-VII
- A few days of stronger-than-usual potency might be necessary for severe flares.
- The duration of treatment with a TCS will vary with the condition being treated. Patients should treat until completely clear. “Clear” means that the skin is no longer red or bumpy, but post-inflammatory pigment change might remain. For patients who have frequent flares of their skin condition, using the TCS twice weekly for maintencance can help prevent flares. The goal is to be “off” the TCS more than they are “on.”

*Note that ointments are often more potent than creams for the same medication

- Wet wraps can be helpful to hydrate the skin and increase the efficacy of topical corticosteroids.

For widespread eruptions:

- Apply emollient or topical steroid to the affected areas
- A pair of damp full body pyjamas
- Place a dry layer of clothes over top to prevent evaporation and heat loss.
- For smaller involved areas, such as the hands or feet a topical steroid can be applied and then covered with a damp sock or glove with a dry overlayer
- Occlusion with dressing such as Tegaderm, plastic wrap or non-breathable gloves is another way to increase the penetration of topical corticosteroids.

		Betamethasone dipropionate 0.05% ointment
Ultra High Potency	**Class I**	Clobetasol propionate 0.05%
		Halobetasol propionate 0.05%
High Potency	Class II	Betamethasone dipropionate 0.05% cream
		Fluocinonide acetonide 0.01%
Medium Potency	Class III	Betamethasone valerate 0.1% ointment
		Mometasone furoate 0.1% ointment
		Betamethasone valerate 0.1% cream
	Class IV	Triamcinolone acetonide 0.1% ointment
		Mometasone furoate 0.1% cream
	Class V	Triamcinolone acetonide 0.1% cream and lotion
Low Potency	Class VI	Desonide 0.05% cream or ointment Fluocinolone acetonide 0.01% oil
	Class VII	Hydrocortisone acetate all strengths

*Note that ointments are often more potent than creams for the same medication

Calcineurin Inhibitors

Topical calcineurin inhibitors (TCI) are a class of anti-inflammatory medication that do not have any risk of skin atrophy with prolonged use. They are therefore useful in areas of the body that may be at risk of this with topical corticosteroids such as the face, or when a topical anti-inflammatory is needed for long-term, ongoing maintenance therapy.

The two available calcineurin inhibitors are pimecrolimus 1% cream and tacrolimus 0.03% and 0.1% ointment. These are thought to be roughly equivalent to a mild-moderate TCS (pimecrolimus) and a moderate TCS (tacrolimus). They are generally not effective on thick skin.

Some patients experience a burning sensation when the TCI is first applied. Fortunately, the sensation decreases after continuous use over several days.

PDE4 Inhibitors

A newer non-steroid topical medication is topical crisabarole 2% ointment. Crisaborole is a phosphodiesterase-4 inhibitor with anti-inflammatory properties and has similar efficacy to the topical calcineurin inhibitors. It can also cause a warm/hot sensation for several minutes when applied to facial skin.

Routine Skin-Care Measures: Anti-inflammatories

Corticosteroids

Topical corticosteroids (TCS) are classified by their ability to cause vasoconstriction, which roughly parallels their anti-inflammatory ability. Class I are the strongest steroids, and Class VII are the weakest.

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- For smaller involved areas, such as the hands or feet a topical steroid can be applied and then covered with a damp sock or glove with a dry overlayer
- Occlusion with dressing such as Tegaderm, plastic wrap or non-breathable gloves is another way to increase the penetration of topical corticosteroids.

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High Potency	Class II	Betamethasone dipropionate 0.05% cream Fluocinonide acetonide 0.01%
Medium Potency	Class III	Betamethasone valerate 0.1% ointment Mometasone furoate 0.1% ointment
	Class IV	Betamethasone valerate 0.1% cream Triamcinolone acetonide 0.1% ointment Mometasone furoate 0.1% cream
	Class V	Triamcinolone acetonide 0.1% cream and lotion

Low Potency	Class VI	Desonide 0.05% cream or ointment Fluocinolone acetonide 0.01% oil
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Routine Skin-Care Measures: Acne Medications

Benzoyl Peroxide

Benzoyl peroxide (BP) has several mechanisms of action helpful in acne, including antimicrobial, anti-inflammatory and comedolytic effects. It is available over the counter in preparations up to 5%, or as a prescription up to 10%. It is also available in combination with other acne medications (discussed below). Benzoyl peroxide can be irritating to the skin and so should be started slowly and increased as tolerated. Importantly, benzoyl peroxide bleaches clothing and towels, and patients should be warned of this prior to use!

Salicylic Acid

Salicylic acid is a keratolytic to exfoliate the top layer of skin. It is helpful in comedonal acne. For acne it can be purchased over the counter in concentrations of 0.5-2%. Salicylic acid can be irritating to the skin, and we recommend initially using several times a week before increasing to daily use.

Retinoids

Topical retinoids are a prescription form of Vitamin A and include tretinoin, tazarotene, trifarotene and adapalene. The price of these varies and some might be cost-prohibitive for certain patients.

- Adapalene gel or cream 0.1%, 0.3% (less irritating)
- Tretinoin gel or cream
  - 0.01%, 0.025%, 0.05%
  - 0.04%, 0.1% micronized (less irritating)
- Tazarotene gel or cream 0.05%, 0.1% (more irritating)
- Trifarotene cream 0.005% (for back acne)

Retinoids affect several genes within the cell that regulate the function of keratinocytes and the thickness of the epidermis. They are useful for the treatment of comedonal acne, wrinkles, thick psoriasis plaques, and actinic keratoses. They can be helpful in mild melasma or with photoaging spots. Finally, they are useful in wound healing and, when applied to early stretch marks, can aid in their healing. Retinoids should be applied at night to decrease their photosensitizing effect. Irritation is the main side effect and can be decreased by using several times per week initially and increasing to daily use as tolerated. Due to potential rick of absorption and subsequent teratogenicity, adapalene and tretinoin are pregnancy category C. Tazarotene is pregnancy category X and its use is prohibited during pregnancy.

Azelaic Acid

Azelaic acid is a natural product produced by Malassezia furfur, a normal commensal yeast on the skin. It is commercially available in 10 or 15% gel for the treatment of rosacea and has antibacterial, comedolytic and anti-inflammatory properties. These same properties make it useful for the treatment of acne as well. It is also used in disorders of hyperpigmentation including melasma and post-inflammatory hyperpigmentation.

Combination Topicals

Combining benzoyl peroxide with a topical antibiotic helps to decrease antimicrobial resistance. There are also combinations of topical retinoids with benzoyl peroxide or antibiotics. These help treat both the comedonal and inflammatory components of acne.

Commercially available combination products:

- Adapalene 0.1% + Benzoyl Peroxide 2.5%
- Adapalene 0.3% + Benzoyl Peroxide 2.5%
- Clindamycin 1% + Benzoyl Peroxide 5%
- Clindamycin 1.2% + Tretinoin gel 0.025%
- Erythromycin 3% + Benzoyl Peroxide 5%

Antimicrobials: Antibiotics

OTC Topical Antibiotics

Bacitracin: Bactericidal against Gm+. Can develop allergic contact dermatitis with prolonged use.

Polymyxin B: Bactericidal against Gm-. Often combined with bacitracin.

Neomycin: Bactericidal against Gm+ and Gm-, good S. Aureus coverage. Can develop allergic contact dermatitis with prolonged use. Often combined with bacitracin and polymyxin B.

Prescription Topical Antibiotics

Due to potential for development of resistance, topical antibiotics should be used as part of a treatment to treat localized infections and not as component of routine maintenance of chronic skin conditions.

Mupirocin: Bactericidal against MRSA, good Gm+ coverage. Excellent choice for impetigo as well as part of decolonization protocol for MRSA.

Fusidic acid: Bacteriostatic against Gm +, especially S. Aureus.

Erythromycin: Bactericidal against most Gm+ and C. acnes. Also has a significant anti-inflammatory effect. Used for acne and rosacea. Safe in pregnancy.

Clindamycin: Broad spectrum coverage, including anaerobic. Used for acne and rosacea. Safe in pregnancy. Bacteria readily develop resistance so must be used in combination (see above).

Antimicrobials: Antifungals

Clotrimazole, Miconazole, Ketoconazole: Active against dermatophytes, malassezia and candida. These are available OTC or can be prescribed.

Terbinafine: Fungicidal against dermatophytes and fungistatic against yeast. Available OTC or can be prescribed.

Tolnaftate: Effective against most dermatophytes, no activity against candida. Available OTC.

Nystatin: Active against yeast (candida) but not dermatophyte infections. Prescription only.

Ciclopirox olamine: Broad spectrum activity against dermatophytes and yeast, as well as both Gm+ and Gm- bacteria. Also has anti-inflammatory properties. Available as Rx only as a cream, shampoo, or nail lacquer.

Efinaconazole: Active against dermatophytes and candida. Used for onychomycosis as a 10% solution. Prescription only.

Antimicrobials: Dandruff Shampoos

Dandruff shampoos must be massaged into the scalp and left for 5-10 minutes before rinsing to have an effect. Some have efficacy in reducing skin yeast and can also be used to treat seborrheic dermatitis and pityriasis versicolour on the body.

Active ingredients of shampoos include:

- Selenium sulfide (anti-yeast)
- Ketoconazole (anti-yeast)
- Zinc pyrithione (anti-yeast)
- Salicylic acid (keratolytic for scale)
- Tar (anti-inflammatory)

Antimicrobials: Anti-pruritics

Over the counter products for anti-pruritic effects may include hydrocortisone, diphenhydramine, calamine or topical anesthetics. For severe pruritus a prescription topical corticosteroid may be used such as hydrocortisone 2.5% with menthol 0.25-0.5% and camphor 0.25-0.5%, this can be applied liberally to all body areas. Menthol and camphor are both counterirritant topical analgesics and provide an immediate soothing sensation when applied to the skin.

Antimicrobials: Other Topical Medications

Beta-blockers

Timolol maleate ophthalmologic gel or gel forming solution is used off-label for the treatment of thin, small, non-ulcerated infantile hemangiomas. It can also be used for pyogenic granulomas. Timolol maleate 0.5% is applied 1 drop to the affected area twice daily.

Cyanoacrylate

Cyanoacrylate is the main ingredient in Dermabond and is also the primary adhesive in some "superglues" available for industrial or household use. Cyanoacrylate adhesives can be used to seal fissures on fingertips and soles of the feet in order to decrease the pain and the chance of infection. Care should be taken to ensure that skin is clean prior to application of adhesive and that adhesive is completely dry before anything comes in contact with treated skin.

Aluminum chloride

Aluminum chloride can be used topically to treat hyperhidrosis. It is available over the counter as 6.25-20% solutions. This is applied nightly to affected areas until sweating decreases and then used once or twice weekly for maintenance. It is also used as a chemical cauterant and is useful for shave biopsies or after injections to control small amounts of bleeding.

Lidocaine/Prilocaine Eutectic mixture

Topical anesthetics are useful for pre-treatment of biopsy or injection sites in children. Some can be purchased over the counter and should be applied under occlusion 60 minutes prior to the procedure and the anesthetic effect lasts for approximately 1-2 hours after removal. Such topical anesthetics should only be used on intact skin and on small surface areas. Prilocaine has a risk of methemoglobinemia if used on large surface areas.

Intralesional Therapies: Corticosteroids

Intralesional corticosteroid injections can be helpful for thick, localized, persistent lesions, such as psoriasis plaques or prurigo nodules. They are also used on the scalp and beard in the management of alopecia areata. Triamcinolone acetonide (Kenalog) is available in 10mg/mL and 40mg/mL concentrations and can be diluted with saline to the desired concentration to prevent atrophy. In general, approximately 0.1-0.2mL is injected per square centimeter of skin for a total dose not exceeding 1-2mL per session. Often injections need to be repeated every 4-8 weeks.

Alopecia	2.5- 5mg/mL
Cysts (persistent deep acne nodules, painful hidradenitis lesions, inflamed epidermoid cysts)	2.5-5mg/mL
Thick or keratotic lesions	5-10mg/mL
Hypertrophic scars	10mg/mL
Keloid scars	20-40mg/mL

Physical Modalities: Phototherapy

Phototherapy can be used to treat many inflammatory skin disorders, including psoriasis, atopic dermatitis, pityriasis lichenoides, and vitiligo. There are different types of phototherapy available, the most common being narrowband UVB. Phototherapy units are available in some hospitals or in private dermatology clinics and may also be purchased for home use. Patients usually get phototherapy two to three times per week, with each session lasting only minutes. It can take several weeks before patients notice a significant improvement in their skin.

Physical Modalities: Lasers

Lasers have many applications in dermatology, including for both medical and cosmetic purposes. Different indications are treated with specific lasers. In pediatric dermatology the most common use of laser is to treat vascular conditions, such as port wine stains. Another common use is an excimer laser to treat small areas of vitiligo. Due to risks of side effects from laser therapy, treatment my well-trained certified practitioners is recommended.

Physical Modalities: Cryotherapy

Cryotherapy, or liquid nitrogen, is used to treat warts and molluscum in pediatric patients. Due to discomfort of cryotherapy, this should be used after careful consideration because causing undue pain in treatment of a benign condition is not recommended. Pre-treatment with EMLA cream can be helpful if treatment is necessary. Lesions are typically treated 3 times at each session. Warts: 5-6 seconds per cycle. Molluscum: 2-3 seconds per cycle. Cryotherapy can be applied using the end of a cotton swab, or with a specialized canister with a spray tip. Treatment can be repeated every few weeks until clearance. For treating warts it is helpful to pare the area prior to freezing to remove the hyperkeratotic debris. Expected side effects after cryotherapy include localized pain, and blistering followed by crusting. In patients with darker skin types cryotherapy may cause post-inflammatory pigment change that can be permanent.

Physical Modalities: Cantharadin

Cantharidin is a topical substance derived from the blister beetle, and when applied acts as a vesicant and causes localized blistering of the skin. It is frequently used to treat molluscum, and occasionally for small, thin warts. This is a physician-applied treatment. A small drop is applied to the lesion using the wooden end of a cotton swab and allowed to dry completely. This is a painless procedure and is tolerated even by young children. The lesion should be washed with soap and water after 2-4 hours. Some patients develop large blisters and so only a few lesions should be treated initially. Retreatment can occur after 2-4 weeks. Warts can also be treated with cantharidin; however, ring warts can develop after such therapy.

Systemic Therapies: Retinoids

Systemic retinoids are Vitamin A derivatives that affect genes involved in the regulation of keratinocyte function and epidermal thickness. Isotretinoin is used in the treatment of acne vulgaris while acitretin is used for ichthyosis and papulosquamous conditions, such as psoriasis and pityriasis rubra pilaris.

The most common side effect that is experienced by essentially all patients is dry skin and mucous membranes. All patients should use moisturizers on their skin and lips liberally, and those who wear contacts might need lubricating eye drops. Some patients may experience muscular aches, especially young and physically active patients. These subside when the medication is stopped. Possible metabolic side effects include increased triglycerides and liver enzymes. A rare but important side effect is pseudotumor cerebri, or benign intracranial hypertension. Patients should stop the medication and alert their health care practitioner if they experience symptoms of this. In addition, patients must not take tetracycline antibiotics while on oral retinoids as this increases the risk of pseudotumor cerebri. The association between mood symptoms and use of isotretinoin is controversial but patients should be screened for any mood symptoms before starting isotretinoin and at follow-up visits. Acne tends to flare when isotretinoin is first started, so it should be initiated at lower dose and increased after the first month or two of therapy.

Female patients taking oral retinoids must not get pregnant, both Isotretinoin and Acitretin are known teratogens, category X. Two forms of birth control must be used and monthly pregnancy tests should be checked. Acitretin has a potentially long half-life and so pregnancy is to be avoided for 2 full years after completion of therapy.

Retinoid laboratory monitoring: liver enzymes, fasting lipids and pregnancy test at baseline, and repeated at 2 months or after dose changes. Pregnancy test monthly for the duration of treatment. In children taking acitretin for a long duration bone age can be assessed annually by x-ray.

Systemic Therapies: Beta-blockers

Oral propranolol is approved for the treatment of infantile hemangiomas that are potentially disfiguring, ulcerated, or threatening vital function. It is typically started at 1mg/kg/day divided into 2 or 3 doses. It can be increased to 2-3mg/kg/day divided over the first few weeks of therapy as tolerated. It is important to thoroughly discuss the side effects of propranolol with parents and review instructions for use. Common side effects include cool extremities and sleep disturbance. Less common but serious side effects include hypoglycemia, hypotension, bradycardia and bronchospasm. Propranolol should be given with feeds and the dose held if there is decreased oral intake or diarrhea to decrease the risk of hypoglycemia. In very young neonates, those with any cardiac history, and those with risk of PHACES, special care should be taken when beginning propanolol and consulation with cardiology prior to initiation should be considered. In treating ulcerated hemangiomas, initiation of propranolol at usual protocol can worsen the ulcer. The dose should begin at 0.5mg/kg/day or less and increased very slowly.

Systemic Therapies: Corticosteroids

Prednisone is largely used to control acute flares of inflammatory skin conditions until the flare subsides or a steroid-sparing immunomodulatory agent can be used. There are many side effects of long-term prednisone use and is therefore not preferred. In the short-term side effects can include weight gain, hyperglycemia, hypertension, mild GI upset and a feeling of being "wired". Prednisone should be avoided in many chronic skin conditions because there is a risk of inducing a rebound flare with discontinuation.

Systemic Therapies: Methotrexate

Methotrexate is used in low doses to treat severe psoriasis, atopic dermatitis, and morphea. When used at dermatologic doses (0.2-0.5mg/kg/week) the risk of side effects is low. Possible side effects can include hepatotoxicity, pulmonary toxicity, and pancytopenias. It is also teratogenic and females must not become pregnant while on methotrexate. Many patients experience some nausea and/or feel unwell the day after taking their methotrexate. They may chose to take it on the weekend as a result. Baseline laboratory monitoring includes CBC diff, liver function tests, BUN and creatinine, hepatitis B & C and HIV screen, and a pregnancy test. Follow-up labs are CBC with differential and LFT's weekly for 2-4 weeks and then every 3 months and after any dose escalations. Kidney function should be checked annually. Folic acid 1-5mg is to be taken daily except on days of methotrexate.

Systemic Therapies: Cyclosporine

Cyclosporine is used for severe psoriasis or atopic dermatitis, and sometimes in severe adverse drug reactions. Possible side effects include renal dysfunction, hypertension, headache, hyperkalemia, hyperuricemia, hypomagnesemia, hyperlipidemia and immune compromise. Baseline laboratory monitoring is CBC, BUN, creatinine, electrolytes (including Mg), uric acid, liver function tests and fasting lipids. Blood pressure should be checked. Follow-up labs for CBC diff, BUN, creatinine and ALT are done monthly, and blood pressure is checked at each follow-up visit. Live vaccines should not be given to patients taking cyclosporine. Due to side effects, the duration of cyclosporin use should be limited.

Systemic Therapies: Biologics

Biologics refer to monoclonal antibodies, which are derived from human or animal tissue. These interact with specific parts of the immune system and are used to treat a variety of severe skin diseases. There are several classes of biologics available and this is an active area of research with more classes being studied. Current options for psoriasis include anti-TNFα, anti-IL17, anti-IL23, and anti-IL12/23 monoclonal antibodies. In atopic dermatitis there is an anti-IL4/13 monoclonal antibody. Biologics are expensive medications and generally require a patient to have failed prior treatments or to have severe disease. Live vaccines should generally not be given patients taking biologics.

Systemic Therapies: JAK Inhibitors

JAK inhibitors are new medications that are now approved for treating atopic dermatitis and are often effective in treating alopecia areata. These are expensive medications and are reserved for patients that have failed other therapies. They require careful monitoring and live vaccines should not be given.

Systemic Therapies: Antihistamines

First generation (sedating H1 Blockers) include diphenhydramine and hydroxyzine. They are used for allergic reactions. In patients with severe pruritus hydroxyzine may be prescribed to be taken at night to help with itch and sleep; however, they are not recommended for long-term therapy in children with chronic skin conditions as they may interfere with normal sleep patterns.

Second generation (non-sedating H1 Blockers) include cetirizine, loratadine, desloratadine and fexofenadine. There are several prescription second generation antihistamines now available including rupatadine and bilastine. These are used for allergic reactions and urticaria (hives).

Unfortunately antihistamines are often ineffective at controlling the itch associated with atopic dermatitis.

Systemic Therapies: Antibiotics

Cephalexin

Used commonly for impetigo, folliculitis, and secondary infections of other dermatologic conditions. Side effects include GI upset and drug hypersensitivity.

Doxycycline/Tetracycline/Minocycline

Used commonly for acne vulgaris, rosacea and periorificial dermatitis. Side effects include GI upset, reflux and photosensitivity. To decrease GI upset tetracyclines should be taken when upright, not right before bed, and with plenty of water. Prolonged use of tetracycline antibiotics should be avoided in children younger than 8 years old due to risk of dental staining, but they can be used safely in all ages for up to 3 weeks if necessary for treatment of infections (ex. Rocky Mountain Spotted Fever). A potential side effect of prolonged use of minocycline is skin hyperpigmentation. All of the tetracyclines have a possible risk of drug hypersensitivity, and minocycline can cause an autoimmune lupus-like reaction.

Trimethoprim-sulfamethoxazole

Useful for resistant acne vulgaris or skin infections. Side effects include GI upset and drug hypersensitivity.

Systemic Therapies: Antivirals

Acyclovir

Used for herpatic infections including herpes simplex, varicella, and zoster. It can also be used prophylactically in patients with recurrent herpetic infections. It is most effective to shorten the duration of illness if started within 48-72 hours of first blisters to shorten the duration of illness. Acyclovir is generally well tolerated. It can occasionally cause nausea. Some other antivirals that can be used are Famciclovir, and Valacyclovir.

Systemic Therapies: Antifungals

Terbinafine, fluconazole, itraconazole and ketoconazole are all oral antifungals that can be used in treatments of cutaneous fungal infections such as tinea capitis and onychomycosis. Fungal infection that invovles hair, nail, or large body surface areas should be treated with systemic therapy as creams are ineffective. Terbinafine is the most commonly used and is given for 4-12 weeks depending on the clinical situation. Screening of liver function prior to therapy was previously standard, but recent studies have demonstrated limited utility. Oral ketoconazole is not often used as other medications have better side effect profiles and fewer drug-drug interactions.

Systemic Therapies: Antimalarials

Hydroxychloroquine and Chloroquine

Used for the treatment of autoimmune diseases, including lupus erythematosus and dermatomyositis as well as polymorphous light eruption and solar urticaria. Possible side effects include corneal deposits, retinopathy, GI upset, and hemolysis in patients with G6PD deficiency. Baseline assessment includes an ophthalmology exam, CBC differential, renal and liver function. Labs are checked monthly for 3 months and then every 6 months. Ophthalmology exam is repeated annually.

Systemic Therapies: Oral Contraceptive Pill

Combined oral contraceptive pills (COC), containing both estrogen and progestin, can be used for the treatment of acne vulgaris in females. Many types of oral contraceptive pills exist, some of which are specifically made for treatment of acne. Potential side effects are high blood pressure, headaches, weight gain, blood clots, abdominal pain, cramping, and nausea. They may not be safe for women with a history of blood clots or migraines, with a family history of breast cancer, or who smoke.

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Versioning History

This page lists major changes to this book with major changes marked with a 1.0 increase in the version number and minor changes marked with a 0.1 increase.

Version	Date	Change
1.0	2023-01-18	Pressbook Created