



# Identification and Management of Common Dermatological Conditions in Primary Care

A Guide for those working in ACCHO's

Wednesday 27<sup>th</sup> September 2023



**AH&MRC**  
Aboriginal Health & Medical  
Research Council of NSW

## Acknowledgement of Country

We acknowledge the First Nations Peoples of the lands from where you are all dialing in today.

We are joining from the Lands of the Gadigal and Bidjigal people of the Eora Nation and we recognise their ongoing connection to culture and country.

We further acknowledge First Nations Peoples, as the Traditional Owners, Custodians and Lore Keepers of the world's oldest living culture.

We pay respects to our Elders past, present and emerging.

We extend that respect to other First Nations People joining us today



# Housekeeping



**AH&MRC**  
Aboriginal Health & Medical  
Research Council of NSW

# Learning Outcomes

- **Gain practical knowledge on how to diagnose and manage eczema for First Nations patients**
- **Understand the different presentations of cutaneous fungal infections and how to manage these conditions.**
- **Improve confidence in identifying rare entities which should not be missed**



# Presenters

- **Dr Dana Slape**, the first Aboriginal Dermatologist is a proud Larrakia woman. Dana works in a variety of settings across priority communities in urban and rural areas including the local Aboriginal Medical Service at Tharawal, Campbelltown Hospital, Darwin Hospital, and custodial facilities for children, women, and men across New South Wales (NSW) and the Northern Territory. Dana is deeply committed to growing the First Nations specialist health workforce.
- **Dr Rhiannon Russell**, Dermatology Registrar is a proud Worimi woman. She currently works in the Western Sydney region at Liverpool hospital. She hopes to return to the NSW South Coast where she is connected to the community through her training as a medical student and junior doctor. She is committed to growing the First Nations medical graduates through her mentorship at Wollongong University.
- **Dr Victoria Snaidr** is a dermatologist with a special interest in rural and remote medicine. Prior to gaining her FACD specialisation, Victoria was a GP whose interest and experience specifically in Aboriginal health was founded after working as a GP in remote Aboriginal communities in Central Australia, and further cemented during her years working at Redfern Aboriginal Medical Service. Victoria is currently working as a dermatologist in the central Sydney area and Gosford.

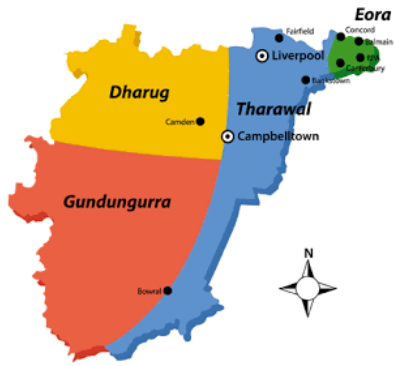




# Diagnosis & management of eczema:

a First Nations Health perspective

Sydney South West Area Health Service  
Aboriginal Nations Map



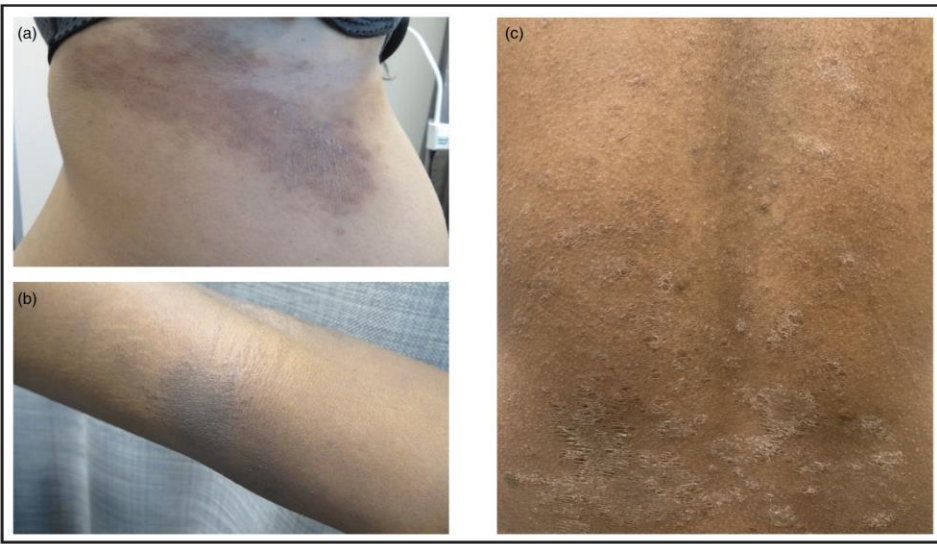
NSW HEALTH

Disclaimer: This map indicates only the general location of larger groupings of people, which may include smaller groups such as clans, dialects, or individual languages in a group. The boundaries are not intended to be exact. This map is not suitable for use in native title or other land claims.









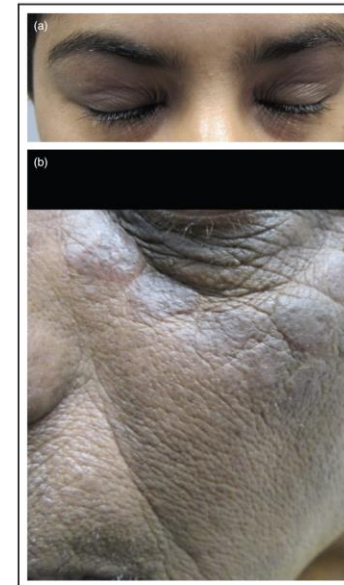
**Figure 1** Images of patients with skin of colour demonstrating the psoriasisiform appearance of atopic dermatitis on (a) the lateral chest, (b) arm and (c) lower back.



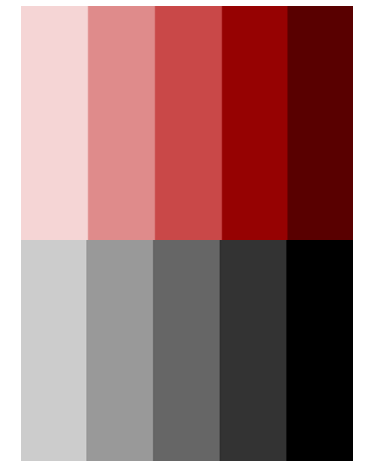
**Figure 2** Violaceous hyperkeratotic plaques with excoriations seen on (a) the lateral leg of a patient with skin of colour with severe atopic dermatitis. (b) Active lichenified eczema with a greyish hue.



**Figure 3** Papular variant of atopic dermatitis on the upper chest of a patient of Sri Lankan ethnicity.



**Figure 4** (a) Eyelid hyperlinearity and dyspigmentation in a young female with atopic dermatitis. (b) Hyperpigmented, lichenified, grey plaques with surrounding eyelid accentuation and infraorbital creases in a middle-aged man with atopic dermatitis.



# Atopic dermatitis in skin of colour. Part 2: considerations in clinical presentation and treatment options

Christian Gan<sup>1,2</sup>, Satveer Mahil<sup>3</sup>, Andrew Pink<sup>3</sup> and Michelle Rodrigues<sup>1,4,5</sup>

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<sup>2</sup>Department of Dermatology, St Vincent’s Hospital, Melbourne, VIC, Australia

<sup>3</sup>St John’s Institute of Dermatology, Guy’s and St Thomas NHS Foundation Trust and King’s College London, London, UK

<sup>4</sup>Department of Paediatrics, The University of Melbourne, Parkville, VIC, Australia

<sup>5</sup>Chroma Dermatology, Pigment and Skin of Colour Centre, VIC, Australia

**Eczema = Dermatitis**



# Eczema – more than just a scratch

## COMMON COMMON COMMON

One in five children

One in ten adults

50% grow out of it (but 50% don't)

Increasing incidence (& awareness)

Onset usually before 5y

Family history biggest risk factor

Urban-living First Nations children & adolescents overrepresented

Higher rates of complications



# Eczema- Complex pathophysiology

## Epidermal Barrier:

transepidermal water loss

filaggrin and other structural proteins and molecules assoc with keratinocytes

## Genetics

polygenic

family history

## Innate immunity of the skin

physical barrier (stratum corneum and intercellular junctions)

antimicrobial peptides, cytokines/chemokines

antigen-presenting cells, keratinocytes, mast cells, neutrophils

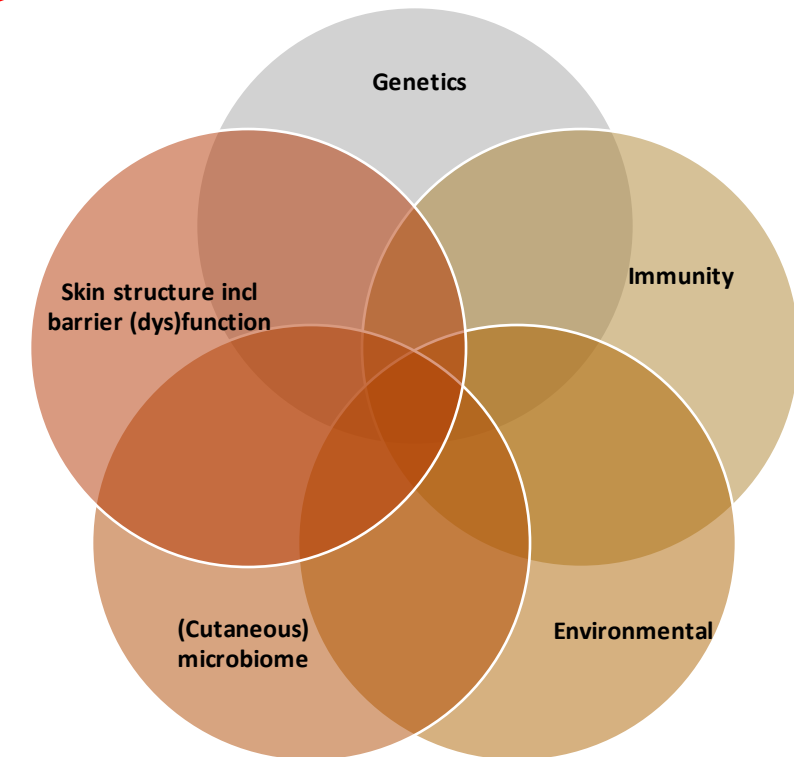
skin-resident normal microbial flora

## Adaptive immunity of the skin

TH2 dysregulation (IL-4, IL-13, IL-31, IL-22) → IgE

Staph + other irritants contribute ++++

**COMPLEX CONTRIBUTORS  
MANY CONTRIBUTORS  
REMAINS POORLY UNDERSTOOD**



# Eczema- history

Itch

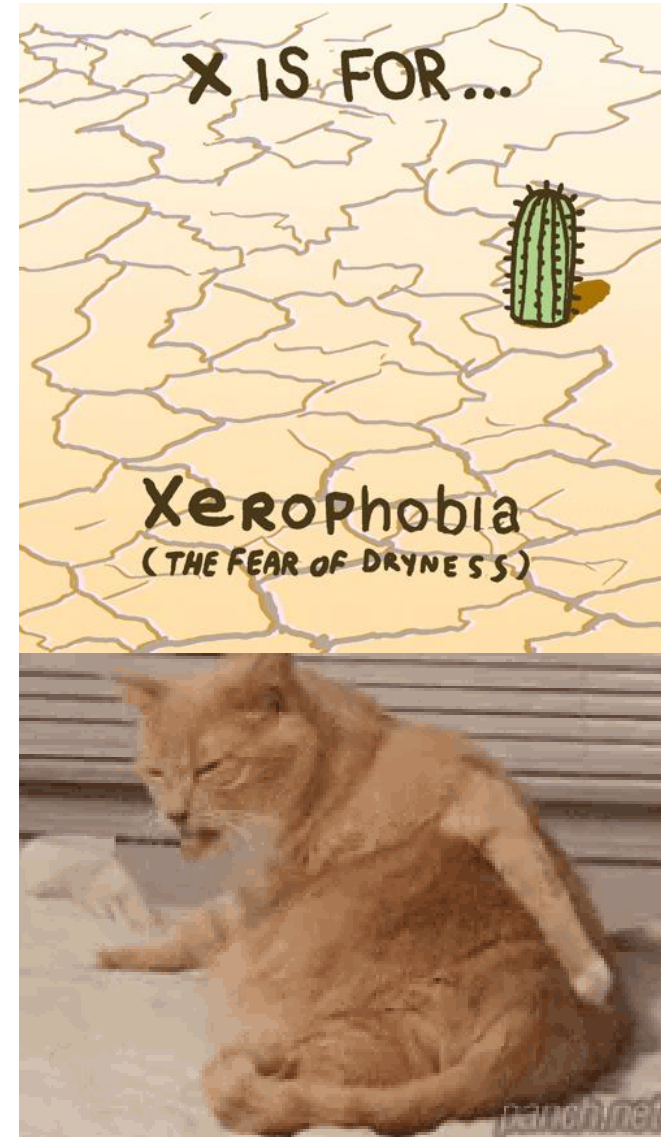
Dryness

Episodic flares

Classic distribution

Secondary infection

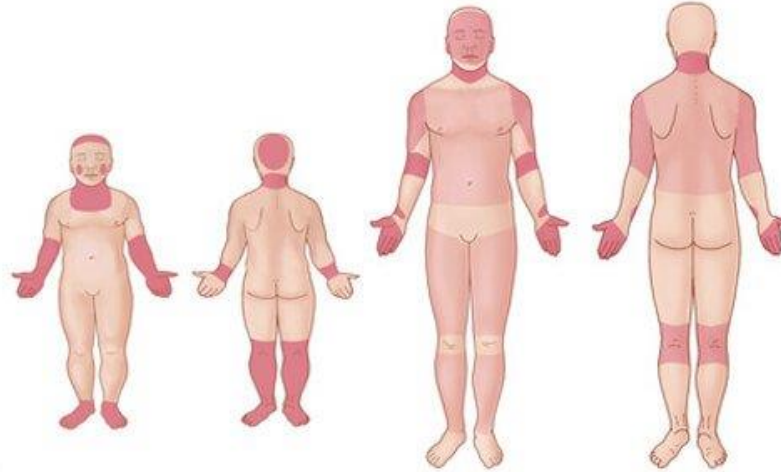
Take time to learn about how it effects them



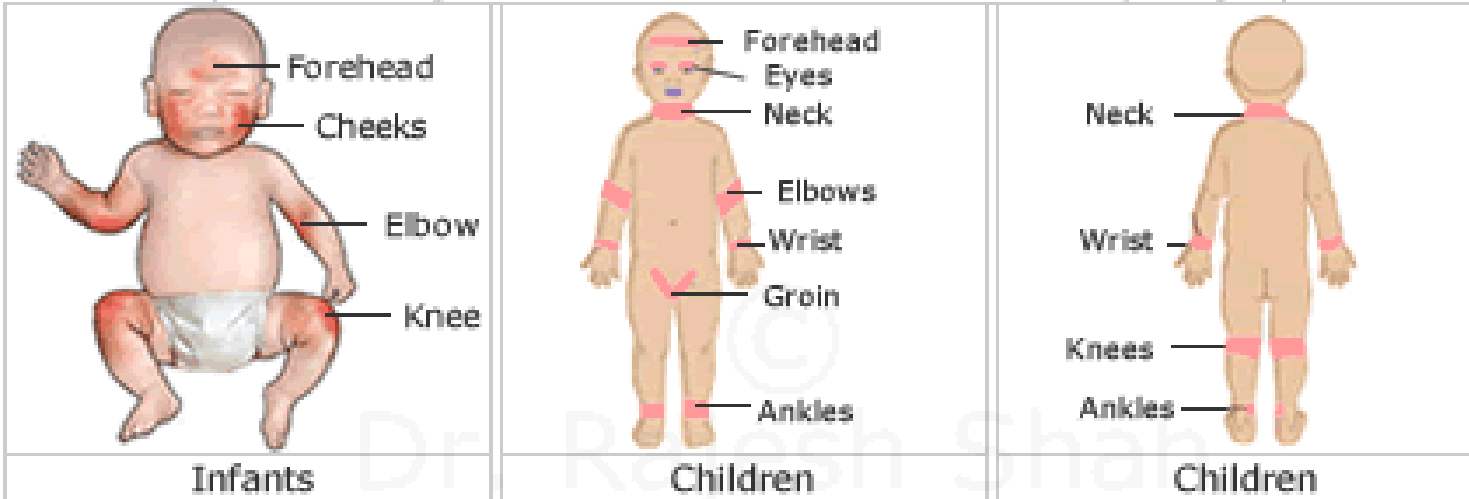
# Eczema- presentation



## Common Sites of Eczema Outbreaks



## Dermatological distribution of the skin lesions as per age



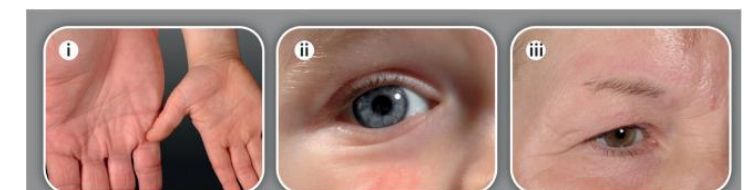
A Typical clinical appearance and location of atopic dermatitis at different ages



B Close-up view of skin



C Associated atopic stigmata





# Eczema- Complications

Bacterial infection

Viral infection

Fungal infection

Parasitic infection

Failure to grow, thrive, develop normally

Anaemia of chronic disease

School and social complications

Hobbies and occupational considerations

Depression, anxiety

# Infectious complications of eczema: bacteria

MRSA/MSSA >>>> Strep

Always swab

History important

Sick or well?

Localised or widespread?

Concurrent eczema mgmt





# Infectious complications of eczema: eczema herpeticum (herpes virus)

Well defined punched out painful ulcers

Contacts common

Always swab

Treatment is safe

Concurrent management of eczema a must



# Eczema- how to confirm a diagnosis?

Major criteria (3 or more required)	Minor criteria (3 or more required)
Pruritus	Xerosis
Typical morphology and distribution <ul style="list-style-type: none"><li>• Flexural lichenification or linearity in adults</li><li>• Facial and extensor involvement in infants and children</li></ul>	Ichthyosis, palmar hyperlinearity, or keratosis pilaris
Chronic or chronically-relapsing dermatitis	Immediate (type 1) skin-test reactivity
Personal or family history of atopy (asthma, allergic rhinitis, atopic dermatitis)	Raised serum IgE
	Early age of onset
	Tendency toward cutaneous infections (especially <i>S. aureus</i> and herpes simplex) or impaired cell-mediated immunity
	Tendency toward non-specific hand or foot dermatitis
	Nipple eczema
	Cheilitis
	Recurrent conjunctivitis
	Dennie-Morgan infraorbital fold
	Keratoconus
	Anterior subcapsular cataracts
	Orbital darkening
	Facial pallor or facial erythema
	Pityriasis alba
	Anterior neck folds
	Itch when sweating
	Intolerance to wool and lipid solvents
	Perifollicular accentuation
	Food intolerance
	Course influenced by environmental or emotional factors
	White dermographism or delayed blanch

**LOTS OF DIFFERENT PRESENTATIONS & VARIABLE MORPHOLOGY**  
**COMPLEX PRESENTATION & VARIABLE MORPHOLOGY**  
**COMMON THEM OF ITCH/SCRATCH, DRYNESS, CLASSIC SITES**

# Diagnostic criteria

Major criteria (3 or more required)

Morphology and distribution  
Facial lichenification or linearity in adults  
Flexural and extensor involvement in infants and children  
Chronicity or recurrently-relapsing dermatitis  
Family history of atopy (asthma, allergic rhinitis, eczema or dermatitis)

Minor criteria (3 or more required)

Xerosis  
Ichthyosis, palmar hyperlinearity, or keratoderma  
Immediate (type 1) skin-test reactivity  
Raised serum IgE  
Early age of onset  
Tendency toward cutaneous infections (e.g., staphylococci, aureus and herpes simplex) or impaired wound healing  
Tendency toward non-specific hand or foot dermatitis  
Nipple eczema  
Cheilitis  
Recurrent conjunctivitis  
Dennie-Morgan infraorbital fold  
Keratoconus  
Anterior subcapsular cataracts  
Orbital darkening  
Facial pallor or facial pallor  
Pityriasis alba  
Anterior neck fold  
Itch when sweating  
Intolerance to wool  
Perifollicular accentuation  
Food intolerance  
Course influenced by seasonality  
White dermographism

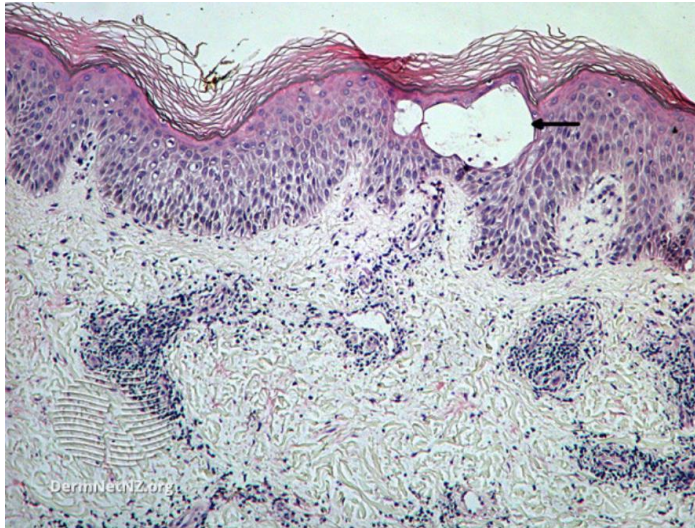
**LOTS OF DIFFERENT PRESENTATIONS & VARIABLE MORPHOLOGY  
COMPLEX PRESENTATION & COMMON THEM OF ITCH/SCRATCH, DRYNESS, CLASSIC SITES**



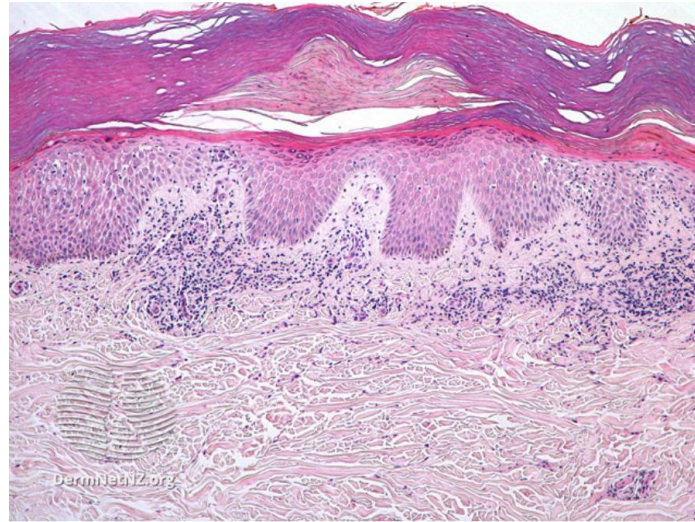




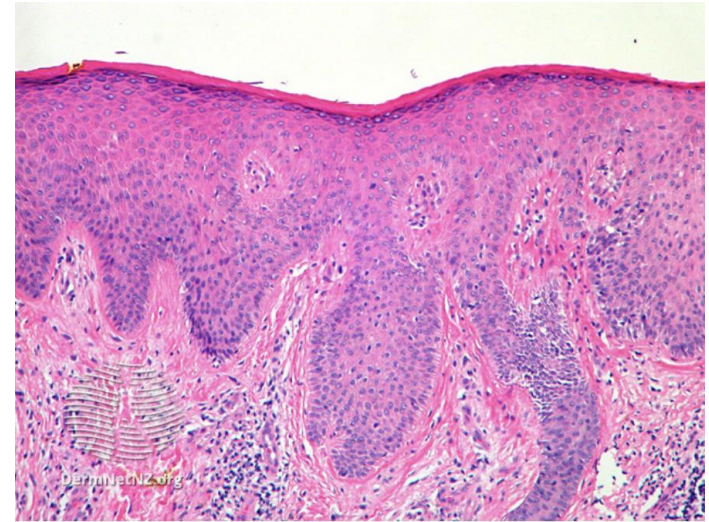
# Eczema- histological journey



Acute eczema pathology

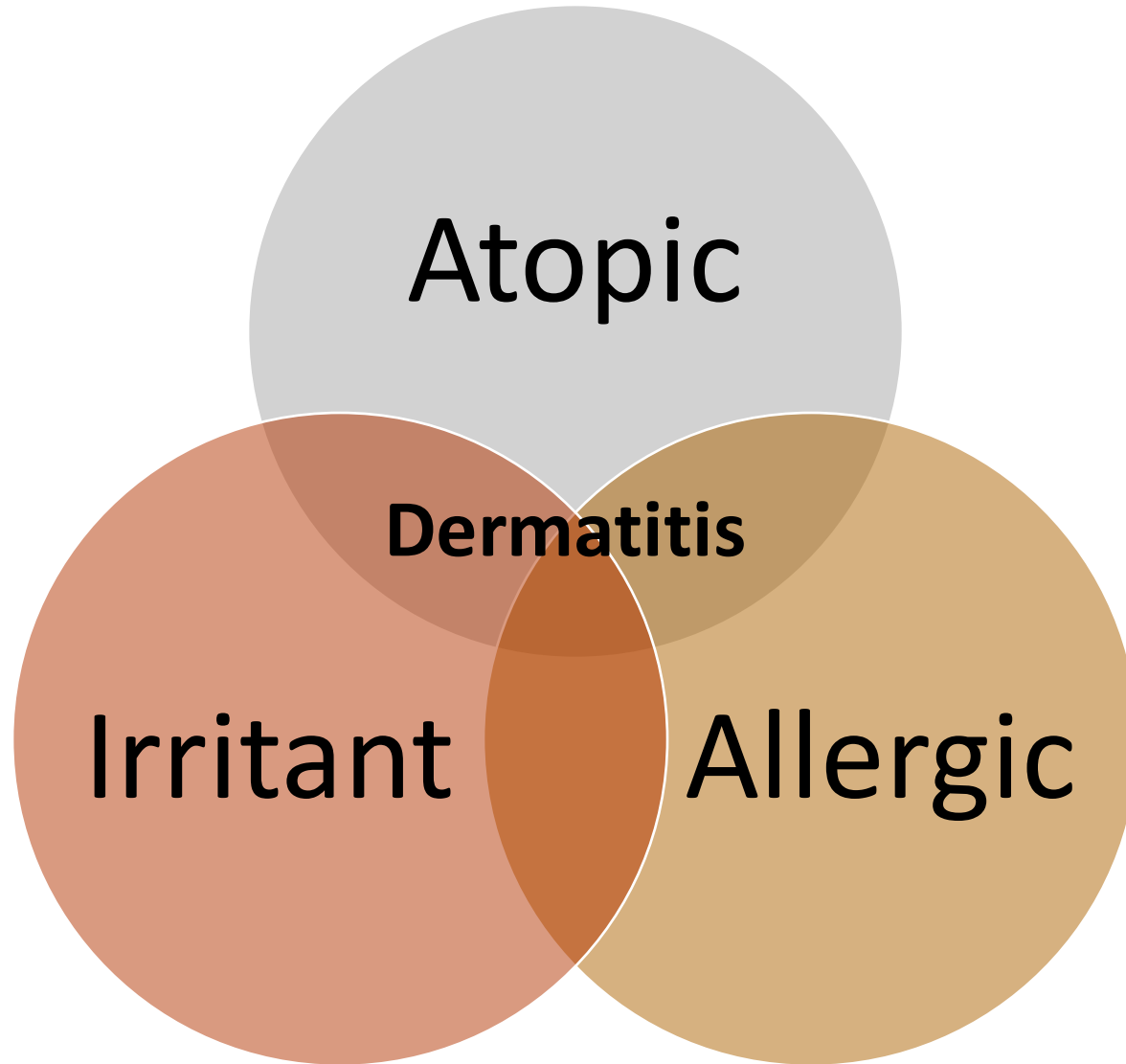


Subacute eczema pathology



Chronic eczema pathology

Physical  
Mechanical  
Chemical } irritation



Delayed type IV  
hypersensitivity reaction  
induced by a specific  
substance/s

# Eczema – Management considerations

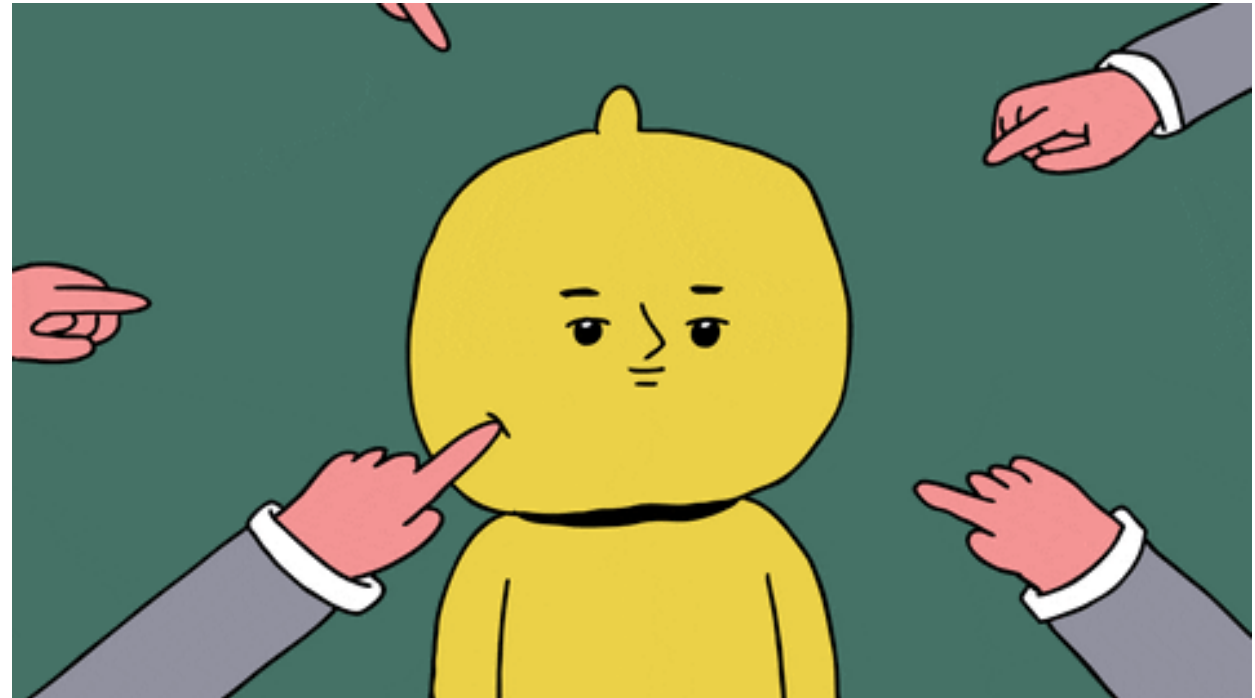
Identify and optimise triggers

Reduce itch by reducing inflammation

Strengthen skin barrier

Minimise/prevent secondary infection

Flare identification and spot fire management





# Eczema- Treatment:

## Identify and optimise triggers

Heat & sweat

Systemic illness (including systemic steroid withdrawal)

Friction incl picking/scratching

Irritants

HDM, pet fur, carpet, sandpits

Herbal, botanical topicals (tea tree, aloe)

Sorbolene lotions (& other highly preserved  
personal care products)

Wet wipes



# Eczema- Treatment: Identify and optimise triggers



Minimise heat

Short cool showers

Avoid scratching

Reduce exposure to triggers

No wipes, pump-lotions, soaps

Prevent infection

**EAT ALL THE THINGS**

**IF YOU CAN EAT IT OR GROW IT IN  
YOUR GARDEN, IT DOESN'T BELONG  
ON YOUR SKIN**

**Be mindful of what you ask people to do...  
they may end up doing nothing**



**KEEP IT SIMPLE**





# topical steroid ointments

THICK LIKE PEANUT BUTTER, NOT THIN LIKE VEGEMITE

Until it is better

# **Eczema- bleach baths**

**Half a coffee mug of  
bleach in a full bath of  
warm H<sub>2</sub>O**

**Soak 15min**

**Weekly if bad**

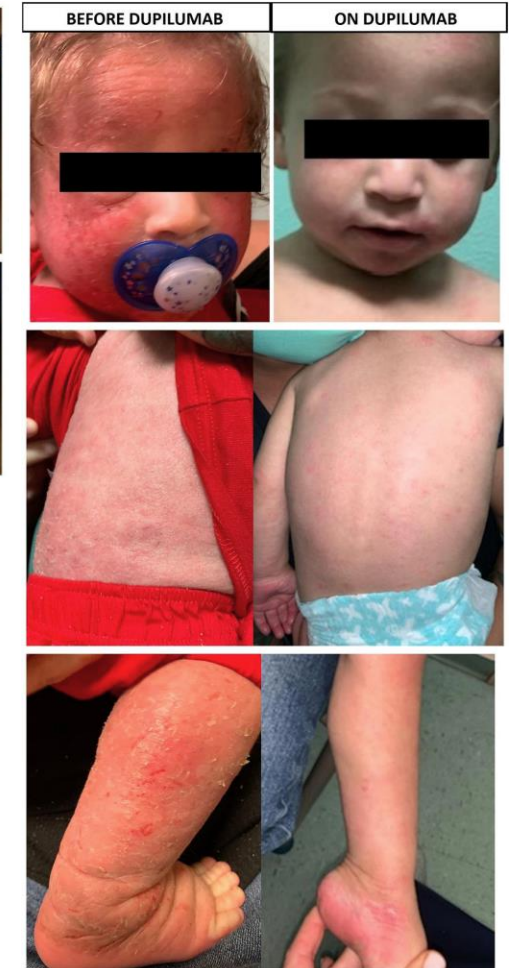


# If severe & everything else fails



BEFORE DUPILUMAB  
Eczema patches  
Onychomycosis

AFTER DUPILUMAB



Dupilumab = Dupixent

New monoclonal antibody injection treatment

Safe & effective

Few side effects

Dermatologist/Immunologist (workforce deserts an issue)



# Don't forget

## Associations

Atopic march

Asthma

Hayfever

Immune dysregulation

Iatrogenic

Disease-related

Inherited

## Mimics: dry, scaly

Psoriasis

HTLV-1

Crusted scabies

Dermatophytes

Nutritional disorders

## Mimics: itchy

Lichen planus

Urticaria (acute)

Some medications

Other occult infections

# Eczema – Don't underestimate the impact

## **Time**

- off school/off work
- treating & recovering
- flare management
- care giving
- modifications
- productivity

## **Medical complications**

- infection
- stunting/failure to thrive
- anaemia

## **Money**

- emollients
- washes
- different topicals
- antihistamines

## **Psychological distress**

- shame
- isolation (contagious)



Healthy Skin, healthy kids

# Further reading



ORIGINAL ARTICLE | [Open Access](#) |

## The burden of atopic dermatitis and bacterial skin infections among urban-living Indigenous children and young people in high-income countries: A systematic review

Bernadette M. Ricciardo MBBS, DCH, FACD , Heather-Lynn Kessar BSc, MD, Prasad Kumarasinghe MBBS, MD, FACD, Jonathan R. Carapetis MBBS, FRACP, PhD ... [See all authors](#)

First published: 09 November 2022 | <https://doi.org/10.1111/pde.15153>

**Funding information:** Australian Government Research Training Program; The Australian National Health and Medical Research Council (NHMRC); Wesfarmers Centre of Vaccines & Infectious Diseases

*Clin Exp Dermatol* 2023; **48**: 1091–1101  
<https://doi.org/10.1093/ced/llad162>  
Advance access publication date: 29 April 2023

Clinical and Experimental Dermatology  
Review Article

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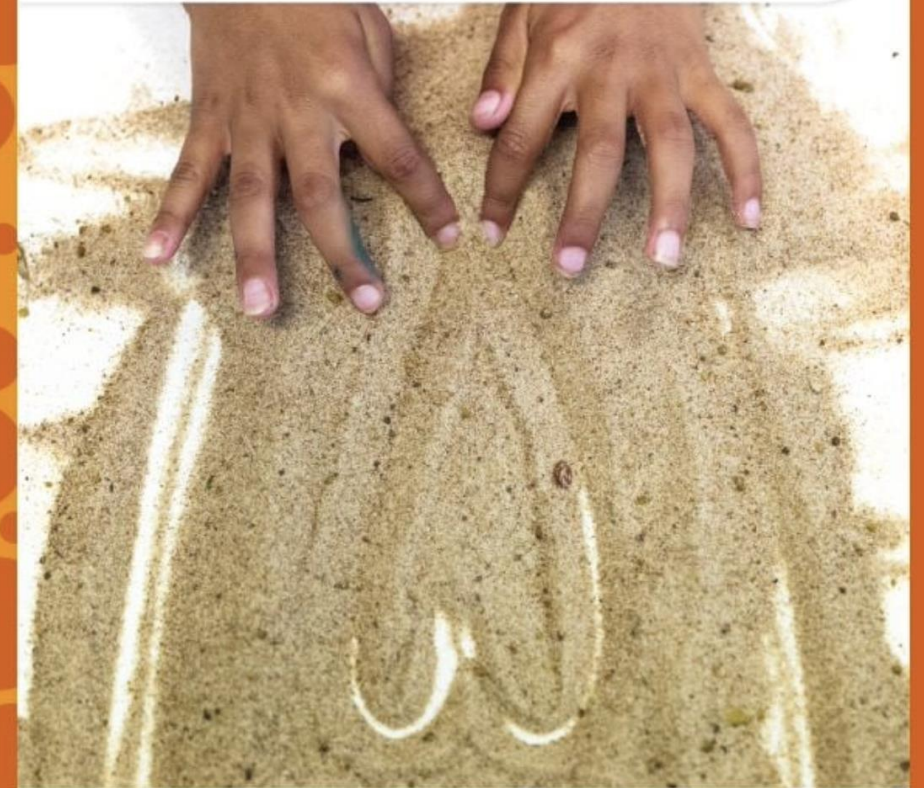
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Correspondence Michelle Rodrigues. Email: [dr.rodrigues@gmail.com](mailto:dr.rodrigues@gmail.com)

National Healthy Skin  
Guideline: for the Prevention,  
Treatment and Public Health  
Control of Impetigo, Scabies,  
Crusted Scabies and Tinea  
for Indigenous Populations and  
Communities in Australia – 1st edition



The Australian Healthy Skin Consortium 2018



# Identification and Management of Fungal Infections and Strongyloides

Dr Rhiannon Russell

Dermatology Registrar

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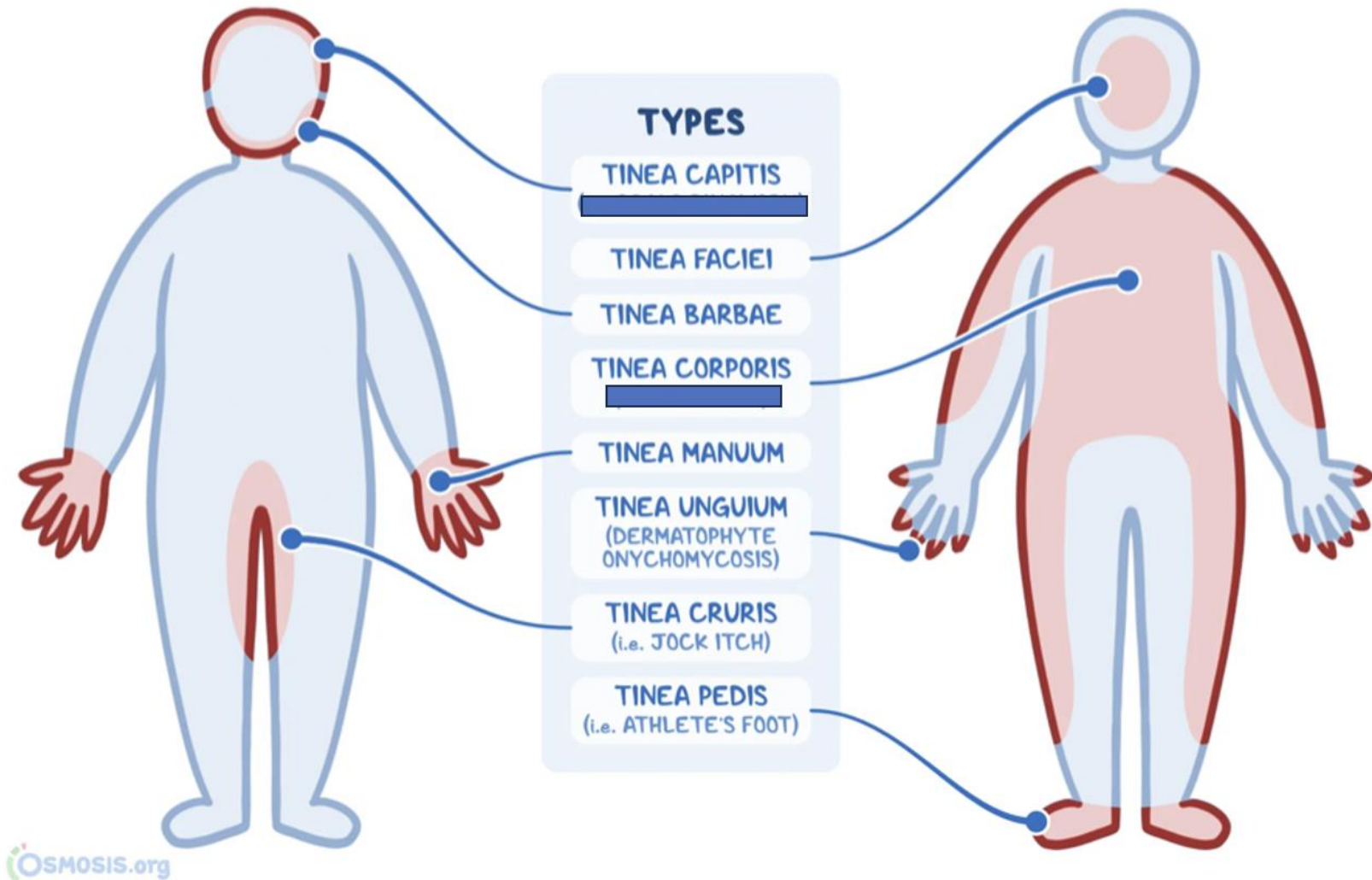
# What will we cover today:

- Superficial fungal infections
  - Tinea Capitis
    - Kerion
  - Tinea Corporis
  - Tinea Pedis
- Yeasts
  - Malassezia
  - Candida
- Strongyloides



TINEA

# Types of Tinea by location



# General Risk factors for developing Tinea

- Animals
  - Cats (Commonly seen in children with new cats) caused by *Microsporum Canis*
  - Horses – *Trichophyton.Equinum*
- Household crowding
  - Spread through bedding, couches, toys, hairbrushes
- Lower SES
- Humid environments
- Children
- DM
- Immunodeficiency
- Hyperhidrosis



# Tinea Capitis



*Trichophyton Tonsurans*





## Inflammatory

### **Diffuse Pustular**

- Patchy alopecia
- Pustules or folliculitis
- Signs of secondary bacterial infection

### **Kerion**

- Painful red boggy plaque
- Associated alopecia
- Scattered pustules
- Can cause permanent hair loss

### **Favus**

- Rare
- Matted hair
- Yellow, crusted cup shaped lesions at base of hair

## Non –Inflammatory

### **Grey Patches**

- Fine Scaling
- Patches of Alopecia
- Grey due to spores covering hair
- Variable erythema.

### **Black dots**

- dots are due to broken hair shafts

### **Diffuse Scale**

- Generalised dandruff +/- alopecia

## Grey Patches



## Black Dots



## Favus



# KERIONS



# Tinea Corporis









# Variations to be aware of

- Tinea imbricata – polycyclic plaques with thick scale that are very pruritic → commonly seen in pacific tropical regions
- Tinea incognito. Treatment with topical corticosteroids to pruritic red eruptions that are in fact tines means the tinea eruption is suppressed
- Majocchi Granuloma → usually an asymmetrical irregular scaly plaque with follicular papules, pustules and nodules usually on the lower leg.

# Tinea Incognita



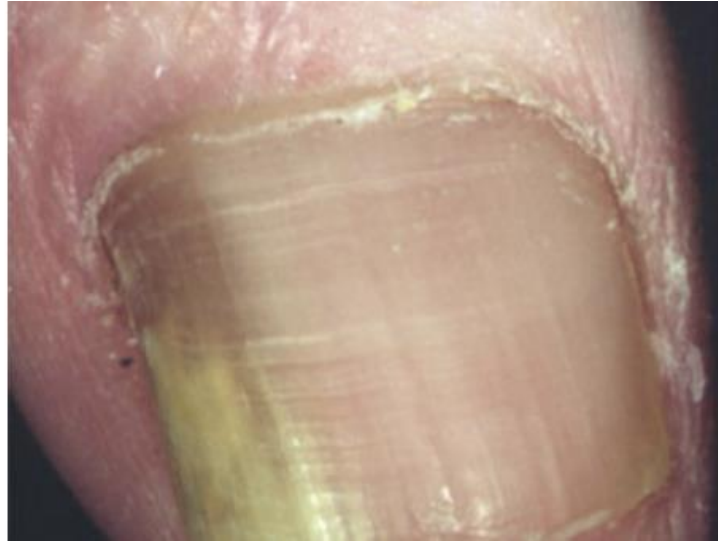
# Tinea Pedis



# Onychomycosis

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# Investigations

Nail clippings – take this from the crumbling free edge of the effected nail as the most proximal areas of the dystrophic nail yield the best results on microscopy

Scrapings – with a 15 blade can be used on the superficial white areas and borders.

Suspected Tinea Infection  
 •Conduct fungal scraping or fungal nail clipping

Positive  
 Assess the extent of infection

Negative  
 Consider differentials  
 Refer to Dermatology  
 Trail of topical anti- fungal infection

Localised

Widespread

Start topical anti-fungal until clinical resolution

Start oral anti-fungal (Terbinafine or Griseofulvin)

If treatment failure:  
 • Incomplete treatment which may require oral anti-fungal  
 • Re-infection

Resolution = continuation of prevention methods  
 1. Keep area affected clean and dry  
     - dry feet and body after showering  
     - Avoid occlusive footwear  
     - Clean showering areas with bleach  
 2. Loose fitted clothing  
 3. Check other household members and pets

Subtypes	Treatment (Refer to ETG for further details)
Capitis -including Kerion	Terbinafine 250 mg (child less than 20 kg: 62.5 mg; child 20 to 40 kg: 125 mg) orally, once daily for 4 weeks. If <i>Microsporum</i> species is identified, use: griseofulvin 20 mg/kg (up to 500 mg) orally, once daily for 6 to 8 weeks
Corporis + variant's	Terbinafine – 250mg per day for one to two weeks Griseofulvin - 500 mg per day for two to four weeks
Nails	terbinafine 250 mg (child less than 20 kg: 62.5 mg; child 20 to 40 kg: 125 mg) orally, once daily until clinical clearance riseofulvin 500 to 1000 mg orally, daily until clinical clearance.





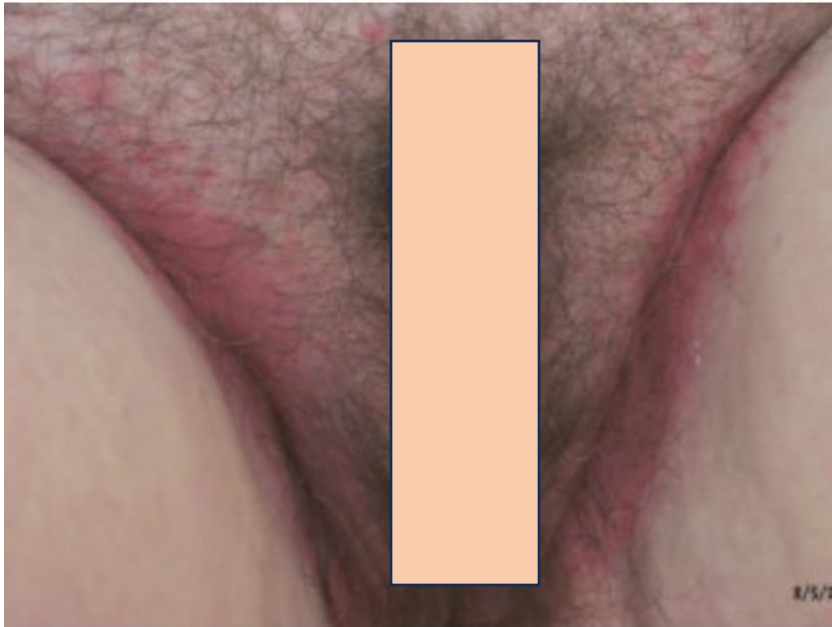
YEASTS

# YEASTS – Malassezia Species



Pityriasis Versicolor

# Yeasts – Candida Species



# Treatment

- Topical treatment:
  - Propylene glycol
  - Sodium thiosulphate solution
  - Selenium sulphide
  - Topical/oral azoles including clotrimazole, miconazole, econazole and ketoconazole in various formulations
  - Terbinafine gel
  - Ciclopirox cream/solution
- If persistent or extensive can use ketoconazole oral 200mg for 7-10 days but often will require specialist approval
- Recommend using an anti-dandruff shampoo twice per week to prevent relapse in the future
- Advise patient it will take months for skin to return to normal colour

STRONGYLOIDES

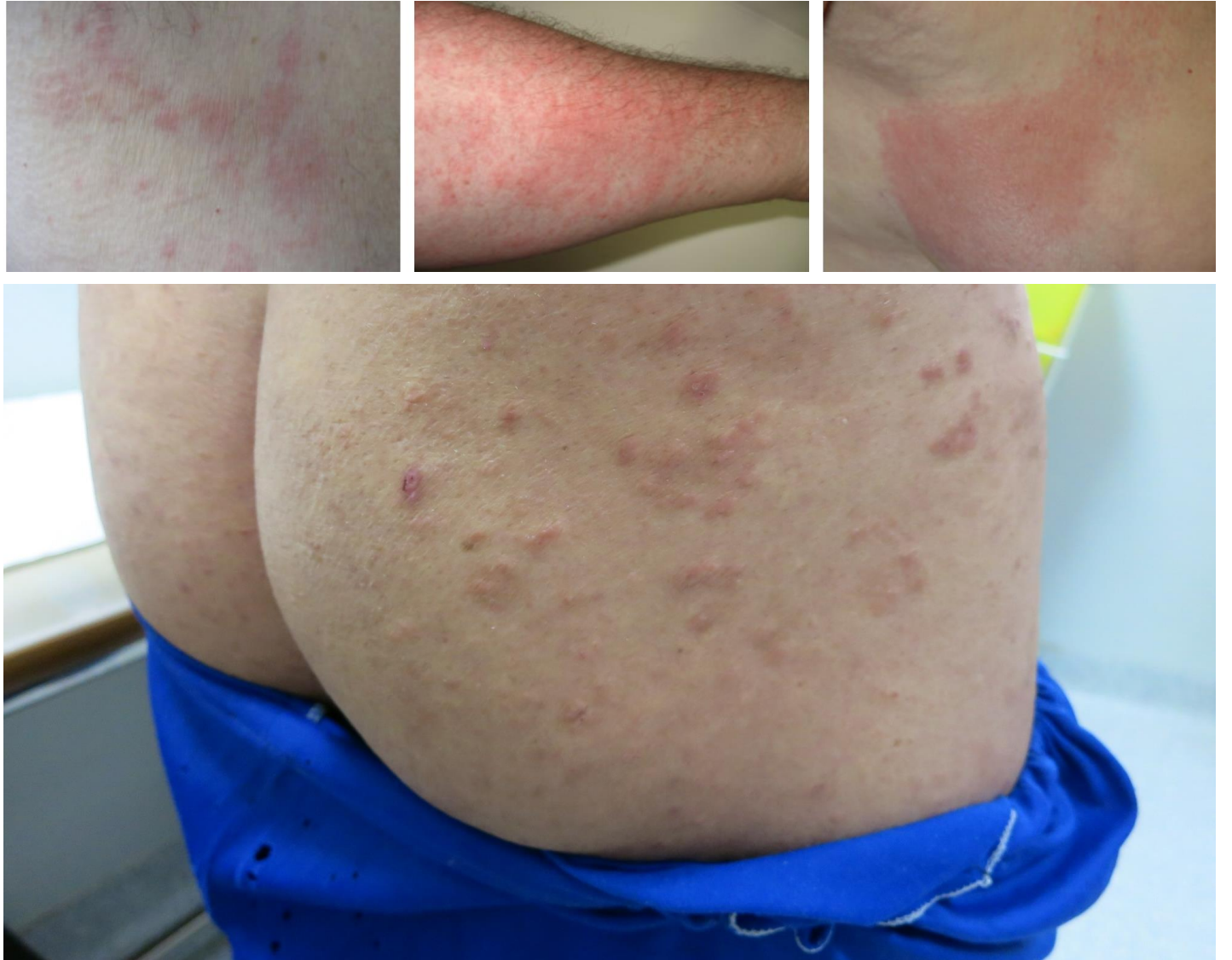


Cutaneous  
Manifestations  
Strongyloidiasis  
infections

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# Urticarial Dermatitis



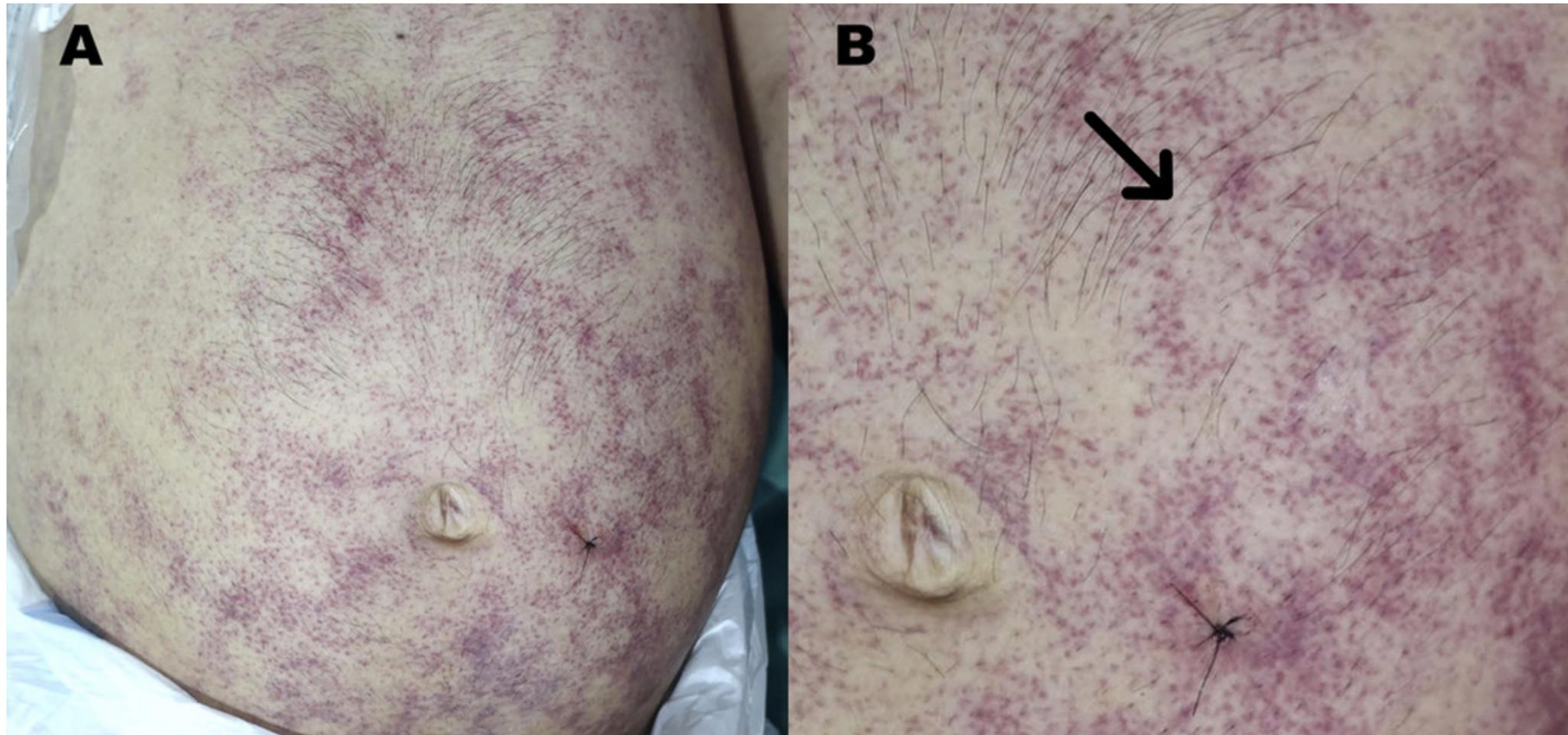


Figure 1.

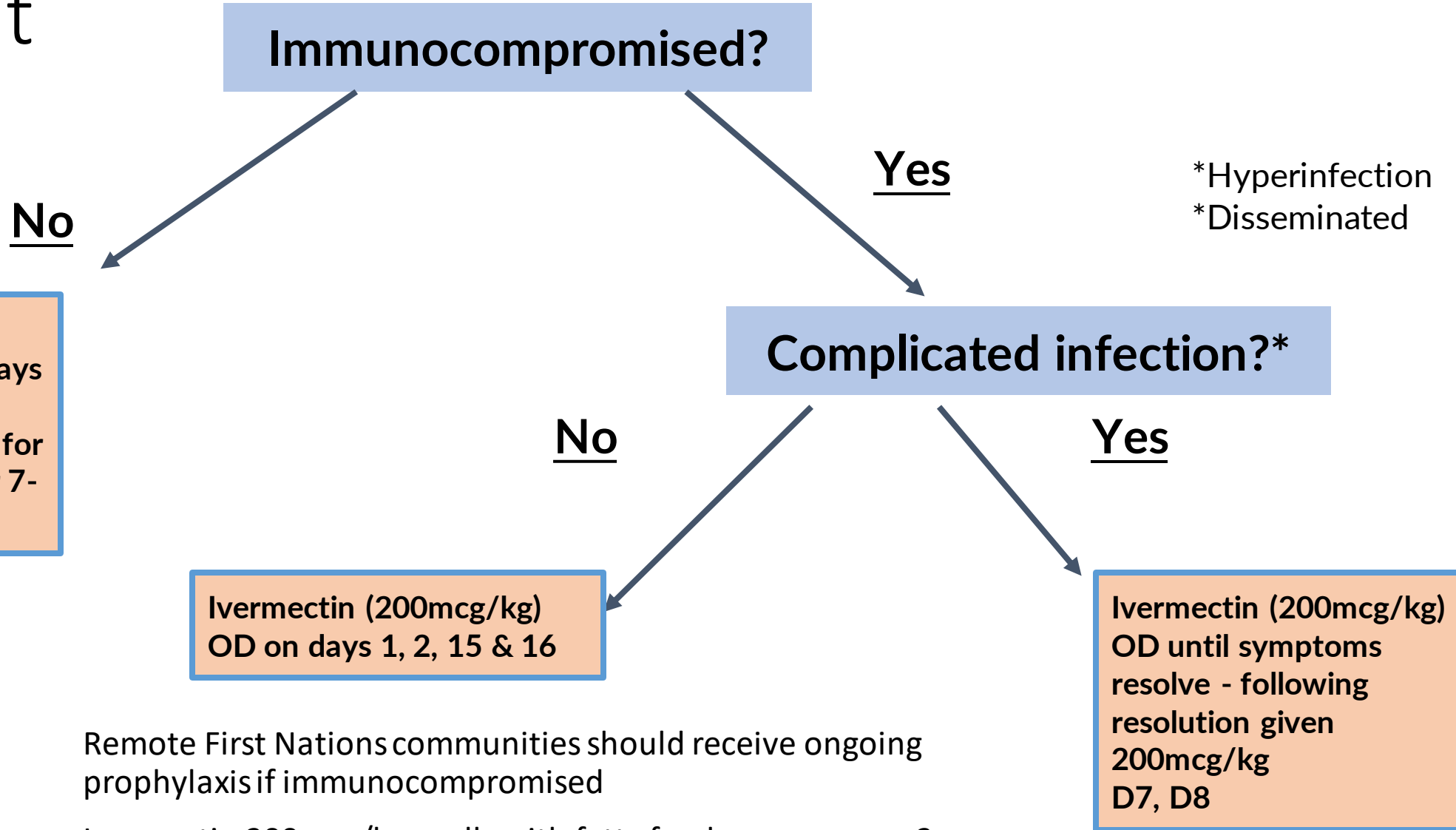
(A) Purpuric rash involving the periumbilical area, abdomen, and flank in an HIV patient with disseminated strongyloidiasis. (B) Close-up of the rash showing purple macules and papules in the periumbilical area resembling thumbprints (arrow). This figure appears in color at [www.ajtmh.org](http://www.ajtmh.org).

Citation: The American Journal of Tropical Medicine and Hygiene 105, 4; [10.4269/ajtmh.21-0464](https://doi.org/10.4269/ajtmh.21-0464)

# Hyperinfection and Disseminated Strongyloidiasis



# Treatment Pathway



Ivermectin (200mcg/kg)  
STAT then repeat in 7 days  
OR  
Albendazole 400mg BD for  
3 days then repeat after 7-  
14 days

Ivermectin (200mcg/kg)  
OD on days 1, 2, 15 & 16

Ivermectin (200mcg/kg)  
OD until symptoms  
resolve - following  
resolution given  
200mcg/kg  
D7, D8

Remote First Nations communities should receive ongoing prophylaxis if immunocompromised

Ivermectin 200mcg/kg orally with fatty food – once every 3 months

# Rare entities not to be missed

Victoria Snaidr

Wednesday 27<sup>th</sup> September 2023

# Topics

- Lupus
  - Cutaneous manifestations of SLE, DLE
  - Lip lupus
  - Neonatal lupus
- The great mimickers
  - Syphilis
  - Leprosy
- Drug reactions

# Lupus Erythematosus

- High prevalence of lupus –systemic and cutaneous - Indigenous Australian population, especially women
- An autoimmune inflammatory condition characterized by erythematous patches, +/- scale, +/- scarring
- Genetic + environmental triggers (smoking, UV, medications)
- More severe
  - Potentially identified later
- Different symptoms
  - Less photosensitivity
  - More renal involvement
- Differentials: psoriasis, eczema, infections (tinea, leprosy, syphilis),
- Complications: scarring/cosmetic impact, cardiovascular, neurological, renal, rheumatological

# Lupus – cutaneous manifestations

- DDx: psoriasis, infections, 'ring worm' eczema, scars, sunburn
- Acute
  - Malar and skin lesions as part of SLE
  - photodistributed
- Subacute lupus
  - Annular
  - Papulosquamous
  - Neonatal
- Chronic
  - Discoid lupus
  - Lupus tumidus
  - Lupus panniculitis
  - Chillblain lupus
- Lip lupus



# New EULAR/ACR criteria for the classification of SLE

Clinical domains	Points
<b>Constitutional domain</b> Fever	2
<b>Cutaneous domain</b> Non-scarring alopecia Oral ulcers Subacute cutaneous or discoid lupus Acute cutaneous lupus	2 2 4 6
<b>Arthritis domain</b> Synovitis or tenderness in at least 2 joints	6
<b>Neurologic domain</b> Delirium Psychosis Seizure	2 3 5
<b>Serositis domain</b> Pleural or pericardial effusion Acute pericarditis	5 6
<b>Hematologic domain</b> Leukopenia Thrombocytopenia Autoimmune hemolysis	3 4 4
<b>Renal domain</b> Proteinuria > 0.5 g/24 hr Class II or V lupus nephritis Class III or IV lupus nephritis	4 8 10

Immunologic domains	Points
<b>Antiphospholipid antibody domain</b> Anticardiolipin IgG > 40 GPL or anti-β2GP1 IgG > 40 units or lupus anticoagulant	2
<b>Complement proteins domain</b> Low C3 or low C4 Low C3 and low C4	3 4
<b>Highly specific antibodies domain</b> Anti-dsDNA antibody Anti-Sm antibody	6 6

**REFERENCE: Aringer et al. Abstract #2928. 2018 ACR/ARHP Annual Meeting**

- ✓ Classification criteria are not diagnosis criteria
- ✓ All patients classified as having SLE must have ANA ≥ 1:80 (entry criterion)
- ✓ Patients must have ≥ 10 points to be classified as SLE
- ✓ Items can only be counted for classification if there is no more likely cause
- ✓ Only the highest criterion in a given domain counts
- ✓ SLE classification requires points from at least one clinical domain

@Lupusreference

# Lupus – an approach

## MEDICATIONS REVIEW

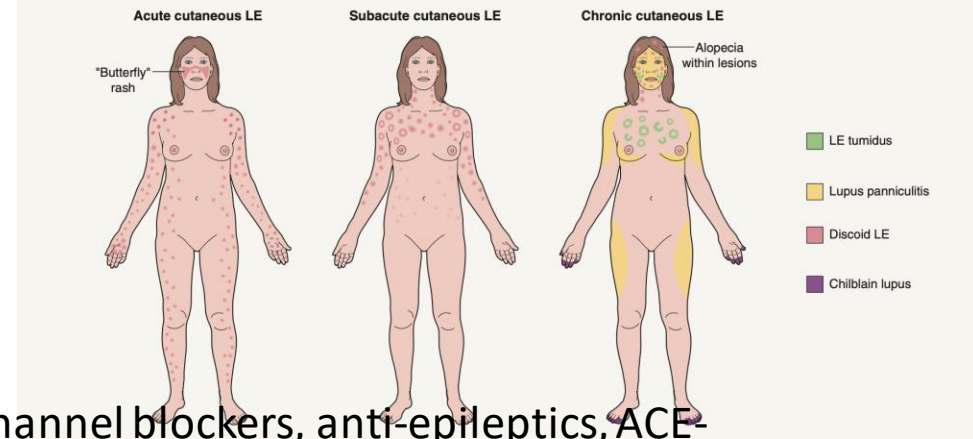
- Eg: terbinafine, thiazide diuretics, proton pump inhibitors, calcium channel blockers, anti-epileptics, ACE-inhibitors, beta blockers, ranitidine

## CLINICAL EXAMINATION AND SYSTEMIC REVIEW

- Skin lesions: annular, scale, erythematous, bullous, scar-like central atrophy, hyperpigmentation
- Location/pattern : widespread versus photo-distributed, “butterfly”
- Special sites: scalp (hair), ears, nails, oral mucosa
- CVS, Respiratory, Rheumatology (joint and neuro), Neurological (memory, cognition)

## INVESTIGATIONS

- Skin scrapings and biopsy
- Bloods: ANA, ENA, dsDNA, FBC, EUC, LFTs, ESR, CRP, C3, C4, anti-phospholipid antibodies + pre-medications screening as required
- Urine: urinalysis including red cell casts and protein
- CXR
- +/- others as per CE/systems review



(Bolognia et al. *Dermatology 4<sup>th</sup> edition. Elsevier 2018*)

# Treatment - Lupus

- Avoid
  - UV, smoking, triggering medications
- Topical
  - Moderate to potent topical corticosteroids
  - Calcineurin inhibitors
- Intralesional corticosteroids
- Oral agents
  - Plaquenil 200mg BD (5mg/kg/daily)
  - Chloroquine
  - Retinoids
  - Dapsone
  - Immunomodulators: Methotrexate, mycophenolate mofetil, azathioprine



# Lip lupus

- Red, friable, cracking lips (early → Chronic ulceration, erosion, discolouration, crusting, thickening)
- Feature of SLE or manifestation of cutaneous DLE
- Increased rates in Indigenous Australian women
- Either isolated or as part of SLE
- DDX: candidiasis, syphilis, strep, HSV, lichen planus, FDE, actinic damage/malignancy
- Risk for SCC transformation of DLE on lip

*(Picture: Warren et al. Lip lupus erythematosus. Medical Journal of Australia 2013; 198(3): 160-161)*



# Management

- Confirm diagnosis - biopsy of most indurated area, not ulcerated
- Exclude differentials - swabs
- Exclude systemic involvement – systemic examination, bloods, urine, CXR
- Identify possible triggers/associations – medications, UV
- Treat/optimize underlying comorbidities
- Sun avoid/protect
- Pain management
- Topical
- Oral systemics: Plaquenil, methotrexate,



# Lupus – neonatal

- Rare: 1:20,000 pregnancies
- Passive transfer of anti-SSA/Ro antibodies → create complications in the skin, heart, liver, blood, brain
- Most mothers (60%) do not have any symptoms and are often unaware they have circulating antibodies
- Onset either birth or within first 2 months of life
- Classic “raccoon face” distribution: periorbital and scalp
- Complications
  - Congenital heart block +/- cardiomyopathy; ~ 10-30% mortality ; 2/3 require pacemaker
  - Hepatobiliary disease
  - Cytopenia
  - Hydrocephalus, macrocephaly
- Skin lesions are usually self-resolving by 6-8 months and management is with sunprotection; +/- topical corticosteroids, general skin care
- Maternal care – immunology, may require systemic agent during any subsequent pregnancy

The great mimickers

Syphilis

Leprosy

# Syphilis – T.pallidum

- Transmission – inoculation is via mucosal surfaces and penetrated skin; sexual contact, blood borne, intrauterine
- Risk factors
- Stages
  - Primary : chancre; single or multiple, regional LN, painless ulcer with raised/rolled edge
  - Secondary : up to 6 months after primary; caused by the haematogenous dissemination of spirochetes; mucocutaneous lesions and systemic signs/symptoms (generalized LNs, malaise, sore throat, body aches, low grade fevers, headaches)
  - Early – non-primary/non-secondary: infection within last 12 months, but no signs or symptoms of primary or secondary syphilis
  - Unknown duration/late: infection occurred >12 months
- Otic/neuro/ocular-syphilis at any stage

# Syphilis – cutaneous features

## 8x mucosal forms

1. Condylomata lata
2. Mucosal patches
3. Snail track ulcers
4. White plaques on tongue – mimic leukoplakia
5. Depapillation of the tongue (soral aspect)
6. Split commissures
7. Enanthem
8. Bullous erosive lesions

## 13x cutaneous morphologies

1. Exanthem
  2. Follicular papules
  3. Lichenoid
  4. Psoriasiform
  5. Corymbiform
  6. Nodular
  7. Annular
  8. Frambesiform
  9. Leukoderma
  10. Lues maligna
  11. Clavi syphilitici
  12. Acral pebbles
  13. pustular
- PLUS alopecia  
PLUS nail changes

# Syphilis - Examination

- Review of systems
  - General: malaise, fever, fatigue, weakness, dizziness
  - Eyes: pain redness, double vision, photophobia, “floaters”
  - Ears: tinnitus, hearing loss
  - GIT: nausea, vomiting
  - MSK: pain, stiffness, muscle weakness
  - Neurological: headache, dizziness, seizures
  - Psychiatric: confusion
- Focused neurological examination: Cranial nerve examination and motor strength
- Nuchal rigidity testing: Brudzinski sign, Jolt accentuation maneuver
- Deep tendon reflexes: assess for hyperreflexia

# Syphilis – investigations and management

- T.pallidum does not culture
- Bloods
  - Treponemal bloods
  - HIV
  - Other STIs
  - FBE, EUC, LFTs
- Biopsy
- Ocular, neuro, ENT on suspicion

## Management:

Contact trace; notifiable disease

- Primary 3/12, Secondary 6/12, late 12/12

Wound care

Pain relief

Abstain sex for 1 weeks and until all symptoms have resolved

**Benzathine penicillin G 2.4million units IM buttocks**

Allergy: Doxycycline 100mg BD x 14 days

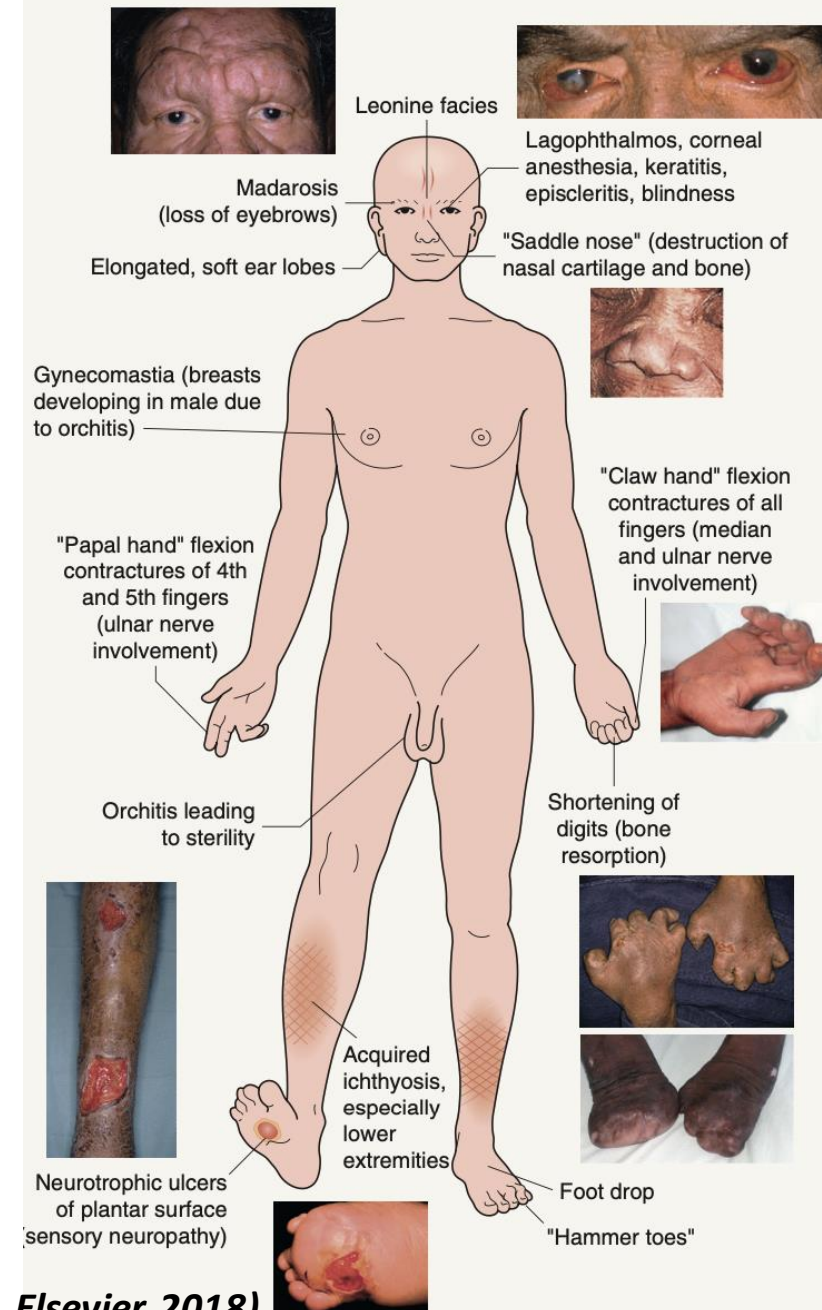


# LEPROSY

- Prevalence
  - Rare
  - No local transmission since 2009
  - 10-20 notified cases per year in Australia
  - Indigenous Australians in remote locations bearing greatest burden of disease
- Transmission of *M. Leprae*
  - Via skin and respiratory tract to affect skin and nerves
- Risk factors
  - Increased age, immune suppressed, close household contact
- Neural involvement - complications
- Reference:



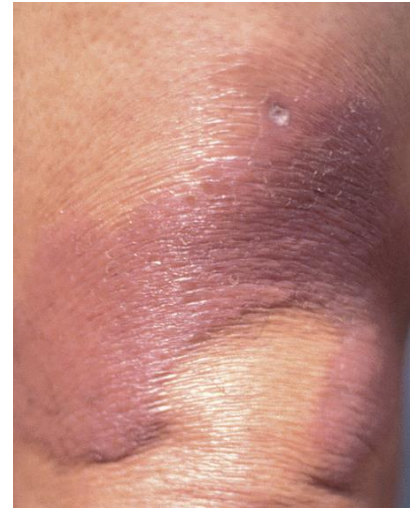
This is an ePublication only available from Centre for Disease Control, publications web page:  
<https://health.nt.gov.au/professionals/centre-for-disease-control/resources-and-publications>.



Picture: Bologna et al. Dermatology 4<sup>th</sup> edition. Elsevier 2018)

# Clinical features

- Early lesions (“indeterminate”)
  - Area of numbness on skin +/- visible skin lesion
  - Face, extensor surfaces of limbs, buttocks, trunk
  - Single to few in number
  - Small, flat, hypopigmented or coppery with an irregular border
  - Majority heal spontaneously or further develop into “established lesions”: tuberculoid or lepromatous.



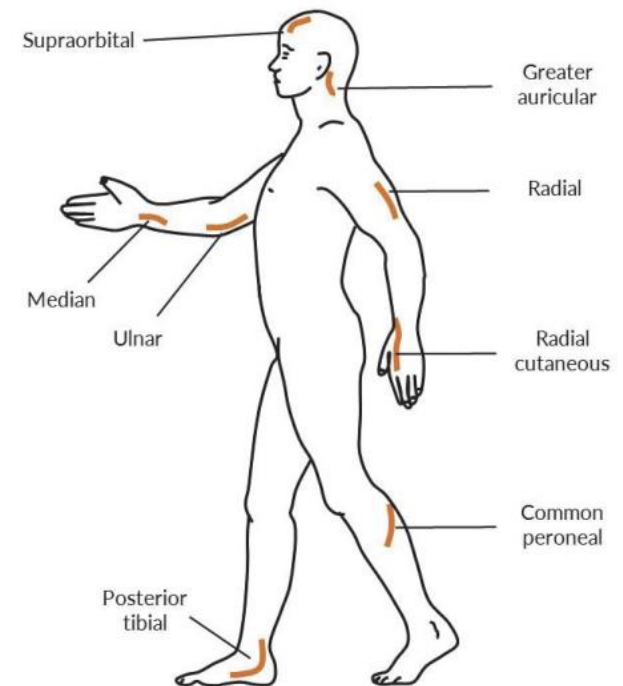
# Clinical features

- Established lesions
  - Tuberculoid
    - More common
    - Low bacillary load
    - Either neural alone (anaesthesia, nerve swelling, muscle weakness)
    - Skin lesions - +/- nerve involvement: hypopigmented, erythematous, well defined border, sometimes raised, non – sweating, +/- decreased hair and sensation
  - Lepromatous
    - More serious and disabling
    - High bacillary count
    - Skin lesions: varied appearance: macular, diffuse, papular, nodular, infiltrative
    - Often associated with nasal symptoms (congestion, papules in nose, lips, tongue), and peripheral oedema
    - Nerve involvement presents later with numbness and anaesthesia → dorsum of hands and feet → progresses to arms and legs then trunk +/- corneal involvement leading to blindness



# Examination

- History
  - Onset, character, symptoms, muscle weakness, eye pain and symptoms
- Full skin examination
- Nerve palpation
- Eye examination



*(Image from: NT guidelines: Guidelines for the control of Leprosy in the Northern Territory 2018 version 3.0.)*

# Management

- Involvement of a multidisciplinary team
  - Infectious disease,
  - Neurology,
  - Ophthalmology,
  - Allied health: OT, PT, dietician, psychology
- Multidrug treatment
  - Treatment regime based on number of lesions
  - Paucibacillary (1-5) versus Multibacillary (>6)
  - Dapsone + Rifampicin +/- Clofazimine
- Regular follow up
  - Blood work, skin and neurological and eye assessments for 24 months
- Contact tracing
  - Defined as those living in the same household for atleast 3 months
  - Can include neighbours and social contacts
  - Chemoprophylaxis with single dose of Rifampicin

Table 10. NT first line MDT regimen with adult (≥15 years old) doses

	PB*	MB—Low BI <4+	MB—High BI ≥4+
<b>Duration</b>	6 months	12 months	24 months
<b>Dapsone</b>	100mg daily self-administered	100mg daily self-administered	100mg daily self-administered
<b>Rifampicin</b>	600mg monthly <sup>†</sup> DOT <sup>‡</sup>	600mg monthly <sup>†</sup> DOT <sup>‡</sup>	600mg monthly <sup>†</sup> DOT <sup>‡</sup>
<b>Clofazimine</b>	-	50mg daily self-administered plus 300mg monthly <sup>†</sup> DOT <sup>‡</sup>	50mg daily self-administered plus 300mg monthly <sup>†</sup> DOT <sup>‡</sup>

*(Image from: NT guidelines: Guidelines for the control of Leprosy in the Northern Territory 2018 version 3.0.)*

# Drug reaction/s

- Skin is one of most common sites for drug reactions
  - Up to 5% of all people given antibiotic with develop a drug reaction;
  - Up to 2% of all drug reactions are considered 'serious' by the WHO
  - 8% of hospitalized people develop a drug reaction
  - 2% patients in outpatient department setting
- Clinical Characteristics?
  - Erythematous, Pustular, Lichenoid, Peeling/blistering, Urticarial\*, exanthematous\*
  - Distribution, pattern of presentation
  - **Associated systemic symptoms?**
  - **Mucosal involvement of eyes, mouth, genitals?**
  - **Pain versus itch?**
- Obtain details re history of onset, course and character most important
  - List of all medications – including vitamins and OTC medications, eyedrops
  - Onset dates of medications
  - Date of first appearance of rash in context of new medication/change of brand
  - History of ?rechallenge
  - Response to removal of medication?
- Obtain details re drug history/timeline most important
  - Antibiotics, antihypertensives, anticonvulsants
- Micromedex, AMH, MIMs, PBS

CHARACTERISTICS OF MAJOR DRUG-INDUCED ERUPTIONS

Clinical presentation	Percentage that are drug-induced (%)	Time interval	Mortality (%)	Selected responsible drugs
Exanthematous eruption	Child: 10–20 Adult: 50–70	4–14 days	0	Aminopenicillins Sulfonamides Cephalosporins Anticonvulsants (aromatic) Allopurinol Abacavir Nevirapine
Urticaria	<10	Minutes to hours	0	Penicillins Cephalosporins NSAIDs Monoclonal antibodies Radiocontrast media <sup>†</sup>
Anaphylaxis	30	Minutes to hours	5	
Fixed drug eruption	100	First exposure: 1–2 weeks Re-exposure: <48 hours, usually within 24 hours	0	TMP-SMX NSAIDs Tetracyclines Pseudoephedrine*
Acute generalized exanthematous pustulosis (AGEP)	70–90	< 4 days	1–2	β-Lactam antibiotics Macrolides Calcium channel blockers
Drug reaction with eosinophilia and systemic symptoms (DRESS)/drug-induced hypersensitivity syndrome (DIHS)	70–90	15–40 days	5–10	Anticonvulsants (aromatic) Lamotrigine (especially in combination with valproate) Sulfonamides Abacavir Allopurinol Dapsone Minocycline Nevirapine
Stevens–Johnson syndrome	70–90	7–21 days	5	Sulfonamides Anticonvulsants (aromatic) Lamotrigine Allopurinol NSAIDs NNRTIs, e.g. nevirapine
Toxic epidermal necrolysis			30	



<sup>†</sup>Often anaphylactoid reaction.  
\*Non-pigmenting.

**(Table and pictures: Bologna et al. Dermatology 4<sup>th</sup> edition. Elsevier 2018)**

# Management

- Stop offending agent
- Topical corticosteroids
  - High potency eg. Diprosone ointment
  - General skin care measures
- +/- Oral prednisolone
  - 0.5mg – 1mg/kg per day
- Escalate treatment with systemic symptoms and urgent admission to hospital with rapid progression, fevers, systemic symptoms, blood indices indicating organ involvement.



# Main points to take home

- Many differentials for your red, scaly rash
- Clear history of medications and medication review VIP
- ALWAYS perform systems review
- ALWAYS check in mouth, nails, hair, eyes and ask re genital involvement
- When in doubt, investigate!
  - Skin scrapings
  - Skin swabs
  - Bloods
    - Consider lupus screen
  - **BIOPSY**

# References

- Bologna et al. Dermatology 4<sup>th</sup> edition. Elsevier 2018
- NT guidelines: Guidelines for the control of Leprosy in the Northern Territory 2018 version 3.0.
  - <https://digitallibrary.health.nt.gov.au/prodjspui/bitstream/10137/526/3/Control%20of%20Leprosy%20in%20the%20Northern%20Territory%20Guidelines.pdf>
- Forrestel, AK. Sexually acquired syphilis. Journal of the American Academy of Dermatology. 2020 volume 82 (1): 1-14
- Warren et al. Lip lupus erythematosus. Medical Journal of Australia 2013; 198(3): 160-161

# Questions & Answers



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