Paediatric Dermatology
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Common neonatal eruptions
Erythema toxicum
Associated with neonatal erythema most prominent on day 2
Occasional pustules
well baby
Gram stain full of eosinophils
‘6 week’ eruption
Otherwise known as ‘milk spots’, infantile acne
Is really a pityrosporum folliculitis
Fades as sebaceous glands settle to quiescent childhood levels
Pityrosporum folliculitis- treatment
Application of either 2% Ketoconazole cream or Ketoconazole shampoo diluted 1 in 10, applied via cotton bud.
Twice per day applications usually clears the eruption in a few days.
Cradle cap
Probably analogous to dandruff in the adolescent/adult
‘greasy’ scale
generally not pruritic
Cradle cap- treatment
Not necessary
Remove scale without irritating scalp
soap substitutes, olive oil, bath oil
may use comb gently if stuck in hair
Ketoconazole shampoo if recurrent
Nizoral, Sebizole
Salicylic acid creams only rarely required

Scalp eczema
Do not diagnose cradle cap after 4 months
Scale is harsher
Pruritus common
Usually eczema elsewhere
Scalp eczema
Treat as eczema elsewhere
Use topical steroid ointments rather than lotions
Shampoos will irritate- use soap substitutes
no role for anti-yeast therapy
Salicylic acid will worsen
Scalp scaling
Neonates have seborrhoeic skin
hence may have cradle cap or seborrhoeic dermatitis
Children have inactive sebaceous glands
hence may develop eczema or tinea
Psoriasis may occur at any age
Nappy rash
Much less common now
Better disposable nappies
Secondary candidal infection (thrush) much rarer
Nappy rash
Irritant contact dermatitis
Skin has a threshold of irritation
Combination of heat, occlusion, urine and faeces
Once broken down, the natural barrier is disturbed as threshold of irritation is less
Secondary candida

Aims of management

Irritation

Avoid contributing to irritation

Protection from urine and faeces

Settle inflammation

Treat any secondary infection

Avoid irritants

Use disposal nappies

Frequent changes

Nappy free time when possible

Wash with diluted bath oil using cotton balls or Chux towels

Dab gently rather than wipe vigorously

Bath oil and no other irritants in bath

No antiseptics/cleansers etc

Protection from urine/faeces

Barrier cream aims

Protective barrier against irritants

Should still be there at next change

Simple, bland formulation

No preservatives, fragrances, antiseptics, essential oils

Protection from urine/faeces

Do not try to remove each change

This only adds to irritation

Individual preference in barrier cream

‘Covitol’ (Cod liver oil in Zinc)

‘Bepanthen’ (Dexpanthenol and lanolin)
Plain Zinc cream
10% Olive oil in Zinc paste
Settle inflammation
1% Hydrocortisone ointment
‘Sigmacort’
‘Egocort’
‘Dermaid’
Should only require daily application
Treat secondary infection
Add in Canesten/Daktarin with hydrocortisone daily if suspicious
Mostly unnecessary
Antifungal creams can be a little irritant
Have area swabbed if concerned
Treatment summary
Disposable nappies
Low irritant wipes
1% Hydrocortisone (+/- Canesten) after bath
Barrier cream every other change
Zinc Deficiency
Zinc deficiency
Seen in essentially two settings:
Inherited (Acrodermatitis Enteropathica)
Acquired
Acrodermatitis Enteropathica
Autosomal recessive
Defect in absorption of zinc from GIT
2-3% vs 27-64% normal absorption
Breast milk is protective
usually presents following weaning
Acquired Zinc deficiency
Usually multifactorial aetiology
prematurity (decreased stores, malabsorption, increased demand)
low breast milk zinc
malabsorption
Historically, TPN major cause but now protective
Clinical features
Sharply demarcated, eroded eruption
symmetrical
may be vesicobullous
distribution is periorificial plus extremeties
Diffuse alopecia
Non-cutaneous features
Diarrhoea
Failure to thrive
Depressed mood, irritability
Immunosuppression
Cognitive/motor delay
Investigations
Blood zinc level
not an accurate measurement of stores (neither is hair or urine)
many false positives and negatives
Maternal breast milk Zinc
Management
Replacement
Elemental Zinc at 1-2mg/kg/day

50mg Zn = 220mg ZnSO₄

Is a therapeutic investigation

Continue until weaned in acquired, lifelong in Acrodermatitis enteropathica

Zinc deficiency - key points

“Think Zinc” if unusual eruption with diarrhoea

Age of onset determines whether congenital or acquired

Blood zinc level may be inaccurate

clinical challenge very useful

Management of acute exanthems

Management of acute exanthems

1. When it is diagnostic

2. When to be concerned

3. Assessment of ‘typical’ scenario

When is it diagnostic?

Parvovirus B19 - Clinical SYNDROMES

'Fifth' disease (Erythema infectiosum)

Arthritis (check mothers)

Purpura, including 'Purpuric gloves and socks syndrome'

If immunodeficient:

chronic anaemia

If sickle cell disease:

aplastic crisis

Parvovirus B19 - Clinical syndromes

In pregnancy

1st trimester fetal loss

2nd trimester hydrops

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3rd trimester stillbirth (uncommon)

Myocarditis reported

Parvovirus B19- Clinical features

'Slapped cheek' appearance with slight fever only

- non-specific

Reticulate erythema of forearms

- presumed immune-complex phenomenon
  - may extend to palms
  - may be recurrent for >2/52

Parvovirus- key points

One of two viral eruptions diagnosable on cutaneous features alone

Non-infectious when eruption present

Important in pregnancy, immunosuppressed and sickle-cell anaemia

Hand, foot and mouth (traditional)

HFMD (recent)

Eczema herpeticum

When is it serious?

Kawasaki's syndrome

- prolonged fever (>5/7)
- conjunctival injection (non-exudative)

May be transient

- mucous membrane changes
- Brick-red indurated extremities
- lymphadenopathy
- rash on body is non-specific (except typically perianal)

Kawasaki- complication

Coronary artery aneurism
Up to 15% untreated
Delay of therapy correlates with likelihood
Other vascular event rare
Kawasaki- Therapy
Aspirin and IVIG first line
Infliximab and others if not responding
Kawasaki syndrome- Key points
High fever persisting while eruption presents
Presence or history of ocular injection
Spares rim around pupil
Not a conjunctivitis
Involvement palms and soles
Groin/perianal involvement (occurs early)
Assessment of ‘typical’ scenario
Viral eruptions- common
   Enterovirus (summer)
   Respiratory (winter)
   EBV
   HH6 (Roseola infantum)
   Parvovirus B19
Viral eruptions- uncommon
   Measles
   Rubella
   Mumps
   CMV
   Hepatitis
   HIV seroconversion (50%)
Ross River and Barmah forest

Viral-induced urticaria

Roseola infantum

HH6

75% infants seroconverted by age 1

High fever for 3-5/7 but child remains comparatively well

Roseola infantum

Rash occurs as temperature is falling and lasts 1-2/7

Uniformly benign

Except febrile convulsions

Measles

Prodromal rash and Koplick spots

viraemia

Conspicuous 'day 4' rash

Cell-mediated response

Cough, Conjunctivitis and Coryza

with fever (and toxic-looking child), occur before rash

Measles- Complications

otitis media

bronchopneumonia

enteritis

encephalitis

SSPE

Rubella

Prodrome 1-5/7

high fever

sore throat without coryza
mild conjunctival involvement only
lymphadenopathy (suboccipital, postauricular, cervical)

Rubella- Complications

arthritis (mainly adults)
purpura
encephalitis reported
teratogenicity

  90% before week 11
  25% between week 12-16
  brain, deafness, heart, eye

CLUES TO DIAGNOSIS: Prodrome illness

Measles- fever, conjunctivitis, cough, coryza

Roseola infantum- high fever for 1-4/7 disappearing with rash

EBV- pharyngitis, tonsillitis, lymphadenopathy

  DDx is HIV seroconversion

CLUES TO DIAGNOSIS

Meningitis
  Enterovirus

Arthritis
  Rubella
  Parvovirus (usually adult females)
  Ross River
  Barmah forest

Notifiable diseases

HIV
Ross River
Barmah forest
Measles
Rubella
Urticaria

Urticaria- Aetiology

Represents reaction pattern to variety of stimuli

Due to mast cell degranulation

Can be due to hypersensitivity:

Type 1 (IgE allergic)

Type 3 (immune complex)

Type 4 (cell mediated)

Type 5 (auto-immune)

Can be due to toxic reaction or intolerance

Urticaria- Aetiology

Best thought as having:

1. Triggers

Reasons that cause the mast cells to suddenly become ‘sensitive’

1. Exacerbating factors

Reasons that cause ‘spillage’ of products from sensitized mast cells

Urticaria- Aetiology

Commonest triggers in children:

Viruses

Viruses

Viruses

Food allergy

Medication allergy

Other infections

Auto immune/Rheumatological/Metabolic

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Insect bites (multiple)

Urticaria

Once triggered, the mast cells may remain sensitive for days, weeks, months or years

Urticaria- exacerbating factors

Heat

Pressure (Dermographism)

Salicylates (Acidic foods)

Viruses and other immune stimuli

Sweating (Cholinergic urticaria)

Cold

Sun

Urticaria- clinical features

Transient nature

Can be diagnosed over the phone

Most plaques do not last more than 24 hours

Red plaques

White halo

May have ‘dusky’ centre

May be strikingly annular

Dermographism often present

Differential diagnosis

Urticarial vasculitis (incl HSP)

Longer lasting lesions

Evidence of purpura or may leave bruising

More likely to have arthralgia

Erythema multiforme

Annular erythemas
Other viral and drug eruptions
Cutaneous lupus
Vasculitis
Erythema Multiforme
Neonatal lupus
Urticaria- management
Screen for cause on H&E
Fever
General well-being
Food ingestion
Urticaria usually commences head and neck
Medication history
Localizing signs for infection/rheumatologic disease
Urticaria- management
Assess for associated angioedema or anaphylaxis
Wheeze
Vomitting
Altered conscious state
Tongue swelling
Urticaria- management
Investigations:
**Most children are well or with virus and therefore need no investigation**
According to underlying symptoms and signs, not due to degree of urticaria
Allergy testing (SPT or RAST)
If <18/12
Suggestive for food on history
?Parental pressure
Urticaria- management

Education:

Explain aetiology

Explain prognosis:

Days, weeks, months, years

Discuss exacerbating factors

Role of therapy is to alleviate symptoms whilst waiting for natural resolution

Urticaria- management

Therapy:

Antihistamines

Best taken prophylactically

Aware of RCH guidelines

Only help with itch and elevation of wheal

Prednisolone if acute and/or severe

Start at 1mg/kg and wean down over 1/52, but may need longer

Explain potential for recurrence after finishing course

Urticaria- management

Second-line therapy for chronic urticaria

Montelukast

H2 blockers

Sodium Chromoglycate

UVB

Other immunosuppressive therapy

RASHES WITH PRESUMED VIRAL AETIOLOGY

Pityriasis rosea

Unilateral laterothoracic exanthem

Papular acrolocated syndrome (Gianotti-Crosti syndrome)
Pityriasis Rosea

Absent or minimal prodrome

'Herald patch' in 80%

usually near proximal joint

larger than other patches

Pityriasis Rosea

Eruption occurs hours to days later

symmetrical and proximal

long axis of patch in Christmas tree distribution

free edge of scale internally

Usually lasts 3-6/52

topical steroids and/or UVB for symptoms

Pityriasis Rosea

Differential diagnosis:

drug eruption

secondary syphilis

guttate psoriasis

discoid eczema

pityriasis lichenoides

Pityriasis Rosea

Treatment

None required

Topical steroids not very helpful

Sunlight/UVB very useful

?Erythromycin

Unilateral laterothoracic exanthem

Recently described entity
Some have preceding illness

0-5 years

Commoner in spring and in girls

Histology- eccrine gland infiltration with lymphocytes

Unilateral laterothoracic exanthem

Discrete erythematous papules eventually coalescing

sometimes with a secondary eczematous component

Commence unilaterally in axilla before spreading ipsilaterally over trunk then bilaterally

Child remains well

Mean duration 5 weeks

Papular Acrolocated Syndrome

‘Giannotti-Crosti syndrome’

Described initially as post-Hepatitis B syndrome but now shown to be a non-specific response to viral infection

Hep B cases usually anicteric

vast majority in Australia due to enteroviruses

Age 6/12 to 12 years usually

Papular Acrolocated Syndrome

Urticaria-like papules develop on limbs, buttocks and face

little to no pruritus

papules remain small and fixed

monomorphic for given patient

Fades after 2-8/52 with mild desquamation

No treatment required- ?role of serology for Hep B

Papular Acrolocated Syndrome- Key points

Well child

May give history of viral disease at time of onset

Spares trunk
Lasts 6 weeks

Vascular Birthmarks

Capillary Malformations

(Port Wine Stains)

Haemangiomas of Infancy

(Strawberry naevus)

Others

Capillary Malformations

Present at birth

Flat

Undergo no growth phase

Grow in proportion to child

Permanent

Capillary Malformations- associations

If large over head

Vascular malformation in brain and eyes

Glaucoma

‘Sturge-Weber’ syndrome

If large over limb

Limb overgrowth

Varicose veins

‘Klippel-Trenaunay’ syndrome

Capillary Malformations

Longer term cosmetic issues

Vascular laser treatment of choice

Early treatment is preferable
Smaller

Can be easily held

Probably better response each therapy

**Therefore if anticipated cosmetic sequelae, refer ASAP**

**Haemangiomas**

Up to 8% of all babies

More common in females and premature

Usually **not** present at birth.

but may be.

Start as flat stains or bruise-like marks

**Always** have a proliferative phase after birth.

**Haemangiomas**

Proliferate for 1-9 months

Usually 3-4 months

Stabilize then involute

Involution by age 1-10 years old

Not always completely

**Segmental haemangiomas**

**PHACES syndrome**

**Posterior fossa malformations**

**Haemangioma (large, segmental)**

**Arterial anomalies**

**Coarctation of the aorta and other Cardiac defects**

**Eye abnormalities**

**Sternal defects**

Investigations for segmental haemangiomas

**MRI brain**
?and other vessels

Paediatric Cardiology review with echocardiogram

Ophthalmology review

Periocular haemangiomas

Amblyopia

Visual deprivation

Unequal refraction (astigmatism)

Other

Strabismus, myopia, tear duct obstruction, proptosis, ptosis

Airway involvement

‘Beard haemangioma’

2/3 will have symptomatic associated airways disease

Usually present 6-12/52

Croup-like cough, stridor, feeding difficulties

Nasal tip and lips

High rate of disfigurement if too large

Difficult to repair later

Low threshold for high dose Prednisolone

Consider early excision

Lumbosacral haemangiomas

Associated with occult spinal dysraphism and/or genitourinary abnormalities

MRI best visualizing study but U/S can be useful screen if performed early enough

‘Haemangiomatosis’

Usually at least 10 cutaneous lesions

Most often present at birth

Regress quickly

Internal involvement can be serious

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Any organ but liver commonest
AV shunting
Cardiac failure
Perineal haemangiomas
Prone to ulceration
As are axillary, neck, perioral
Complications
Haemorrhage
Pain
Infection
Perineal haemangiomas- ulcer management
Occlusive dressings (Tegaderm)
Bactroban/Fucidin
Metronidazole
Xylocaine jelly
Alternative is Intrasite gel
?Laser
Consider surgery/ Propranolol
Haemangiomas and Propranolol
First line therapy
Serendipidous finding 5 years ago
Mechanism unclear
Threshold for treating systemically is lower
Topical Timolol 0.5% eye gel now used as well
Cosmetically burdensome haemangiomas
Who should get Propranolol?
Who should get Prednisolone?
Need 2-4mg/kg weaning over at least 6/52

Consider early surgery

Need considerable hand-holding

Haemangioma precursors

Haemangioma and laser

Can have some effect

Only if used in the very early stage

Useful in late stage for ‘leftover’ telangiectasia

When do I refer urgently?

Any large red birthmark

Particularly if ‘segmental’

Any changing/growing birthmark in concerning area

Eyes

Nose

Mouth

Perineal

Thankyou!