Emergency Medicine Course
Chelmsford
Dermatology Workshop Handouts

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Dermatology Emergencies

Things to look out for

PAINFUL SKIN

- Widespread rash
- Affecting mucous membranes
- Blisters
- Redness of the skin
- Signs of vasculitis
- On immunosuppressant drugs
- Systemic signs – shivering, temperature etc
Dermatological Emergencies

• Erythema multiforme, Stevens-Johnson syndrome, Toxic Epidermal Necrolysis
• Blistering /Bullous Disorders
• Staphylococcal scalded skin syndrome
• Generalised pustular psoriasis
• Infections – Cellulitis, Herpes Simplex, Meningococcal and Necrotising Fasciitis
• Erythroderma
• Vasculitis
• Drug Rashes
• Anaphylaxis
<table>
<thead>
<tr>
<th>Erythema Multiforme</th>
<th>SJS/Toxic Epidermal Necrolysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Acral mainly</td>
<td>• Central/ widespread</td>
</tr>
<tr>
<td>• Target lesions</td>
<td>• Purpuric or blisters</td>
</tr>
<tr>
<td>• Mucous membrane involvement milder</td>
<td>• Mucous membrane involvement severe</td>
</tr>
<tr>
<td>• Rare to involve eye/genital areas</td>
<td>• Conjunctival/genital involvement</td>
</tr>
<tr>
<td>• Commonest reason is herpes simplex</td>
<td>• Commonest reasons mycoplasma or drugs</td>
</tr>
<tr>
<td>• Few systemic symptoms</td>
<td>• Marked systemic symptoms</td>
</tr>
<tr>
<td>• Crops of lesions</td>
<td>• Sudden onset of rash</td>
</tr>
<tr>
<td>• Target lesions</td>
<td>• Tender dusky erythema</td>
</tr>
<tr>
<td>• Fade over 1-2 weeks with no scarring</td>
<td>• Prodrome up to 2 weeks</td>
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</tbody>
</table>
Toxic Epidermal Necrolysis

• Prodromal illness - flu-like illness with Fever – persistent and high, cough, sore throat, difficulty swallowing, runny nose, sore red eyes, conjunctivitis, painful skin, general aches and pains

• Then abrupt onset of a tender/painful red skin rash starting on the trunk and extending rapidly over hours to days onto the face and limbs. The maximum extent is usually reached by 4 days
Toxic Epidermal Necrolysis

• The skin lesions may be: Macules – flat, red and diffuse or purple spots, Targets – as in erythema multiforme or Blisters – flaccid

• The blisters then merge to form sheets of skin detachment, exposing red, oozing dermis

• The Nikolsky sign is positive in areas of skin redness – pushing the skin causes the blister to extend

• Histology shows full thickness skin necrosis (compared to Staph Scalded Skin Syndrome which is superficial skin necrosis)
## Drugs that can cause TEN

### Drugs that most commonly cause SJS/TEN

<table>
<thead>
<tr>
<th>Category</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Antibiotics</strong></td>
<td>Sulfonamides, e.g., cotrimoxizole; beta-lactams i.e., penicillins, cephalosporins</td>
</tr>
<tr>
<td><strong>Antifungals</strong></td>
<td>Imidazole antifungals, Nevirapine (non-nucleoside reverse-transcriptase inhibitor)</td>
</tr>
<tr>
<td><strong>Antivirals</strong></td>
<td>Allopurinol, Nonsteroidal anti-inflammatory drugs (NSAID) (oxicam type mainly), Anti-convulsants</td>
</tr>
</tbody>
</table>

- Nonsteroidal anti-inflammatory drugs (NSAID) (oxicam type mainly):
  - Naproxen
  - Ibuprofen

- Anti-convulsants:
  - Carbamazepine
  - Phenytoin
  - Phenobarbital
  - Valproic acid
  - Lamotrigine
Toxic Epidermal Necrolysis

- Nursed as for burns in specialised units- high mortality and morbidity
- SCORTEN - Age over 40, Presence of a malignancy, Heart rate >120, Initial percentage of epidermal detachment >10%, Serum urea level >10 mmol/L, Serum glucose level >14 mmol/L, Serum bicarbonate level <20 mmol/L
- **SCORTEN 5 or more >90% Mortality**
Causes of Blisters

- Drugs - antiepileptic drugs TEN
- Contact dermatitis/acute eczema - eg; hair dye allergies
- Photodermatitis/plants - linear streaky distribution
- Infective causes of blisters - staph toxin in origin - bullous impetigo, Herpes Simplex and Herpes Zoster
- Linear – dermatome- shingles
- Insect bites
- Inflammatory - bullous pemphigoid
- Back of the hands – porphyria
- Dermatitis Herpetiformis – small vesicles elbows and knees
- Fixed drug eruption – comes in same place
- Friction especially in lower legs
- Cardiac failure – oedema can blister
<table>
<thead>
<tr>
<th><strong>Bullous Pemphigoid</strong></th>
<th><strong>Pemphigus</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Frequency</strong></td>
<td>Uncommon</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td>Usually over 70 years old</td>
</tr>
<tr>
<td><strong>Blisters</strong></td>
<td>Tense, clear fluid, like pompholyx on hands/feet. Intact blisters</td>
</tr>
<tr>
<td><strong>Adjacent skin</strong></td>
<td>Eczematous or urticated</td>
</tr>
<tr>
<td><strong>Mucous membrane</strong></td>
<td>About 25% minor involvement</td>
</tr>
<tr>
<td><strong>Direct Immuno studies</strong></td>
<td>Positive at basement membrane zone</td>
</tr>
<tr>
<td><strong>Response to treatment</strong></td>
<td>Usually good</td>
</tr>
</tbody>
</table>
Bullous Pemphigoid

- Crops of tense, fluid-filled blisters develop

- Bullous pemphigoid is usually very itchy

- Diagnosed by skin biopsy with direct immuno-fluorescence

- Can do indirect immuno-fluorescence on serum

- Treated with oral steroids

- Is an auto immune disease
Pemphigoid Gestationis

- IF shows linear C3 on BMZ
- Indirect BP180 positive
- 11% have Graves disease
- FH of autoimmune problems
- Can skip pregnancies
- No association with HLA compatibility between mum and child
- No association with age of partner
Pemphigus

- Rare autoimmune disease
- Mostly adults 40-60, but can affect any age group
- 50-70% of patients get oral lesions
- Blistering superficial and often appears as erosions
- Since the introduction of oral steroids mortality rate dropped from 99% to 5-15%
- Diagnosed on a skin biopsy
- Treated with oral steroids, Azathioprine etc
Staph Scalded Skin Syndrome

• Usually affects small children esp neonates
• Red blistered skin like burns or scalds
• Tissue paper wrinkling, then large fluid filled blisters in armpits, groins and around ears and nose
• Then top layer peels off leaving raw skin
• Causes by exotoxins from certain strains of staph
• Mortality low but needs intensive care
Generalised Pustular Psoriasis

• Rare in Children
• Has been caused by treating an undiagnosed rash with oral steroids
• Caused usually by the sudden withdrawal of either oral steroids or strong topical steroids
• This destabilises the psoriasis
• Sheets of small pustules appear – can be life threatening
• Is a dermatological emergency
Infections

- Measles
- Erysipelas
- Cellulitis
- Herpes Simplex
- Meningococcal Septicaemia
- Necrotising Fasciitis
Measles

- Incubation 14 days
- Preceded by fever, cough and very red eyes. Photophobia in older children. Oral Koplik’s spots. Pink then red macules. **Looks sick.**
- First face, then chest and abdomen, then limbs
- Lasts 4-7 days
- Spread from sneezing and coughing – very contagious
- Infectious from prodromal symptoms to 4 days after the rash onset
- The virus can live on infected surfaces for up to 2 hours
- Can cause pneumonia, encephalitis, cardiac problems and death.
Erysipelas

- Superficial form of cellulitis
- A potentially serious bacterial infection affecting the skin.
- Most often affects infants and the elderly
- Usually Caused by Strep
- Affects lower limb or face – on face is has a characteristic butterfly distribution over cheeks and bridge of the nose.
- Abrupt in onset and often accompanied by general illness in the form of fevers, chills and shivering.
- Affected skin has a well-defined, raised border.
- May be finely dimpled -like an orange skin
- It may be blistered or purpuric
Cellulitis

- Usually caused by Strep
- Similar but deeper and more diffuse than erysipelas
- Can be very acute with high fever, vomiting and can be delirious - systemic signs of fever/malaise/chills
- If leg involved it can lead to permanent oedema of leg
- Fungal infections of feet can be the portal of entry – look for portal of entry
- Need high doses of antibiotics to control it
- Tends to be lower extremities
- Local tenderness, pain, erythema, swelling, tight, swollen skin. Can be blisters – sign of skin breakdown
- Borders of erythema not well demarcated
- Local lymphadenopathy
Predisposing Factors to Cellulitis

• Break in skin integrity - such as trivial injury to the skin or surgical wounds, radiotherapy
• Venous insufficiency & ulcers
• Diabetes mellitus
• IV drug use
• Malnutrition
• Venous disease (e.g. gravitational eczema, leg ulceration) and/or lymphoedema
• Diabetes
• Alcoholism
• Obesity
• Pregnancy
## Diagnosis of Cellulitis

| Sudden onset                                      | √ |
| One sided hot, deep, well demarcated swelling     | √ |
| Fever                                             | √ |
| CRP raised                                        | √ |
| WCC raised                                        | √ |
Differentials of a Red Swollen Leg

- Eczema – Varicose, Discoid, Craquelé
- Lipodermatosclerosis
- Contact dermatitis
- Tibial compartment syndrome
- Gout
- Acute Oedema - due to heart failure, protein↓
- Superficial Thrombophlebitis
- Deep vein thrombosis
- Early Herpes Zoster
Eczema Herpeticum

• Eczema herpeticum is any skin problem complicated by herpes simplex virus – not just eczema.
• Facial involvement is the most common presentation, especially around the eyes – but can be anywhere.
• The classic lesions are small punched out vesicles
• Rarely, it can disseminate to other sites leading to keratoconjunctivitis, encephalitis, hepatitis or even death.
• Acute management includes treatment of the herpetic infection, the underlying skin problem and any bacterial infection.
• In severe cases, hospital admission for IV antivirals and antibiotics may be needed
• Milder cases oral therapy in the community may be appropriate.
Meningococcal Disease

- Acute fever and chills
- Headache, Neck stiffness, Low back and thigh pain
- Nausea and vomiting
- Confusion, unconsciousness, epileptic fits
- Unstable vital signs, e.g. very low blood pressure, reduced blood flow, low urine output. Collapse from septic shock
- Petechiae that do not disappear when pressure is applied to the skin occur in 50-75% of cases
- Rash may progress to larger red patches or purple lesions
- Most often found on the trunk and extremities but may progress to involve any part of the body
- In severe cases lesions may burst and lead to necrosis
Necrotising fasciitis

• Early signs
  – Pain is more than you would expect for appearance of lesion – agonising pain
  – CRP is way up 200 - 400
  – Often history of taking NSAI drugs like Ibuprofen
  – Personal/family history of strep infection – throat, impetigo, erysipelas or cellulitis

• Later
  – Within hours affected area can become cyanotic, blistered and necrotic, with deep gangrene.
  – Patients then present with systemic involvement - high fever, tachycardia, hypotension and septic shock.
  – Management is in secondary care and involves IV antibiotics, fluid resuscitation and aggressive surgical debridement if the infection is progressing.
  – Group A Strep NF has higher death rate than meningococcal disease – up to 23%
Causes of Erythroderma

- **Inflammatory diseases** – eczema, psoriasis
- Infection – candidiasis, staph, strep, syphilis
- Inherited – Ichthyoses
- Autoimmune – blistering diseases
- Cutaneous T cell Lymphoma
- Immunologic – immune deficiency states such as HIV
- Nutritional and metabolic – zinc deficiency
- Drug related
- Internal malignancies eg carcinoma of rectum, lung, fallopian tubes, colon
- Haematological malignancies eg lymphoma, leukaemia
- Graft vs Host disease
Vasculitis

• Direct injury to the vessel wall by bacteria or viruses
• Indirect injury by activation of antibodies, which then generate inflammation within the vessel wall
• Indirect injury through activation of complement which damages the vessel wall
• In most cases an underlying cause is not found and the disease is self-limiting.
• In a minority of patients, cutaneous vasculitis can be part of a more severe vasculitis affecting other organs in the body - systemic vasculitis.
• Test for liver and kidney function and a urine test looking for protein or blood
Causes of Vasculitis

- Infection - Particularly hepatitis B and C and haemorrhagic fever.
- Drugs – Antibiotics, Thiazides, Thiouracil, Warfarin, Coumarin and NSAIs
- Food and food additives - uncommon
- Reduced blood flow - PVD, cold weather, beta blockers, varicose veins and stasis
- Malignancy
- Autoimmune disorders - SLE, Dermatomyositis, Polyarthritis Nodosa and Rheumatoid Arthritis
Drug Rashes

- Unless the patient has been previously sensitised to a drug the interval between initiation of the therapy and the onset of reaction is rarely less than one week or more than one month
Drugs that commonly cause Serious Reactions

- Allopurinol
- Anticonvulsants
- NSAIDs
- Sulpha Drugs
- Bumetanide
- Captopril
- Furosemide
- Pencillamine
- Piroxicam
- Thiazide diuretics
Rates of Reactions to Commonly Prescribed Drugs

- Amoxicillin 5%
- Trimethoprim/sulfamethoxazole 4.7%
- Ampicillin 4.2%
- Penicillin 1.6%
- Cephalosporins 1.3%
- Gentamicin 1%
- Heparin 0.7%
Drugs Unlikely to Cause Skin Reactions

- Digoxin
- Aspirin
- Paracetamol
- Prednisone
- Codeine
- Tetracycline
- Prochlorperazine
- SSRIs
History

• All drugs including OTC ones
• Interval between starting and onset of eruption
• Route, dose, duration and frequency
• Parenterally given drugs more likely than oral to cause problems
• Multiple courses of therapy and prolonged administration can cause sensitisation
Penicillin Rash

• 90% of patients who report penicillin allergy are not allergic

• The first time it comes on it is usually at END of the course or the week afterwards and gets worse

• A strep infection or a viral illness is often mistaken for a penicillin rash – will fade quickly

• It is not a reason to withhold penicillin if someone in the family is allergic to penicillin
Drug hypersensitivity syndrome

- High fever
- Widespread skin rash made up of redness, papules and sometimes blisters
- The rash can last many weeks and may progresses to erythroderma or exfoliative dermatitis
- Anti-epileptics – Lamotrigine, Carbamazepine, Phenobarbitone, Phenytoin and Allopurinol most likely culprits

Triad of fever, extensive skin rash and organ involvement, supported by a finding of eosinophilia and abnormal liver function tests.
Fixed Drug Reactions

- Paracetamol /phenacetin and other pain killers
- Tetracycline antibiotics
- Sulphonamide antibiotics including cotrimoxasole (Salazopyrin)
- Acetylsalicylic acid/aspirin
- Non steroidal anti inflammatories (NSAIDS)
- Sedatives including barbiturates, benzodiazepines and chlordiazepoxide
- Hyoscine
- Dapsone
- Phenolphthalein
- Quinine
Anaphylaxis

- Anaphylaxis is a severe response to triggers, such as arthropod bites, drugs and foods. Cutaneous manifestations include pruritus, erythema, urticaria and angioedema.

- Low-grade anaphylaxis can be treated with oral antihistamines, but there is a danger of anaphylactic shock, in which bronchospasm and hypotension can lead to collapse and death.

- Warning signs are wheeze, tachycardia and vomiting, indicating a need for subcutaneous adrenaline and transfer to A&E. Patients with anaphylaxis may need to carry an adrenaline pen.
<table>
<thead>
<tr>
<th>Angioedema type</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acute allergic angioedema</strong>&lt;br&gt;(almost always occurs with urticaria within 1-2 hours of exposure to the allergen)</td>
<td>• Food Allergy - esp nuts, shellfish, milk, eggs&lt;br&gt;• Drugs, e.g. penicillin, NSAIDs, Sulfa drugs, vaccines&lt;br&gt;• Radiocontrast media, Insect venoms, Latex</td>
</tr>
<tr>
<td><strong>Non-allergic drug reaction</strong>&lt;br&gt;(onset may be days to months after first taking the medication)</td>
<td>• Angiotensin-converting enzyme (ACE) inhibitors</td>
</tr>
<tr>
<td><strong>Idiopathic angioedema</strong>&lt;br&gt;(frequently chronic and relapsing and usually occurs with urticaria)</td>
<td>• In most cases the cause of angioedema is unknown&lt;br&gt;• Recent research indicates that 30-50% of this type of angioedema may be associated with some types of autoimmune disorders including systemic lupus</td>
</tr>
<tr>
<td><strong>Hereditary angioedema</strong>&lt;br&gt;(very rare autosomal dominant inherited disease)</td>
<td>• Inherited abnormal gene that causes a deficiency of a normal blood protein&lt;br&gt;• Decreased C1 inhibitor activity</td>
</tr>
<tr>
<td><strong>Acquired C1 inhibitor deficiency</strong></td>
<td>• Acquired during life rather than inherited&lt;br&gt;• May be due to B-cell lymphoma or antibodies against C1 inhibitor</td>
</tr>
</tbody>
</table>
Treatment of Acute Dermatology Problems

• Take Swabs
• Treat infection – Staph and Strep
• Avoid soap and shower gel
• Soap substitute
• Emollients – creams and ointments- lots of it
• Strong Topical steroids
• Antihistamines - sedating at night and non sedating in the day
• Lots of fluids
• Close review – look for blisters and signs of deterioration
• Avoid oral steroids as they may make things worse
Treatment of Severe Skin Disease

• May need admission for Intravenous fluids and antibiotics and specialised nursing

• Acute Skin Failure can lead to
  – Heart failure
  – Kidney failure
  – Overwhelming infection and death

• This is due to fluid loss and protein through the damaged skin, increased cutaneous blood flow doubling cardiac output and bacteria and viruses entering the body through the damaged skin
Treatment of Severe Skin Disease

- Antibiotics
- Fluids
- Generous amounts of thick moisturisers to prevent fluid loss through the skin – such as 50/50 White Soft Paraffin and Liquid Paraffin
- 500g a day for a generalised skin problem
- Warming it in a basin of warm water can ease application
- Avoid tapes onto skin
- Dressings held in place with bandages
Treatment of Severe Skin Disease

- Daily dressing changes
- Bed Linen and Nightwear soft materials – nothing rough or starched
- Mouth and Eye Care
- If topical steroids are required these go on after the skin is well moisturised with emollients
- The amount required varies with amount of skin that is affected
- Calamine lotion has no place in the treatment of severe skin problems