Dermatology for Dentists

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I) Type I Hypersensitivity

A. Introduction

- Hypersensitivity is an attempt at protection gone wrong
- The immune system overreacts to a harmless antigen, resulting in harm to the self
- There are 4 types of hypersensitivity based on the Coombs and Gell classification:
    - I: Immediate
    - II: Cytotoxic, antibody-dependent
    - III: Immune complex disease
    - IV: Delayed-type (cell-mediated immune memory response, antibody-independent)

B. Urticaria

- Wheals
    - Transient, oedematous papules and plaques
    - Pruritic, sometimes burning
    - Superficial and well-defined
    - Oedema of papillary body of the dermis
    - Fleeting (1-24 hours)

C. Angioedema

- Sudden, pronounced erythematous/skin-coloured swelling
- Larger oedematous area
- Deep and ill-defined
- Frequently below mucous membranes
- Sometimes pain rather than itch
- Lower dermis, subcutis
- Resolution slower than wheals (up to 72 hours)

D. Anaphylaxis

1. Definition

- Serious, life-threatening generalised or systemic hypersensitivity reaction
- Rapid onset
- May cause death

2. IgE dependent immunologic mechanisms

- Foods: peanuts, tree nuts, shellfish, fish, milk, egg, soybean, fruits, sesame
- Venoms: stinging insects
- Medications: beta-lactam antibiotics, non-steroidal anti-inflammatory drugs (NSAIDs), biologic agents
- Latex
- Occupational allergens
- Seminal fluid
- Aeroallergens
- Radiocontrast media

3. Diagnostic criteria for the diagnosis of anaphylaxis (World Allergy Organization)

Anaphylaxis is highly likely when any one of the following 2 criteria are fulfilled:

(1) Acute onset of an illness (minutes to several hours) with simultaneous involvement of the skin, mucosal tissue, or both (e.g. generalised hives, pruritus or flushing, swollen lips-tongue-uvula)

AND AT LEAST ONE OF THE FOLLOWING:

(a) Respiratory compromise (e.g. dyspnoea, wheeze-bronchospasm, stridor, reduced peak expiratory flow, hypoxaemia)

(b) Reduced blood pressure or associated symptoms of end-organ dysfunction (e.g. hypotonia [collapse], syncope, incontinence)

(c) Severe gastrointestinal symptoms (e.g. severe crampy abdominal pain, repetitive vomiting), especially after exposure to non-food allergen

(2) Acute onset of hypotension or bronchospasm or laryngeal involvement after exposure to a known or highly probable allergen for that patient (minutes to several hours), even in the absence of typical skin involvement

4. Management

- ABCs
- IM adrenaline (may require infusion)
- Volume expansion +/- inotropes
- Bronchodilators e.g. nebulised beta-2 agonists
- Glucocorticoids: IV hydrocortisone (PO prednisolone if mild)
- Antihistamines: IV chlorpheniramine
- Glucagon for refractory cases
- Drug allergy reporting, MedicAlert

II) Type IV Hypersensitivity

A. Allergic contact dermatitis

1. Definition: delayed type-hypersensitivity reaction triggered by contact with an allergen in previously sensitised individuals through the activation of allergen-specific T-cells

2. Examples of dental allergens:

- Chlorhexidine
- Rubber accelerators
- Acrylics
- Dental restorative materials
- Others e.g. nickel, colophony, glutardaldehyde

3. E.g. cheilitis caused by dentrifices, dental restorations, orthodontic devices, rubber dams

4. Diagnosis confirmed by patch testing

- Applying specific allergens at low concentration to the patient's skin (usually on the back) which induces cutaneous inflammation
- Patient has the patch on for 2-3 days
- When removed, severity of any delayed hypersensitivity reactions is graded

B. Stevens-Johnson syndrome (SJS)/toxic epidermal necrolysis (TEN)

1. Definition: rare, acute, serious and potentially fatal skin reaction in which there are sheet-like skin and mucosal loss

- SJS: <10% epidermal detachment
- SJS/TEN overlap: 10-30% epidermal detachment
- TEN: >30% epidermal detachment

2. Aetiology

a. Drugs (80% in TEN, 50% in SJS): high risk – allopurinol, sulfamethoxazole, sulfadiazine, sulfapyridine, sulfadoxine, sulfasalazine, carbamazepine, lamotrigine, phenobarbital, phenytoin, phenylbutazone, nevirapine, oxicam NSAIDs, thiacetazone

b. Chemicals

c. *Mycoplasma pneumoniae*

d. Viral infections

e. Immunisations

3. Clinical manifestations

a. First drug exposure to onset of symptoms: 1-3 weeks, more rapid with rechallenge

b. Prodrome

- Fever, malaise, arthralgia 1-3 days prior to mucocutaneous lesions
- Mild-moderate skin tenderness/conjunctival burning or itching → skin pain, burning sensation, tenderness, paraesthesia
- Painful/tender mouth lesions, impaired alimentation, photophobia, painful micturition
- Anxiety

c. Skin lesions

- Prodromal: morbilliform, targetoid (2 zones: flat and irregular shape with darker colour/blister in centre), +/- purpura, rapid confluence; can also start with diffuse erythema
- Early: macular areas with crinkled surface, enlarge and coalesce, sheetlike loss of epidermis, Nikolsky sign, trauma causes exposed/red/oozing dermis (c.f. second-degree thermal burn)
- Recovery within days, completed in > 3 weeks; skin/nail/eyelashes shed

d. Mucous membranes

- Erythema, painful erosions
- Eyes: conjunctival lesions – hyperaemia, pseudomembrane formation, keratitis, corneal erosions, synechiae

e. Other findings

- Fever, tachycardia
- Acute renal failure (tubular necrosis)
- Respiratory and gastrointestinal tracts: sloughing of epithelium with erosions

4. Management

- Early diagnosis, withdrawal of drug
- Intermediate or intensive care unit
- Replacement of IV fluids and electrolytes
- Analgesics
- Systemic glucocorticoids (early disease)
- Intravenous immunoglobulin (IVIg)
- Cyclosporine
- Etanercept
- Supportive

C. Drug exanthem

- Morbiliform skin rash due to a drug

III) Eczema/Dermatitis

1. Definition: Red, scaly itchy skin condition +/- weeping

Endogenous	**Exogenous**
• Atopic eczema • Asteatotic dermatitis/eczema craquale • Discoid eczema • Seborrhoeic dermatitis/dandruff • Stasis eczema • Pityriasis alba • Hand eczema	• Irritant contact dermatitis • Allergic contact dermatitis • Infective dermatitis

2. Clinical features

a. Acute eczema

- Rapidly evolving red rash
- Blistered, swollen, weeping

b. Chronic eczema

- Longstanding irritable area
- Dark, thickened, scratched
- Lichenification
- Nodular prurigo

3. Treatment

a. Moisturisation

Class	**Mechanism of action**	**Examples**
Occlusives	Hydrophobic film to reduce transepidermal water loss	Petrolatum, beeswax, mineral oil, silicones, lanolin, zinc oxide
Humectants	Attract and bind water from deeper epidermis to stratum corneum	Glycerol, propylene glycol, panthenol sorbitol, urea, alpha hydroxy acids, hyaluronic acid
Emollients	Smooth skin by filling cracks between desquamating corneocytes	Cholesterol, squalene, fatty acids, fatty alcohols, pseudoceramides
Protein rejuvenators	Replenish essential proteins	Collagen, elastin, keratin

b. Anti-inflammatory

i. Topical: topical corticosteroids, topical calcineurin inhibitors (tacrolimus, pimecrolimus), tar preparations

- Potency of topical corticosteroid used depends on area of skin and severity of eczema (e.g. mild for thinner skin, more potent for thicker skin)
- Topical calcineurin inhibitors for thinner/delicate skin
- UK classification of topical corticosteroids

Potency	Examples
Mild	Hydrocortisone 1%
Moderate	Clobetasone butyrate 0.05% Betamethasone valerate 0.02%
Potent	Betamethasone valerate 0.1% Betamethasone dipropionate 0.05% Mometasone furoate 0.1%
Very potent	Clobetasol propionate 0.05%

- One fingertip unit (FTU) – medication from crease at the base of the finger to the tip → approximately 2 handprints in adult

ii. Systemic: corticosteroids, cyclosporine, azathioprine, methotrexate, mycophenolate mofetil, interferon gamma, dupilumab, JAK inhibitors (upadacitinib, abrocitinib, baricitinib)

iii. Phototherapy

c. Antipruritics

- Sedating systemic antihistamines: improve sleep
- Topical antihistamines not recommended (sensitisation)

d. Treat infections

- Bacterial infection: topical or systemic antibiotics
- Antiseptics: bleach baths
- Eczema herpeticum: acyclovir
- Fungal infections: topical or systemic antifungal therapies

e. Other treatments specific to certain types of eczema

i. Seborrhoeic dermatitis

- 3-5% salicylic acid
- Imidazole antifungals or shampoos
- Tar shampoo
- Selenium disulphide shampoo
- Low-potency topical corticosteroid

ii. Venous/stasis eczema

1. Treat varicose veins

IV) Psoriasis

1. Definition: Chronic disorder with polygenic predisposition and triggering environmental factors e.g. bacterial infection, trauma, drugs

Types of psoriasis	Types of psoriatic arthritis (PsA)	Nail involvement
1. Chronic plaque psoriasis 2. Guttate psoriasis 3. Inverse/flexural psoriasis 4. Pustular psoriasis 5. Erythrodermic psoriasis	1. Distal arthritis involving distal interphalangeal joints 2. Oligoarthritis 3. Symmetric polyarthritis resembling rheumatoid arthritis 4. Arthritis mutilans (pencil-in-cup deformity on X-ray) 5. Spondyloarthropathy	1. Pitting 2. Onycholysis 3. Yellowing (oil spot) 4. Ridging 5. Subungual hyperkeratosis

2. Chronic plaque psoriasis

a. Definition

- Systemic, inflammatory disease that predominantly affects the skin
- Common sites
    - Extensors of limbs especially elbows and knees
    - Trunk
    - Umbilicus
    - Lower back
    - Scalp involving hairline
    - Skin inside and behind ears
    - Palms, soles, nails
- May also affect skin folds e.g. axillae, between buttocks, genitals, under breast

b. Essential clinical diagnostic criteria

- Well demarcated lesion with or without silvery/white scales

c. Supportive clinical examination diagnostic criteria

- Pink to red (grey in deeply pigmented skin)
- Vary in size
- Palpable
- Symmetrically distributed
- Family history in first degree relatives
- Nail involvement
- Joint pain and/or stiffness
- Itching

3. Treatment

a. Mild-to-severe: topical therapy

- Tar
- Corticosteroids
- Vitamin D analogues
- Calcineurin inhibitors (face, flexures)
- Dithranol

b. Moderate-to-severe

- Phototherapy
- Systemic therapy: methotrexate, acitretin, cyclosporine
- Biologics

4. Dental aspects

- Oral lesions rare: white/reddish mucosal plaques
- Higher prevalence of erythema migrans and fissured tongue, particularly in pustular psoriasis
- TMJ involvement in PsA (up to 60%)
- Oral complications from treatment
    - Gingival swelling from cyclosporine
    - Ulceration from methotrexate

V) Lichen planus

1. Definition

- Acute or chronic inflammatory dermatosis
- Predilection for flexural aspects of arms and legs
- Alteration in cell-mediated immunity
- Associations
    - Drugs
    - Metals: gold, mercury
    - Hepatitis C virus
    - HLA-associated genetic susceptibility

2. 4Ps of lichen planus

- Papule
- Purple
- Polygonal
- Pruritic

3. Oral lesions

a. Wickham striae (fine white or gray lines or dots)

b. Desquamative gingivitis

4. Treatment

a. Local therapy

- Cutaneous: topical glucocorticoids
- Cutaneous/oral/lips: intralesional triamcinolone
- Severely symptomatic oral: cyclosporine/tacrolimus solutions

b. Systemic therapy

- Cyclosporine
- Glucocorticoids
- Acitretin

c. Phototherapy

d. Other treatments: mycophenolate mofetil, enoxaparin, azathioprine

5. Dental aspects

- Oral lesions
    - Can persist for years
    - Bilateral, symmetrical
    - Posterior buccal mucosa > tongue/gingivae/other sites
- Gingival lesions atrophic and red (desquamative gingivitis)
- Replacement of amalgam with non-metallic alternatives may lead to resolution but relapse is possible

VI) Pemphigus vulgaris

1. Pemphigus

- Autoimmune bullous disorder
- Loss of cell-to-cell adhesion in epidermis (acantholysis)

2. Pemphigus vulgaris

- Usually starts in oral mucosa
- Less frequently, starts with generalised acute eruption of bullae

- Burning and painful erosions
- Epistaxis, hoarseness, dysphagia
- Weakness, malaise, loss of weight

3. Clinical manifestations

a. Vesicles and bullae

- Serous content
- Flaccid
- Easily ruptured
- Weeping
- On normal skin

b. Erosions

- Bleed
- Crusts (scalp)

c. Signs

- Nikolsky sign: dislodging of normal-appearing epidermis by lateral finger pressure in vicinity of lesions
- Pressure on bulla → lateral extension of blister

d. Mucous membrane erosions

- Nose
- Mouth
- Pharynx
- Larynx
- Vagina

4. Progression

- Localised for months → generalised
- Death without treatment
- Morbidity related to glucocorticoids and immunosuppression

5. Investigations

a. Skin biopsy for histopathological examination and immunofluorescence (IF)

- Intraepidermal: suprabasal bulla
- Direct immunofluorescence: intercellular deposits of IgG and C3 in epidermis

b. Serum autoantibodies (IgG) detected by indirect IF or ELISA

- Autoantibodies against desmoglein 1 and 3

6. Management

a. Glucocorticoids

b. Concomitant immunosuppressive therapy (glucocorticoid-sparing)

- Rituximab
- Mycophenolate mofetil
- Azathioprine
- Methotrexate
- Cyclophosphamide
- Plasmapheresis
- High-dose intravenous immunoglobulin (IVIg)

c. Other measures

- Cleansing baths
- Wet dressings
- Topical and intralesional glucocorticoids
- Antimicrobial therapy in documented bacterial infections
- Correction of fluid and electrolyte imbalance

d. Monitoring

- Clinical
    - Improvement of skin lesions
    - Development of drug-related side effects
- Laboratory
    - Pemphigus antibody titres
    - Haematologic and metabolic indicators of drug-induced adverse effects

VII) Other relevant skin conditions

1. Herpes labialis (herpes simplex)

2. Basal cell carcinoma: rodent ulcer, pearly appearance, rolled edges

3. Squamous cell carcinoma

4. Melanoma (ABCDEF rule)

- Asymmetry
- Border – irregular
- Colour – not uniform/multiple
- Diameter (>6 mm) or “Ugly Duckling” sign
- Elevation/evolving
- Funny mole

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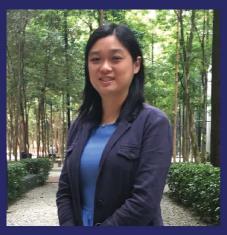

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