

Medicine

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# Chapter 1

## Cardiology

### 1.1 Presenting problems in CVS disease

#### Features of benign murmur

- Soft
- Midsystolic
- Heard at left sternal edge
- No radiation
- No other cardiac abnormalities

### 1.2 ECG

#### Anatomy of an ECG



## Abnormalities of components

### Pathological Q

- Depth  $> 2\text{mm}$
- Height  $> 1\text{mm}$
- Present in  $\geq 2$  leads
- Assocd with loss of R height ( $Q > R/4$ ; normally  $Q \leq R/4$ )
- Indicates *transmural* myocardial necrosis

### Segments vs intervals

- e.g. ST segment = end of S  $\rightarrow$  start of T
- PR interval = start of P  $\rightarrow$  start of R

### ST segment elevation

- Normal: upto 1mm in limb leads, upto 2mm in chest leads
- Causes
  - **STEMI: convexity** upwards
  - **Acute periCARDitis:: conCAvity** upwards
- Indicates ongoing myocardial injury

## Myocardial infarction

A somewhat interesting physiological explanation on how the changes arise

### Sites of infarction based on lead

- Septal:  $V_1, V_2$
- Anterior:  $V_3, V_4$
- Lateral: I, aVL,  $V_5, V_6$
- Extensive anterior:  $V_1-V_6$
- Anterolateral: I, aVL,  $V_1-V_6$

### Reciprocal changes

- Acute STEMI in some surface of the heart  $\rightarrow$  ST elevation in corresponding leads, and ST depression in reciprocal leads

Site	Facing	Reciprocal
<i>Septal</i>	$V_1, V_2$	$V_7, V_8, V_9$
<i>Anterior</i>	$V_3, V_4$	None
<i>Lateral</i>	I, aVL, $V_5, V_6$	II, III, aVF
<i>Inferior</i>	II, III, aVF	I, aVL
<i>Posterior</i>	$V_7, V_8, V_9$	$V_1, V_2$

### Basic pathophys of STEMI

- Occurs due to proximal **complete occlusion of major coronary artery**



A. Before the onset of infarction

↓

B. In acute phase, ST elevation

↓

C. Progressive loss of R and deepening Q

↓

D. Resolution of ST elevation; fully developed pathological Q; T inversion

↓

E. In old infarcts, T-wave inversion may or may not persist

- ST elevation resolves after a few days

**NSTEMI**

- **Partial occlusion of major or complete occlusion of minor** coronary artery
- *Subendocardial/partial-thickness MI* → **no pathological Q**
- **ST depression + T inversion** in chest leads

**1.3 Coronary Artery Disease**

- Diseases arising due to narrowing of the lumen of one or more coronary arteries and the resulting myocardial ischaemia/infarction.
- **Types:**
  - Stable angina: Fixed atheromatous stenosis
  - Unstable angina:
    - \* dynamic obstruction
    - \* due to plaque rupture/erosion with thrombosis
  - MI
  - Heart failure
  - Arrhythmia
  - Sudden cardiac death
    - \* ventricular arrhythmia
    - \* asystole
    - \* massive MI

**1.4 Arrhythmias****Classification according to ECG morphology**

- **Narrow complex:** QRS < 120ms (3 small sqs)
  - Sinus tachycardia
  - Atrial fibrillation (irregular narrow complex tachycardia)
  - Atrial flutter
  - AV Nodal Re-entry Tachycardia (AVNRT aka SVT)
- **Broad complex:** QRS > 120ms (3 small sqs)
  - Ventricular tachycardia
  - AV Re-entry Tachycardia (AVRT e.g. Wolff-Parkinson-White syndrome)
    - \* Abnormal band of conductive tissue connecting atria and ventricles (accessory pathway)

**Management of SVT**

- Carotid sinus massage or
- Valsalva manoeuvre
- If the manoeuvre fails,

- Adenosine (3-12mg IV) or
- Rate-limiting CCB (Verapamil 5mg IV) or
- $\beta$ -blocker
- If haemodynamic state compromised, DC cardioversion
- Recurrent SVT  $\rightarrow$  catheter ablation

## 1.5 Atrial fibrillation

### Causes

- CAD (including acute MI)
- Mitral stenosis (MS; rheumatic mitral valve disease)
- Hypertension
- Thyrotoxicosis
- Cardiomyopathy
- Pulmonary embolism

### Investigations

- ECG
- Echo: to see valvular condition
- Thyroid function test: to exclude thyrotoxicosis

### Management of AF

- **Rhythm control:**
  - Pharmacological cardioversion
    - \* Pt stable + no history of heart disease  $\rightarrow$  IV flecainide
    - \* Structural / ischaemic heart disease  $\rightarrow$  IV amiodarone
  - DC cardioversion if drugs fail
- **Rate control**
  - $\beta$ -blockers
  - Digoxin
  - Rate-limiting CCB: verapamil / diltiazem
- **Thromboprophylaxis:**
  - Oral Warfarin
  - Target INR: 2.0-3.0
  - Reduces risk of stroke by  $\frac{2}{3}$
  - Start 4wks before cardioversion, continue till 3mo after successful cardioversion



# Chapter 2

## Dermatology

### 2.1 Anatomy and physiology

- Layers of skin:
  - Epidermis: further layered into (from out→in)
    - \* corneum
    - \* lucidum
    - \* granulosum
    - \* spinosum
    - \* basale
  - Dermis: contains
    - \* blood vessels
    - \* nerves
    - \* pilosebaceous units (hair follicle + sebaceous gland)
  - Subcutis: adipose

### Epidermal appendages

- Hair follicles:
  - phases of growth
    - \* anagen:
      - active growth
      - lasts years in scalp hairs
    - \* catagen:
      - transitional
      - lasts days (in scalp)
    - \* telogen:
      - resting
      - lasts months (in scalp)
- Sebaceous glands
  - usually *associated with a hair follicle*

- androgens  $\rightarrow \uparrow$  sebum
- oestrogen  $\rightarrow \downarrow$  sebum

- Sweat glands
  - innervated by *sympathetic cholinergic* fibres

## 2.2 Principles of management of skin disease

### Topical treatments

- Ointments vs Creams
  - Ointments preferred to creams for dry skin (e.g. chronic eczema) as
    - \* more hydrating
      - 80% oil + 20% water in ointments (vs 50-50 for creams)  $\rightarrow$  prevent water loss from skin by oil layer
    - \* less preservatives  $\rightarrow$  less risk of allergy
- Emollients
  - Moisturise, lubricate, protect skin
  - *Vehicles without active drug*
- Glucocorticoids

### Phototherapy

- UVB
- Psoralen UVA
  - Psoralen:
    - \* natural photosensitiser from plant source
    - \* cross-link DNA strands on excitation with UVA
  - Cumulative exposure to PUVA  $\rightarrow \uparrow$  risk of SCC, so reserved for UVB resistance
- Uses
  - Psoriasis
  - Atopic eczema
  - Vitiligo
  - Chronic urticaria

### Systemics

- Antihistamines
- Retinoids
  - *Anti-inflammatory*
  - Promote *differentiation of skin cells*

- **Teratogenic**
  - \* must be prescribed with robust contraception
  - \* females must have negative pregnancy test before, during, and after therapy
- **Immunosuppressants**
  - Glucocorticoids e.g. prednisolone
  - Methotrexate
  - Azathioprine

## Biologics

- Biological *inhibitors of proinflammatory cytokines*
- **TNF- $\alpha$  inhibitors**
  - Infliximab
  - Etanercept
- **Interleukin inhibitors**
  - Ustekinumab: IL-12, 23
  - Guselkumab: IL-23
  - Secukinumab: IL-17
- *Rituximab*:
  - Binds to CD20  $\rightarrow$  cause ADCC of B cells
  - As terminally differentiated plasma cells don't have CD20 they're safe
  - Use: pemphigus vulgaris

## Non-surgical therapy

- **Cryo**
  - *Liquid N<sub>2</sub>*
  - Causes cell membrane destruction  $\rightarrow$  death
- Laser
- PDT / photodynamic therapy

## 2.3 Skin cancers

### Classification

- Non-melanoma skin cancer (NMSC): most common
  - SCC
  - BCC
- Melanoma
  - Less common
  - More metastatic risk  $\rightarrow$  cause of most skin cancer deaths

## 2.4 Fungal infections

### Types

- Superficial
  - Dermatophytes: aka **ringworm** / **tinea**sis
    - \* *Trichophyton*
    - \* *Epidermophyton*
    - \* *Microsporum*
  - Yeast
- Deep: less common
  - Chromomycosis
  - Sporotrichosis

## 2.5 Scabies

### Agent

Caused by the mite *Sarcoptes scabiei hominis*

### Diagnosis

- Identify the skin burrow
- Visualize the mite by dermatoscope / extracting with a needle

### Treatment

- Affected + all asymptomatic family members / physical contacts
- Topical permethrin / malathion
  - 2 applications
  - 1 wk apart
  - Whole body, except head
- Oral Ivermectin:
  - Single dose
  - For poor adherence, immunosuppression or heavy infestation

## 2.6 Acne

- *Chronic inflammation of pilosebaceous units*

## Pathogenesis

Key components are:

- ↑ Sebum production
- Colonisation of pilosebaceous ducts by *Propionibacterium acnes*
- Occlusion of pilosebaceous ducts

## Features

- Hallmark: **comedone**
- Greasiness of skin

## Management

- **Mild disease**
  - Topical Benzoyl peroxide
  - Topical Retinoids
  - Topical antibiotics
    - \* Erythromycin
    - \* Clindamycin
- **Moderate disease:** topical *plus*
  - Systemic tetracycline
  - Oestrogen containing OCP
  - Isotretinoin: if inadequate response to topical+systemic therapy for 6 months
- **Severe disease**
  - Isotretinoin 0.5-1 mg/kg for 4 months:
    - \* Reduce sebum secretion and follicle colonisation
    - \* Teratogen
    - \* Pregnancy must be avoided during treatment *and* within 2 mo of drug cessation
  - Systemic glucocorticoid (with isotretinoin)
  - If unable to use isotretinoin
    - \* UVB phototherapy
    - \* PDT

## 2.7 Eczemas

- Seborrhoeic dermatitis is associated with *Malassezia* yeasts

### Features

Most types have the following clinical features:

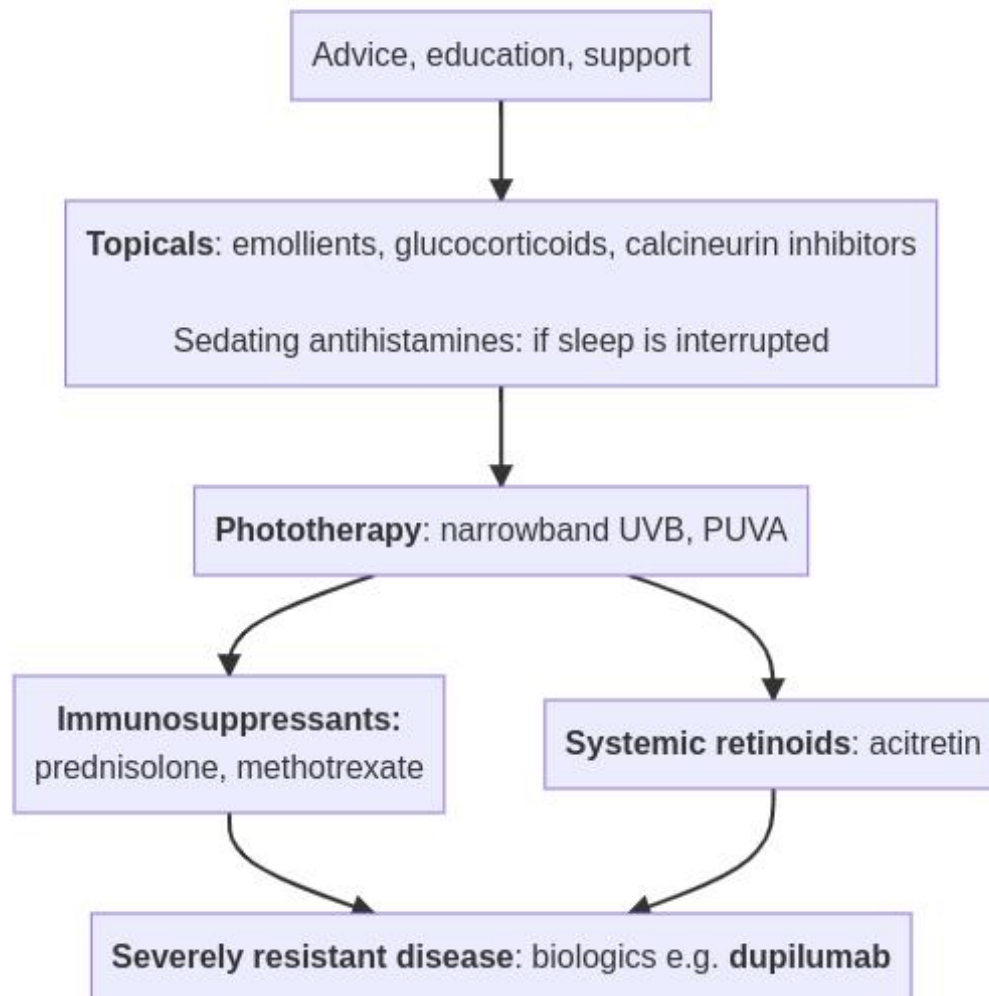
#### Acute

- Ill-defined erythema, oedema
- Papules, vesicles, bullae
- Exudation
- Scaling

#### Chronic

- Above features
- Lichenification
  - Skin thickening with pronounced skin markings, 2° to chronic scratching
  - Fissures
  - Dyspigmentation

## Management of eczema



## 2.8 Psoriasis

- Chronic inflammatory hyperproliferative skin disease
- **Characteristics**
  - **Well-defined erythematous scaly plaques**
  - Affecting **extensor surfaces, scalp, nails**

### Histological features

- Keratinocyte hyperproliferation + abnormal differentiation → nucleated stratum corneum cells (transit time from basale to corneum reduced to 5 from 28 → keratinocytes reach the surface while immature)
- Inflammation with Th-1 and Th-17 infiltration
- Tortuosity of dermal capillaries and release of VEGF

### Exacerbating factors

- **Sunlight**
- **Trauma**
- **Infection**
  - $\beta$ -haemolytic strep ↑ guttate psoriasis
  - HIV may initially present with severe psoriasis
- **Drugs**
  - Antimalarials
  - $\beta$ -blockers
  - Lithium
  - NSAIDs
- **Stress and anxiety**

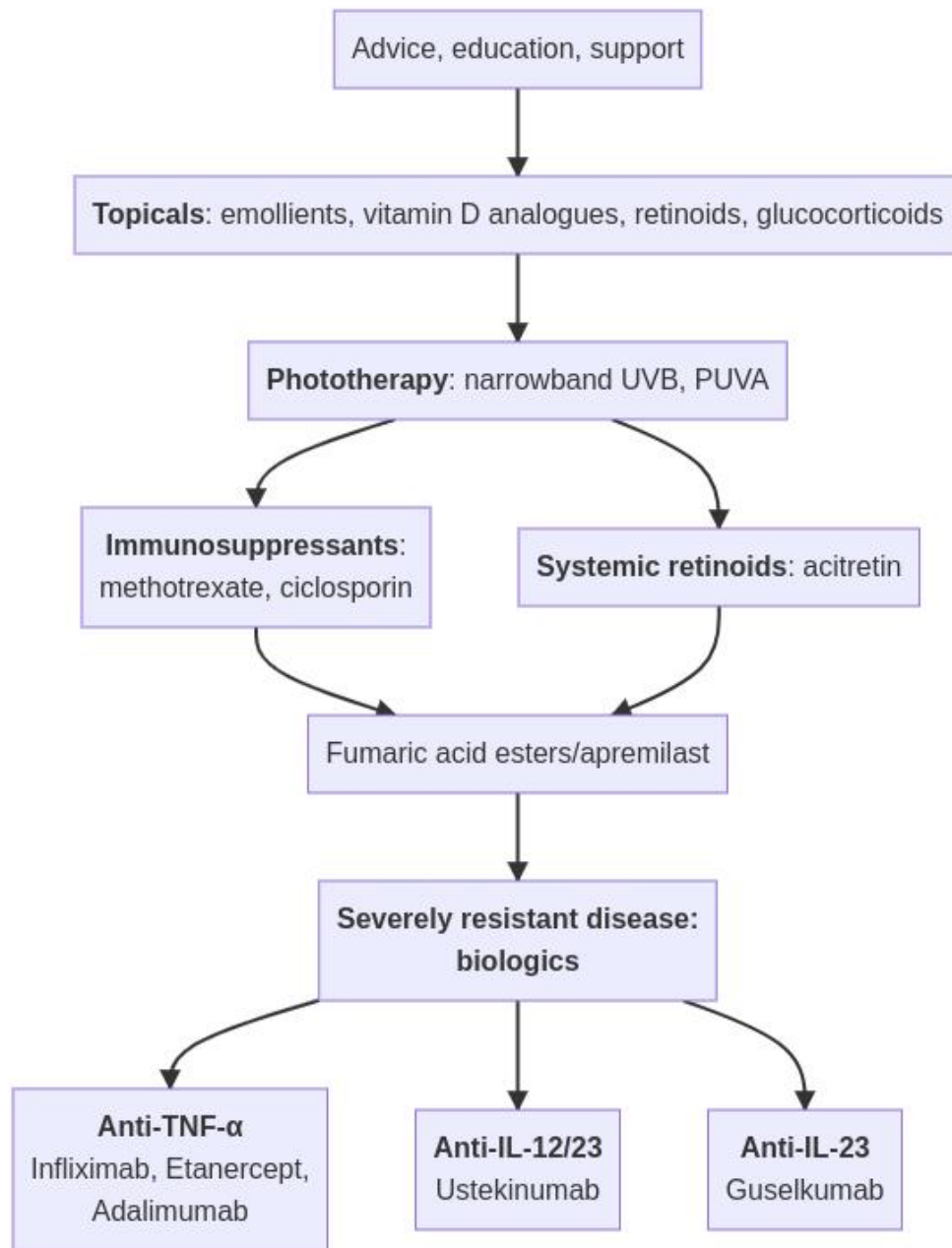
### Clinical types

- **Plaque psoriasis:**
  - most common
  - well-demarcated erythematous plaques
  - silver-white scales in untreated
    - \* bleed on scraping (due to dilated vessels underneath) → **Auspitz sign**
  - **Sites**
    - \* extensor surfaces
      - elbows
      - knees
      - lower back
    - \* scalp
    - \* nails



- **Guttate** psoriasis:
  - follows *Strep* throat
  - common in children/adolescent
  - UVB highly effective
  - may herald the onset of plaque psoriasis in adulthood
- **Erythrodermic** psoriasis: generalised → medical emergency
- **Pustular** psoriasis

## Management of psoriasis



## 2.9 Hypopigmentation

### Causes

- Vitiligo
- Albinism
- Pityriasis alba
- Pityriasis versicolor

### Vitiligo

- **Acquired**
- Cell-mediated **autoimmune destruction of melanocytes**
- Loss of melanocytes → hypopigmented patches

### Albinism

- **Autosomal recessive**
- **Reduced melanin production by normal number of melanocytes**
- ↑↑ risk of sunburn, skin cancer

## 2.10 Hyperpigmentation

### Causes

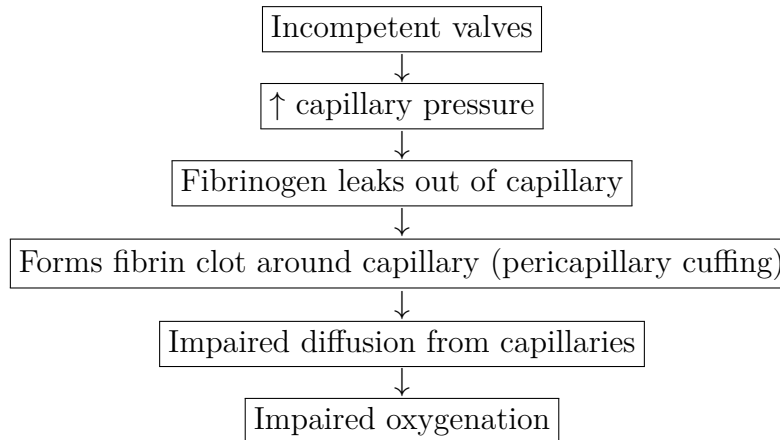
- **Endocrine**
  - Melasma/chloasma:
    - \* in pregnancy / some OCP users
    - \* discrete patches of facial pigmentation
  - Addison's disease
  - Cushing's syndrome
  - Nelson's syndrome
    - \* hyper-ACTH 2° to bilateral adrenalectomy for Cushing's
    - \* due to loss of -ve feedback from plasma cortisol
  - CKD
- **Drugs**
  - Amiodarone
  - Anti-cancers:
    - \* Bleomycin: Hodgkin's
    - \* Busulfan: CML
  - Chloroquine
  - Psoralens

## 2.11 Pseudorandom factoids

### SPF (sun protection factor)

- $\frac{\text{UV dose for producing erythema with sunscreen}}{\text{UV dose for producing erythema without sunscreen}}$

### Mechanism of venous ulceration



# Chapter 3

## Neurology

### 3.1 Raised ICP

- Normal ICP = **5-15 mmHg**

#### Causes

- **ICSOL**
  - Intracranial haemorrhage
  - Tumours e.g. glioma
  - Brain abscess
- **Hydrocephalus:** blockade of CSF circulation
  - Obstructive / non-communicating
  - Communicating
- **Cerebral oedema** e.g. meningoencephilitis
- **Venous sinus obstruction** e.g. cerebral venous thrombosis

#### Features

- **Headache**
- **Vomiting**
- **Diplopia / blurred vision:** Due to *6th nerve palsy*
  - 6th nerve palsy due to
    - \* stretching of the long, slender nerve
    - \* compression against petrous temporal bone
- **Papilloedema**
- **Bradycardia**
- **Hypertension**
- **Depressed consciousness**

## Management

- According to cause:
  - Mass lesion → surgical decompression
  - Hydrocephalus → *ventriculoperitoneal shunt* operation
  - Oedema → glucocorticoids
- Supportive:
  - Head elevation
  - Fluid balance
  - BP control
  - Diuretics: mannitol

## 3.2 Neurological emergencies

- **Status epilepticus**
- **Stroke** (if thrombo)
- **Subarachnoid haemorrhage**
- **Cord compression**
- **GBS**
- **Myasthenia gravis** (if bulbar and/or respiratory)