MFDS PART 1 · ORAL MEDICINE

Oral Medicine 1

Lecture 11 · Dr Mohamed J. Abuasi (BDS, MJDF, MFD RCSI) · CoNNect International Academy

EXAM-DAY SUMMARY

everything on this page → detail in §§ overleaf

SCARLET FEVER & TB → §1

- Scarlet fever: 4–8 yrs; strep erythrogenic toxin → strawberry tongue
- White coating + oedematous fungiform papillae → sheds → red smooth dorsum
- TB: 1/3 population; oral involvement rare; deep painful ulcer, raised borders
- TB site: posterior dorsum of tongue → refer chest physician

SYPHILIS → §1

- Spirochaetes; condyloma lata in moist warm areas
- 1° chancre (tongue) → 2° snail-track (hard palate) → 3° gumma (palate perforation)
- Gumma → oro-antral communication; premalignant

GIANT CELL LESIONS → §2

- Peripheral GCG + pyogenic granuloma = reactive lesions, gingival margins, pedunculated with a neck
- Central GCG: inside bone, lower anteriors, bone resorption on x-ray

COXSACKIE → §3

- A → herpangina · hand, foot and mouth; B → heart/liver/pancreas, myocarditis
- Herpangina: children; uvula + palate; gingiva NOT affected
- HFM: whole cavity, mainly palate; rash hands + feet; gingiva rarely affected
- Both self-heal 10–14 days

HERPESVIRUSES & ONCOGENIC → §3

- HHV-1/2 HSV · HHV-3 VZV · HHV-4 EBV · HHV-5 CMV
- EBV: mononucleosis, hairy leukoplakia, Burkitt's, nasopharyngeal ca
- CMV: salivary glands → sialadenitis; transplant + HIV
- HTLV-1 leukaemia · HBV/HCV hepatocellular ca · HPV SCC/warts/papilloma

ORAL CANCER → §4

- SCC; 30 000/yr; 5-yr survival 50% (76% localised, 19% metastatic)
- Sites: lips (most common) · floor of mouth · lateral tongue
- Signs: leuko-/erythroplakia; painless non-healing ulcer; numbness = late neural invasion
- Survival ↓: stage, site, age, medical history, male, white/black race

TNM CUT-OFFS → §4

- T1 ≤2 cm + DOI ≤5 mm · T3 >4 cm or DOI >10 mm
- T2 = ≤2 cm with DOI 5–10 mm, or 2–4 cm with DOI ≤10 mm
- T4b: masticator space, pterygoid plates, skull base, encases carotid
- N1 single ipsilateral ≤3 cm ENE− · N3a >6 cm ENE− · M1 distant

PRIMARY HSV → §5

- PHG: children 6 months – 6 years; vesicles → ulcers on ALL oral mucosa
- Prodrome: fever, headache, malaise, anorexia, lymphadenopathy
- Heals 10–14 d → dormant in trigeminal ganglion
- Avoid touching ulcers → herpetic whitlow

ACYCLOVIR → §5

- Topical, prodrome only: 5 times a day for 5 days
- After vesicles erupt → supportive only (rest, fluids, nutrition) + analgesic/antihistamine
- Systemic: very young, immunocompromised, severe, unresponsive, >14 d unhealed
- Herpes labialis = reactivation (cold, sun, ↓ immunity)

VARICELLA ZOSTER → §6

- Chickenpox: 5–9 yrs, crusted in 1–2 days, oral rarely affected
- Shingles: unilateral, trigeminal branches, NEVER crosses midline; clears 2–4 weeks
- Acyclovir within first 3 days ↓ post-herpetic neuralgia; eye → ophthalmologist
- Ramsay Hunt: facial palsy + ulcers, deafness; systemic acyclovir 400–800 mg

EM & STEVENS–JOHNSON → §7

- EM: recurrent blistering, ruptures 2–3 d; skin = target/iris lesions
- Minor: oral, eye, genital, skin. Major: + multi-organ, bloody crusted lips, symblepharon
- Major → GP + ophthalmologist + steroids
- SJS: drug reaction; flu-like → rash → blisters → skin peels; ITU/burns unit emergency

PV VS MMP → §7

- PV intraepithelial (prickle cell layer): flaccid, fluid + blood, ruptures easily, Nikolsky +ve
- MMP subepithelial: tense, fluid only, resists rupture, Nikolsky −ve
- PV histology: acantholysis, intact basement membrane. MMP: BM separation
- PV fatal → fluid + blood loss, dehydration, superimposed infection
- Ix: biopsy + direct/indirect immunofluorescence

LICHEN PLANUS GROUP → §8

- Bilateral buccal mucosa; Wickham's striae; erosive form premalignant; F:M 3:2
- Six forms: papular, plaque-like, reticular, erosive, bullous, atrophic
- Histology: hyperkeratosis, saw-shaped rete pegs, basal cell degeneration, lymphocytic band
- Lichenoid: amalgam, HCV, DM/HTN drugs, NSAIDs → remove cause
- Steroids: topical 4×/day × 1 month → intralesional after 3 weeks → systemic

§1 Bacterial infections of the oral mucosa

KEY POINTS

Four bacterial entries: scarlet fever · tuberculosis (TB) · syphilis · giant cell granulomas (§2). Only scarlet fever and syphilis give **pathognomonic** oral signs — strawberry tongue and snail-track ulcers.

1.1 Scarlet fever

- **Age**: ★**4–8 years**
- **Cause**: streptococcal **erythrogenic toxin** → delayed hypersensitivity reaction
- **Oral mucosa**: redness · soreness — non-specific
- **Tongue = pathognomonic**, two phases:
  - **Early** → white coating on dorsum + oedematous fungiform papillae showing through
  - **Later** → coating sheds → dorsum red + smooth, oedematous fungiform papillae persist = ★**"strawberry tongue"**

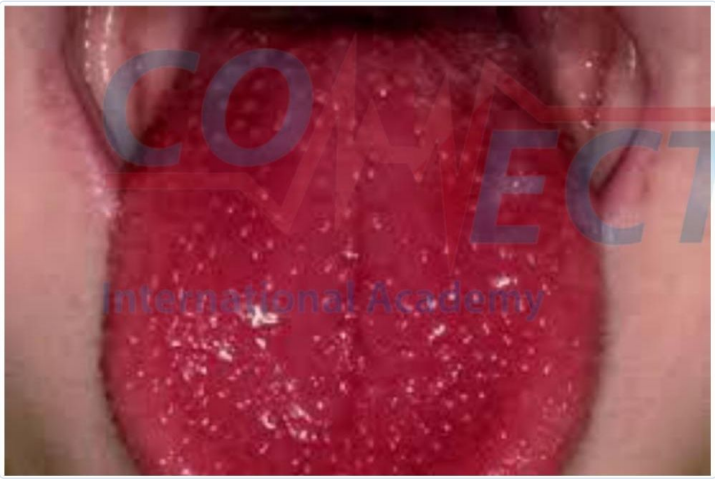

Fig. 1 — Strawberry tongue: coating shed, dorsum red and smooth, oedematous fungiform papillae standing out as white-red dots.

1.2 Tuberculosis

- **Nature**: severe chest infection · usually **immunocompromised** patient
- **Burden**: ★**1/3 of the population affected** — but oral involvement **rare**
- **Oral lesion if present**: **deep painful ulcer** · raised borders · gradually ↑ in size
- **Site**: anywhere in the oral cavity, esp. ★**posterior dorsum of tongue**
- **Action**: refer to **chest physician** for investigation + management

1.3 Syphilis

- **Organism**: bacterial **spirochaetes**
- **Condyloma lata**: large raised grey/white lesions · develop in **moist warm areas** → underarm, oral cavity

Table 1 — Syphilis: the three stages and their oral lesions

Stage	Lesion	Description	Site
1st	**Chancre**	Rounded, **hard**, elevated ulcer with central necrosis	Mainly tongue
2nd	**Snail-track ulcer**	Small ulcers adhering to each other → snail-track shape	Hard palate
3rd	**Gumma**	Perforation of palate → oro-antral communication · **premalignant**	Palate

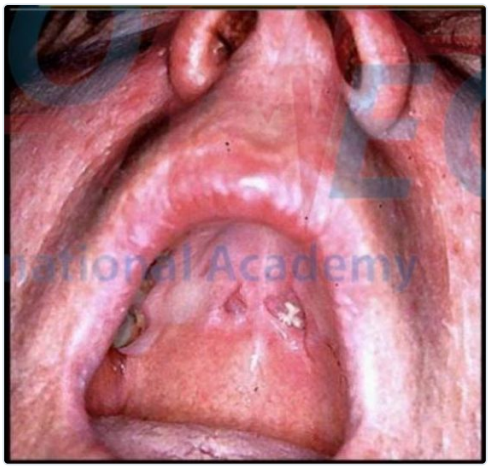

Fig. 2 — Gumma of tertiary (here congenital) syphilis: destructive palatal perforation opening the oro-antral/oro-nasal communication.

★ EXAM PEARL

The deck writes the third stage as **"Gamma"** and the defect as "oro-*anteral*" — read **gumma** and **oro-antral**. Also note **condyloma lata is a secondary-stage lesion**, not a general feature of the disease, and the premalignant change of late syphilis is the **leukoplakia of syphilitic glossitis** rather than the gumma itself. Learn the deck's mapping (chancre → snail track → gumma) but use the correct names in an exam.

§2 Reactive lesions and the giant cell granulomas

KEY POINTS

Peripheral giant cell granuloma (PGCG) and pyogenic granuloma = **reactive lesions**, **soft tissue**, gingival margins, pedunculated. Central GCG = **inside bone**, lower anteriors, radiographic resorption.

2.1 Peripheral GCG and pyogenic granuloma

- **Mechanism**: chronic infection → irritation → reactivation of the tissue response = **"reactive lesions"**
- **Site**: soft tissue, esp. ★**gingival margins**
- **Form**: **pedunculated with a neck**

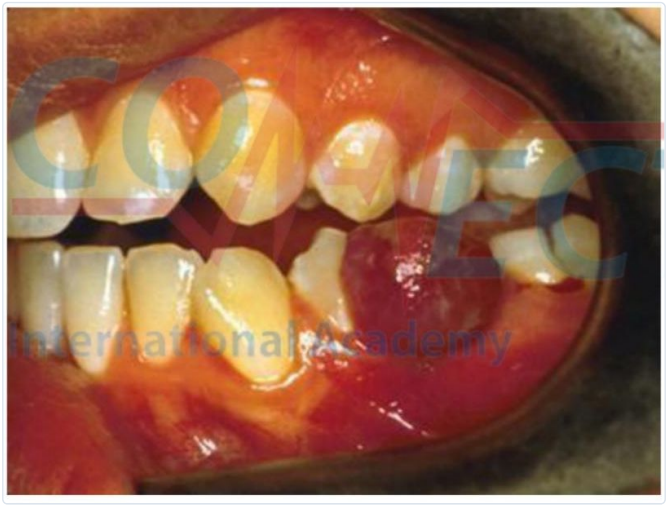

Fig. 3 — Peripheral GCG: pedunculated, neck-bearing gingival mass arising from the marginal gingiva, displacing the adjacent teeth.

2.2 Central GCG

- **Site**: **inside bone**, region of the ★**lower anteriors**
- **Radiograph**: associated **bone resorption**

★ EXAM PEARL

Peripheral vs central is a **site** question, not a histology question — both are giant-cell lesions; peripheral sits *on* gingiva, central sits *in* mandibular bone anteriorly. Note also that the accepted driver is chronic **irritation/trauma** (calculus, ill-fitting margins, extraction sockets), not infection as the slide states.

§3 Viral infections — mapping the viruses to the lesions

3.1 Coxsackie viruses

- **Coxsackie A** → **herpangina · hand, foot and mouth (HFM) disease**
- **Coxsackie B** → infections of **heart, liver, pancreas** → ★**myocarditis**

3.2 Herpangina

- **Who**: confined to **children**
- **Lesions**: small ulcers on oral mucosa, esp. ★**uvula + palate**
- **Gingiva**: ✗ not affected
- **Systemic**: fever · general upset
- **Course**: self-healing in **10–14 days**

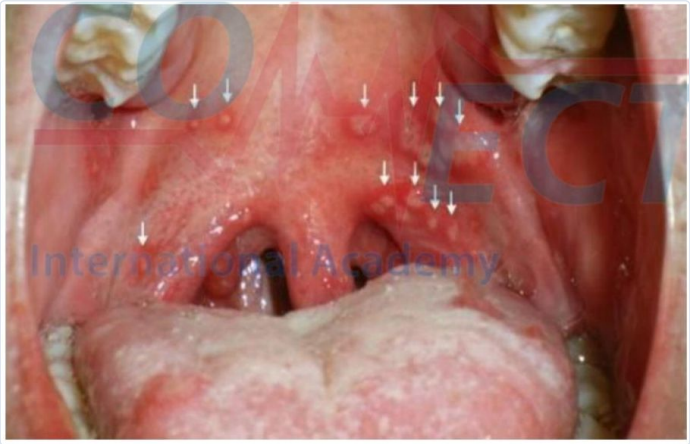

Fig. 4 — Herpangina: crops of small ulcers on the soft palate, uvula and tonsillar pillars; gingiva spared.

3.3 Hand, foot and mouth disease

- **Vs herpangina**: same picture but lesions **throughout the oral cavity**, mainly **palate**
- **Lesions**: papular, vesicular rash → ruptures → painful **superficial ulcers**
- **Extra-oral**: rash on ★**hands and feet** + nasal congestion
- **Gingiva**: rarely affected
- **Course**: self-healing in **10–14 days**

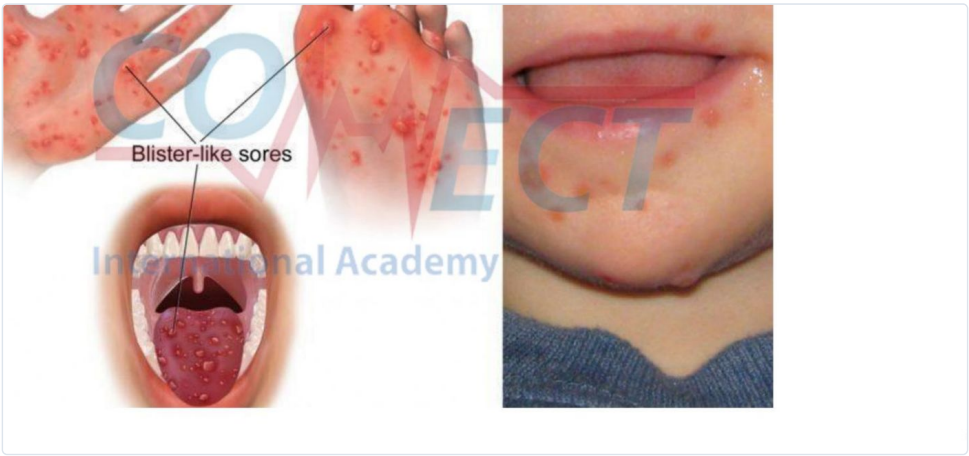

Fig. 5 — Hand, foot and mouth disease: blister-like sores distributed on palms, soles and oral mucosa, with perioral spread.

Table 2 — The three childhood vesicular stomatitides compared

	Herpangina	Hand, foot and mouth	Primary herpetic gingivostomatitis (§5)
Virus	Coxsackie A	Coxsackie A	HSV-1/2 (HHV-1/2)
Age	Children	Children	6 months – 6 years
Oral site	Uvula + palate	Whole cavity, mainly palate	All oral mucosa
Gingiva	Not affected	Rarely affected	**Affected** — gingivostomatitis
Skin	—	Hands + feet, nasal congestion	—
Course	Self-healing 10–14 d	Self-healing 10–14 d	Self-healing 10–14 d, then latency

★ EXAM PEARL

All three heal in **10–14 days**, so duration cannot separate them — the discriminators are **gingiva** (only herpes reliably involves it) and **skin** (only HFM gives hand/foot rash).

3.4 The human herpesvirus family

Table 3 — HHV-1 to HHV-5 and their disease associations

Virus	Name	Associated disease
HHV-1, 2	Herpes simplex (HSV)	Primary HSV · recurrent HSV infections
HHV-3	Varicella zoster (VZV)	Chickenpox · shingles · Ramsay Hunt syndrome
HHV-4	Epstein–Barr (EBV)	Infectious mononucleosis · hairy leukoplakia · Burkitt's lymphoma · nasopharyngeal carcinoma
HHV-5	Cytomegalovirus (CMV)	Viral sialadenitis · life-threatening disease in the immunocompromised

- **CMV detail**: mostly found in **salivary glands** · unnoticed in healthy patients · **life-threatening** in immunocompromised → viral **sialadenitis**
  - special consideration in ★**organ transplant and HIV**

3.5 Oncogenic viruses

- **HTLV-1** (human T-cell leukaemia virus 1) → **leukaemia**
- **Hepatitis B & C** → **hepatocellular carcinoma**
- **HPV** (human papilloma virus) → ★**squamous cell carcinoma** · warts · benign papilloma

§4 Oral cancer

KEY POINTS

Oral cancer = **squamous cell carcinoma (SCC)**, epithelial. **30 000** new cases/year · 5-year survival **50%** overall → **76%** localised vs **19%** metastatic. Early detection is the whole game.

4.1 Epidemiology and survival

- **Type**: usually epithelial = **squamous cell carcinoma**
- **Incidence**: ★**30 000 new cases diagnosed every year**
- **5-year survival**: **50%** overall · ★**76% localised · 19% metastatic**

4.2 Warning signs

- **Mucosal**: **leukoplakia** · **erythroplakia**
- **Ulcer**: ★**non-painful, non-healing, long history** — "no pain" is itself a warning
- **Function**: difficulty moving jaw or tongue · hoarseness of voice
- **Referred/other**: ear pain · jaw swellings
- **Iatrogenic driver**: chronic inflammation from ill-fitting restoration margin or denture
- **Late + neural invasion**: numbness of tongue or mouth

4.3 Role of the dentist in protection

- **Risk-factor elimination**: tobacco · alcohol · sunlight exposure
- **Screening**: detailed **extra-oral and intra-oral** examination of lymph nodes + soft tissues **at every visit**, with follow up
- **High-risk sites**:
  - ★**Lips** — most common site per the lecture, esp. skin often exposed to sun or active radiation
  - Floor of the mouth
  - Lateral surfaces of the tongue

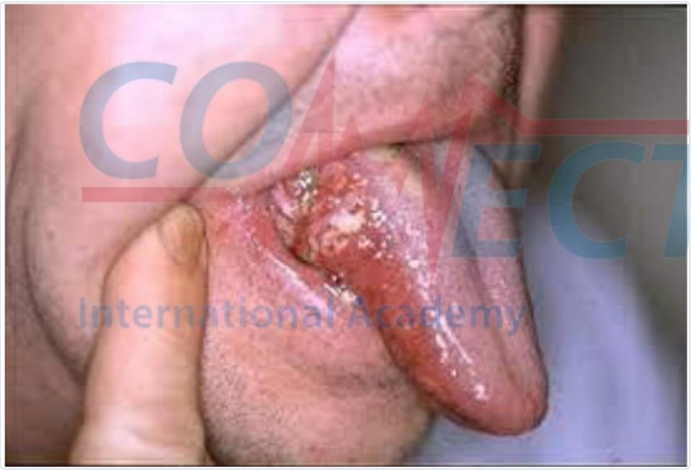

Fig. 6 — SCC of the lateral border of tongue: raised, rolled, indurated margins around a persistent ulcer — the classic high-risk site and appearance.

4.4 TNM staging

Table 4 — T category, oral cavity (size and depth of invasion, DOI)

T	Criteria
TX	Primary tumour cannot be assessed
Tis	Carcinoma in situ
T1	≤2 cm **and** DOI ≤5 mm
T2	≤2 cm with DOI >5 and ≤10 mm · **or** >2 cm but ≤4 cm with DOI ≤10 mm
T3	>4 cm · **or** any tumour with DOI >10 mm
T4	Moderately advanced or very advanced local disease
T4a	**Moderately advanced.** Lip → invades through cortical bone, or involves inferior alveolar nerve, floor of mouth, or skin of face (chin/nose). Oral cavity → invades adjacent structures only, e.g. through cortical bone of mandible/maxilla, or involves maxillary sinus or skin of face. **Superficial erosion of bone/tooth socket alone by a gingival primary is NOT sufficient for T4.**
T4b	**Very advanced.** Invades masticator space, pterygoid plates, or skull base, and/or encases the internal carotid artery

Table 5 — N category (ENE = extranodal extension)

N	Criteria
NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Single **ipsilateral** node ≤3 cm, **ENE−**
N2	Single ipsilateral ≤3 cm **ENE+** · or >3 but ≤6 cm ENE− · or multiple ipsilateral, none >6 cm, ENE− · or bilateral/contralateral, none >6 cm, ENE−
N2a	Single ipsilateral *or contralateral* ≤3 cm ENE+ · or single ipsilateral >3 but ≤6 cm ENE−
N2b	Multiple **ipsilateral**, none >6 cm, ENE−
N2c	**Bilateral or contralateral**, none >6 cm, ENE−
N3	Node >6 cm ENE− · or single ipsilateral >3 cm ENE+ · or multiple ipsilateral/contralateral/bilateral with any ENE+
N3a	Node >6 cm, ENE−
N3b	Single ipsilateral >3 cm ENE+ · or multiple ipsilateral/contralateral/bilateral with any ENE+

- **M**: **Mx** metastasis cannot be measured · **M0** no distant metastasis · **M1** distant metastasis

4.5 Treatment and prognosis

- **Radiation**: destruction of cells to stop their growth
- **Chemotherapy**: chemicals to destroy the cells
- **Surgery**: surgical removal of the tumour
- **Combination**: between methods
- **Factors affecting survival**:
  - Grading and staging · site of the primary tumour · complicating medical history
  - **Age** older groups more prone to SCC · **gender** males > females · **race** whites and blacks > Indians and Asians

> ★ EXAM PEARL
> The two numbers examiners lift straight out of the T table are the **DOI cut-offs, 5 mm and 10 mm** — size alone no longer determines T. Separately: the deck calls the **lip** the most common site (true in the older sun-exposure literature), but the most common **intra-oral** site is the **lateral border of the tongue** — know which one the question is asking for.

§5 Herpes simplex — HSV-1 and HSV-2 (HHV-1/2)

> KEY POINTS
> **Vesiculo-bullous lesions** covered in this lecture: acute herpetic gingivostomatitis · recurrent herpes labialis · shingles (§6) · erythema multiforme, pemphigus vulgaris, mucous membrane pemphigoid (§7) · lichen planus (§8). All are **immune-mediated or autoimmune** apart from the frank viral infections.

5.1 Primary herpetic gingivostomatitis

- **Cause**: HSV 1 and 2 → primary herpetic gingivostomatitis *and* herpes labialis
- **Age**: common in children ★**6 months – 6 years**
- **Prodromal / general**: fever · headache · malaise · anorexia · regional lymphadenopathy · sore mouth lesions
- **Intra-oral**: multiple **vesicles** → rupture into ulcers in **all** oral mucosa
- **Course**: self-healing in **10–14 days** → virus stays dormant in the ★**trigeminal ganglion** awaiting reactivation

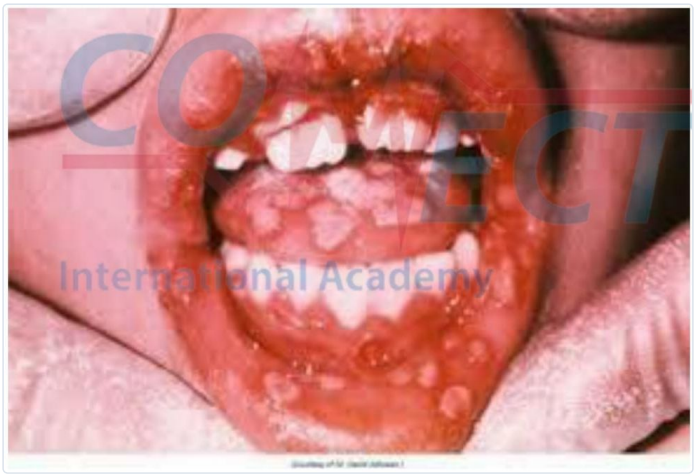

Fig. 7 — Primary herpetic gingivostomatitis: ruptured vesicles across the whole mucosa with fiery marginal gingivitis and crusted lips.

- **Management**: acyclovir in the **prodromal phase**
  - topical *or* systemic acyclovir in severe cases, children under 6 months, immunocompromised
- **Cross-infection**: avoid contact with others
- **Self-inoculation**: avoid touching ulcers → ★**herpetic whitlow**
- **Supportive**: bed rest · fluid intake

5.2 Herpes labialis (cold sores)

- **Mechanism**: **reactivation** of HSV 1 or 2 after the primary infection, triggered by lowered immunity
  - common cold · long sun exposure · immunity disorder
- **Sequence**: prodromal phase → vesicles → rupture into ulcers
- **Treatment**: **topical** acyclovir in the prodromal phase · **systemic** acyclovir if multiple lesions or immunocompromised

Fig. 8 — Recurrent herpes labialis: crop of vesicles at the mucocutaneous junction/perioral skin, the classic reactivation site.

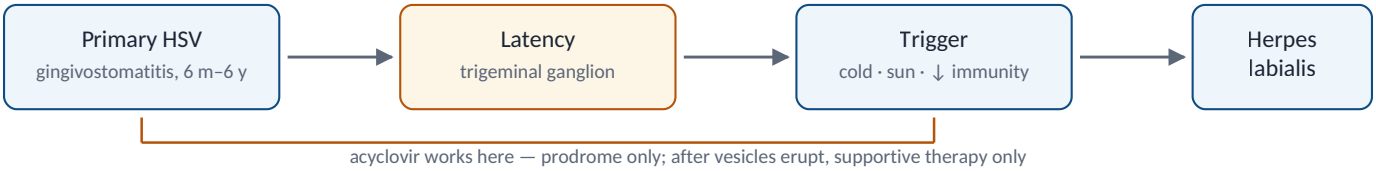

Diagram 1 — HSV life cycle: primary disease → trigeminal ganglion latency → trigger → recurrence, with the acyclovir window.

5.3 Acyclovir protocol

- **Topical, prodromal phase**: cream ★**5 times a day for 5 days**
- **After vesicles erupt**: **supportive therapy only** — bed rest, fluid intake, good nutrition
- **Palliative**: analgesics + antihistamine to control signs and symptoms

- **Systemic acyclovir when**: very young children · immunocompromised · severe cases · unresponsive to topical therapy · ★**lesions persisting >14 days without healing**

> ★ EXAM PEARL
> Acyclovir is a **prodrome drug** — once the vesicles are out it changes nothing, and the answer becomes supportive care. The other favourite discriminator: **primary** disease involves the **gingiva** in a young child; **recurrent** disease sits on the **lip/perioral skin** in anyone.

§6 Varicella zoster virus — HHV-3

6.1 Chickenpox (primary infection)

- **Lesion**: itchy **vesicular cutaneous rash**
- **Age**: children ★**5–9 years**
- **Oral mucosa**: rarely affected
- **Course**: in **1–2 days** all lesions are crusted

6.2 Shingles (reactivation)

- **Who**: commoner in **immunocompromised elderly** patients
- **Distribution**: **unilateral**, confined to the nerve distribution · ★**NEVER crosses the midline**
  - facial/oral lesions arise from areas supplied by branches of the **trigeminal nerve**
- **Sequence**: pre-eruptive pain + redness → painful vesicles → rupture → ulcers + crusted skin wounds → usually clear in **2–4 weeks**
- **Antiviral window**: acyclovir within the ★**first 3 days** reduces the risk of **post-herpetic neuralgia**

> CAUTION
> Eye lesions → refer to an **ophthalmologist**. Ophthalmic-division zoster threatens sight, and the pre-eruptive pain phase is readily mistaken for dental pain — do not extract a tooth into a prodrome.

Fig. 9 — Herpes zoster of the trigeminal nerve: crusting vesicles filling one dermatome and stopping abruptly at the midline.

6.3 Ramsay Hunt syndrome

- **Causative agent**: varicella zoster virus (HHV-3)
- **Nerve**: **facial nerve** → ★**facial palsy + unilateral ulcers along the course of the facial nerve trunk**
- **Complication**: ulcers around the ear may lead to **deafness**
- **Management**: **multidisciplinary** · systemic acyclovir ★**400–800 mg**

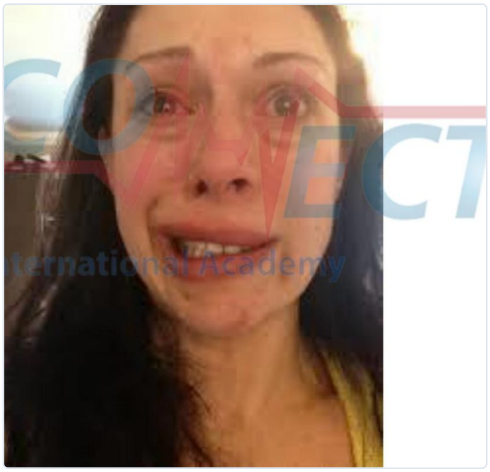

Fig. 10 — Ramsay Hunt syndrome: unilateral lower motor neurone facial palsy — loss of nasolabial fold and drooping commissure on the affected side.

> ★ EXAM PEARL
> Two zoster rules answer most questions: the rash **never crosses the midline**, and the antiviral window is **72 hours**. Note the deck's spelling "Ramzy/Ramsy Hunt" — the examinable spelling is **Ramsay Hunt**, and it is the only one of these that pairs **vesicles with a facial palsy**.

§7 Immune-mediated vesiculo-bullous disease

7.1 Erythema multiforme

- **Pattern**: **recurrent** episodes of acute blistering → rupture in **2–3 days** → painful ulcers
- **Minor form**: oral · eye · genital · skin lesions
- **Oral lesions**: red, painful, **irregular** ulcers on lips or buccal mucosa, tongue, soft palate
- **Skin lesions**: flat rounded macules or papules → typical ★**target or iris form** (well defined and recurrent)
- **Major form**: the same **+ multi-organ dysfunction**
  - bloody crusted lips · eye conjunctivitis · **symblepharon** (adhesion of eyelid to globe)
- **Management of major form**: refer to **GP** and **ophthalmologist** + **steroids**

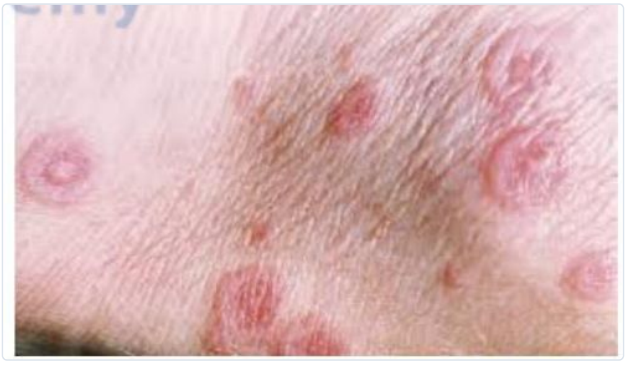

Fig. 11 — Erythema multiforme: target (iris) lesions — concentric rings, well defined, on the skin.

7.2 Stevens–Johnson syndrome

- **Nature**: rare but very serious · usually an **unpredictable adverse reaction to certain medications** · sometimes triggered by infection
- **Course**: flu-like symptoms → red or purple rash spreads → forms blisters → affected skin dies and peels off
- **Care setting**: ★**medical emergency — hospital, often intensive care or a burns unit**
- **Treatment aims**: identify the underlying cause · control symptoms · prevent complications

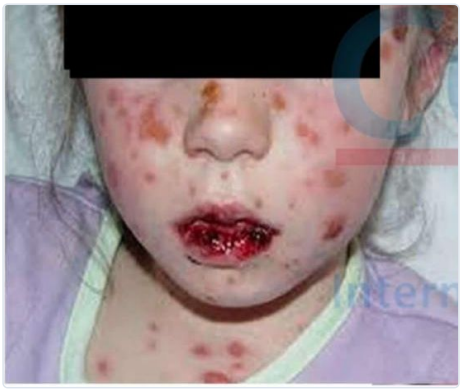

Fig. 12 — Stevens–Johnson syndrome: haemorrhagic crusted lips with a blistering rash — the oral sign that should trigger emergency referral.

> **CAUTION**
> Suspected SJS is not a dental problem to observe — it is an emergency admission. Any patient with bloody crusted lips + a spreading blistering rash + a recently started drug goes to hospital **today**.

7.3 Pemphigus vulgaris and mucous membrane pemphigoid

- **Shared picture**: fluid-filled **bullae** → rupture → **painful erosions**
- **Oral sites**: labial mucosa · palate · tongue
- **Other sites**: genitals · eyes · larynx · pharynx
- **Investigations**: **biopsy** · **direct and indirect immunofluorescence** → shows the **level** of the lesion and basement membrane separation

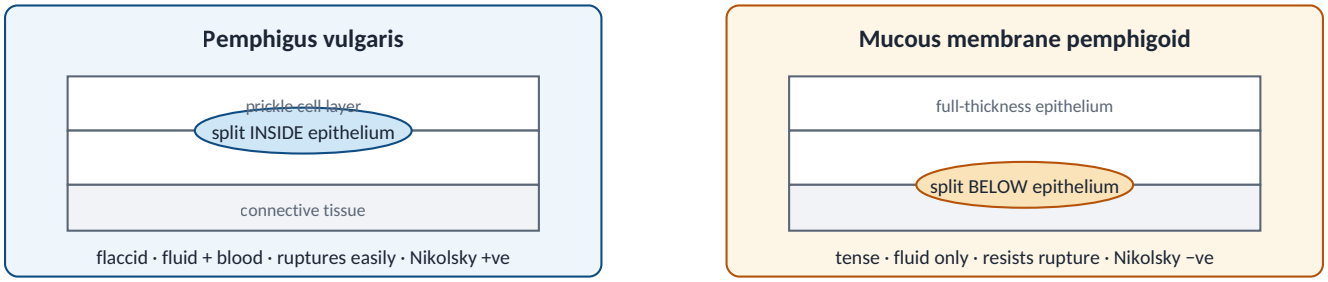

Diagram 2 — The split level decides everything: intraepithelial (PV) gives fragile flaccid bullae; subepithelial (MMP) gives tough tense bullae.

Table 6 — Pemphigus vulgaris vs mucous membrane pemphigoid

	Pemphigus vulgaris (PV)	Mucous membrane pemphigoid (MMP)
Level	**Intraepithelial** — between prickle cell layer and basement membrane	**Subepithelial** — deep under the basement membrane
Bullae	Superficial, **flaccid**, fluid **and blood**, easily ruptured	Deep, **tense**, fluid only, not easily ruptured
Nikolsky's sign	**Positive**	**Negative**
Histopathology	**Acantholysis**, intact basement membrane, intraepithelial lesion	Separation of the basement membrane, subepithelial lesion; no lymphocytic infiltration, no basal cell degeneration
Severity	**Potentially fatal** (see below)	Chronic, mucosa-limited; scarring risk at eye

- **Nikolsky's sign (as taught in the deck)**: pressure applied to a bulla with a finger for **2 minutes** then released → fluid diffuses and the bulla **enlarges**
- **Why PV is fatal**: superficial intraepithelial vesicles rupture easily → ulcers **all over the body**
  - → loss of large amounts of fluid and blood → massive bleeding + dehydration
  - → increased possibility of **superimposed infection**

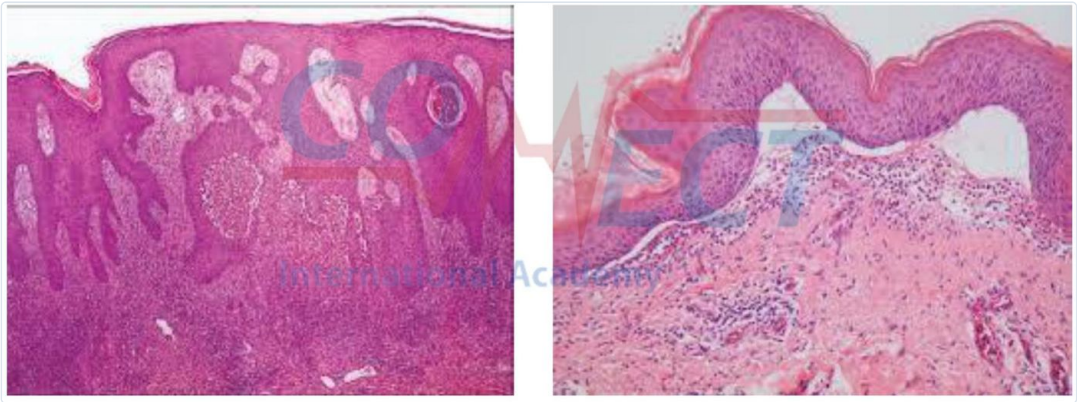

Fig. 13 — Histopathology side by side: **left** PV, acantholytic split *within* the epithelium over an intact basement membrane; **right** MMP, the full-thickness epithelium lifted cleanly off the connective tissue.

★ EXAM PEARL

The standard exam definition of **Nikolsky's sign** is firm **sliding/lateral pressure on clinically normal mucosa or skin** producing an epithelial slough or new blister — not the deck's 2-minute press-and-release on an existing bulla. Either way the answer to "positive Nikolsky" is **pemphigus vulgaris**. Note too that MMP does contain inflammatory cells; the "no lymphocytic infiltrate, no basal cell degeneration" line is really the contrast with **lichen planus** (§8).

§8 Lichen planus, lichenoid reaction and desquamative gingivitis

KEY POINTS

Lichen planus = **bilateral** buccal mucosa, **Wickham's striae**, remission/exacerbation, biopsy-confirmed, **erosive form premalignant** → follow up. Lichenoid reaction = the same picture with a **findable cause** (amalgam, drug, HCV) → remove the cause.

8.1 Lichen planus

- **Nature**: common **chronic inflammatory** disease of skin and oral mucosa · immune mediated
- **Two defining features**:
  - periods of **remission and exacerbation**
  - white papules coalescing into a net-like appearance = ★**Wickham's striae**
- **Six recognised forms**: papular · plaque-like · reticular · ★**erosive (premalignant)** · bullous · atrophic
- **Distribution**: **bilateral** on buccal mucosa
  - intra-oral → buccal mucosa, tongue, gingivae · skin → **flexor surfaces** of extremities
- **Demographics**: middle-aged **females** most affected · ★**F:M 3:2**
- **Possible drug causes**: antihypertensives · antimalarials · oral hypoglycaemics
- **Diagnosis**: confirmed by **biopsy**
- **Histopathology**: hyperkeratosis · hyperparakeratosis · **saw-shaped rete pegs** · **basal cell degeneration** · **lymphocytic infiltrate** (band-like)

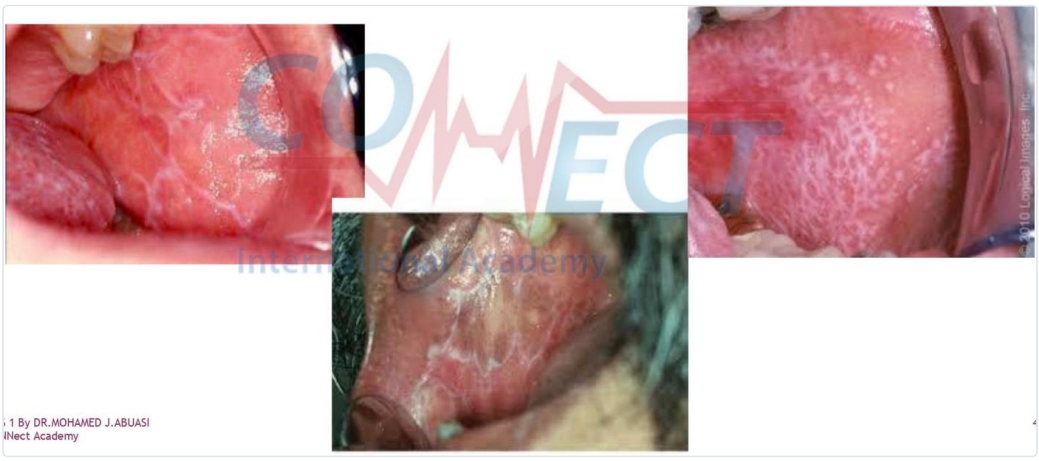

Fig. 14 — Wickham's striae: fine white papules coalescing into a lace-like network on the buccal mucosa.

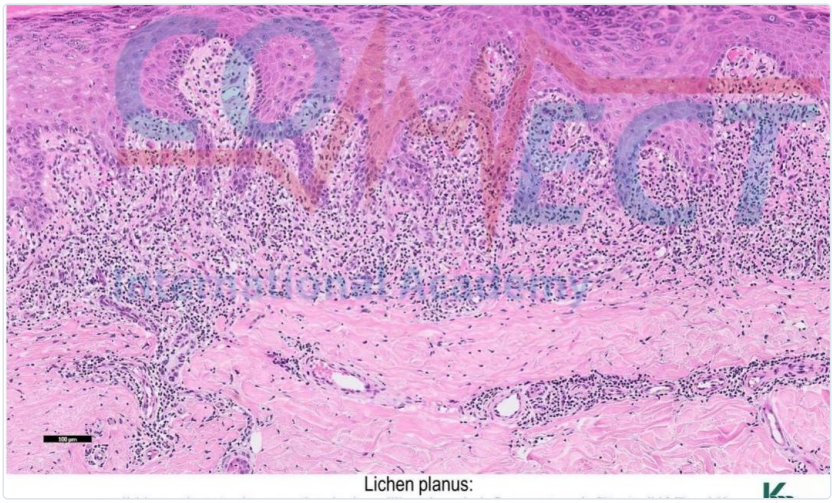

Fig. 15 — Lichen planus (H&E ×10): mild hyperkeratosis, acanthosis and the diagnostic band-like chronic inflammatory infiltrate hugging the basal layer.

- **Management**:
  - asymptomatic lesions → **no treatment**
  - topical or systemic **steroids** according to severity
  - control local factors and any ill-fitting dental fillings
  - control **D.M.**, **HCV** and any suspected drugs
  - **follow up very important** — especially the erosive type and all premalignant types

8.2 Lichenoid reaction

- **Definition**: lesions with **the same clinical and histopathological characteristics** as lichen planus
- **Mechanism**: mostly a **hypersensitivity reaction** — always related to an underlying cause
- **Causes**:
  - dental restorations, mainly ★**amalgam**
  - DM and hypertension · **HCV**
  - drugs → antihypertensives · oral hypoglycaemics · antimalarials · **NSAIDs**
- **Management**: replace the restoration · oral hygiene measures · refer to a specialist to alter the medical treatment · **topical steroids**

8.3 Desquamative gingivitis

- **Appearance**: **full-width** gingivitis with a **fiery red** appearance
- **Diagnostic mismatch**: ★**severity of inflammation does not match the amount of plaque and calculus**
- **Underlying conditions** (in patients who also don't maintain good oral hygiene): **lichen planus** · **pemphigus vulgaris** · **mucous membrane pemphigoid**

Fig. 16 — Desquamative gingivitis: fiery red, full-width gingival inflammation out of all proportion to the visible plaque.

8.4 Treatment protocol with steroids

1. **Topical corticosteroids**: ★**4 times daily for 1 month** · e.g. **kenakort, kenacomp**
2. **Intra-lesional cortisol**: if topical is not effective after **3 weeks** of therapy
   - injection around the lesions with a cortisol vial
   - **submucosal** under the ulcers · **ring block** around the vesicles
3. **Systemic cortisol**: if no response to treatment, and in systemic manifestations

> ★ EXAM PEARL
>
> Bilateral + no identifiable cause = **lichen planus**; unilateral, or hugging an amalgam, or starting after a new drug = **lichenoid reaction** — and the first-line treatment is **removing the cause**, not steroids. The premalignant word belongs to the **erosive** form, which is why follow-up is examinable.