



NOTES

ORAL DISEASE

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Infectious, inflammatory diseases; affect oral cavity, associated structures

RISK FACTORS

- Poor oral hygiene, dehydration, concomitant illness, malnutrition

SIGNS & SYMPTOMS

- Inflammation
 - Redness, swelling, pain, loss of function, warmth
- Infection
 - Fever, malaise, localized pain

DIAGNOSIS

DIAGNOSTIC IMAGING

X-ray

- See individual diseases

CT scan

- Soft tissue inflammation extension

TREATMENT

MEDICATIONS

- Nonsteroidal anti-inflammatory drugs (NSAIDs) for pain
 - For inflammation
- Antibiotics, antifungal medications
 - For infection

APHTHOUS ULCERS

osms.it/aphtous-ulcers

PATHOLOGY & CAUSES

- Painful lesions inside mouth; benign, non-infectious; AKA canker sores

TYPES

Minor

- Small (3–4mm), last 7–10 days, recur 3–4 times/year; if recurrent, > 4 times/year

Major

- Lesions > 1cm, last 10–30 days

Herpetiform

- Coalesce, recur frequently

CAUSES

- Idiopathic; likely multifactorial; may be part of TH1 autoimmune response, hormonal factors influence epithelium thickness, connected to vitamin B₁₂ deficiencies

RISK FACTORS

- Stress, systemic autoimmune disorders (e.g. celiac), nutritional deficiencies, stopping smoking, oral cavity trauma (e.g. biting lips, dentures)

COMPLICATIONS

- Recurrent aphthous stomatitis (Mikulicz ulcers), infection; may interfere with eating/drinking

SIGNS & SYMPTOMS

- Round/oval ulcerations in oral mucosa, white/yellow sharply demarcated center covered with fibrous membrane cap, surrounded by red erythematous margins; yellowish exudate
- Inside of cheeks, lips; under tongue; painful swallowing, if in back of throat

Minor

- Small, mildly painful, annoying, round/oval, disappear within seven days, resolve spontaneously, no scarring; more common on non-keratinized epithelium

Major

- Larger, painful, recur more often, may scar

Herpetiform

- Not herpes virus connected, vesicles coalesce into patches



Figure 39.1 The clinical appearance of aphthous ulcers.

DIAGNOSIS

OTHER DIAGNOSTICS

- Recurrence of ulcers

TREATMENT

MEDICATIONS

- Vitamin B₁₂ supplementation
- Topical analgesics, corticosteroids, sucralfate suspension
- Anti-tumor necrosis factor (TNF)-alpha agents
 - Recalcitrant, recurrent ulcers

OTHER INTERVENTIONS

- Avoid triggers

DENTAL CARIES DISEASE

osms.it/dental-caries

PATHOLOGY & CAUSES

- Odontogenic infections; tooth decay caused by acids produced by bacteria.
- Bacteria → plaque → ↓ pH → demineralization → caries

CAUSES

- *Streptococcus mutans*, *Streptococcus sobrinus*, *Lactobacillus spp.*
 - Metabolically produce acids

RISK FACTORS

- Prolonged bottle use (baby bottle tooth decay), poor oral hygiene, sugar-rich foods, diabetes mellitus (DM), salivary gland disorders (e.g. Sjogren's), medications that decrease salivation

COMPLICATIONS

- Hematogenous spread of bacteria to heart valves, joints, implanted prosthetics
- Spread from enamel to tooth pulp, alveolar bone
- Abscesses
- Soft tissue infections in extraoral perforation
- Deep head, neck infections
- Jaw osteomyelitis
- Tooth loss

SIGNS & SYMPTOMS

- Yellow/black teeth staining, enamel softening; appearance of pits, cracks
- *If severe:* tooth collapse
- *If pulp affected:* dull pain exacerbated by cold, soft food
- *If root caries:* lower, where teeth close together, food difficult to extract; more difficult to diagnose

DIAGNOSIS

DIAGNOSTIC IMAGING

Odontogram (jaw X-ray)

- Examine depth of lesions

CT scan

- If widespread, soft tissue infection

OTHER DIAGNOSTICS

Clinical presentation

- Teeth discoloration, changes

TREATMENT

MEDICATIONS

- Topical/systemic antibiotics

SURGERY

- Extraction of infected material, replacement with fillings

OTHER INTERVENTIONS

- Dietary counselling, hygiene improvement



Figure 39.2 A dental cavity in the tooth of a ten-year-old boy.

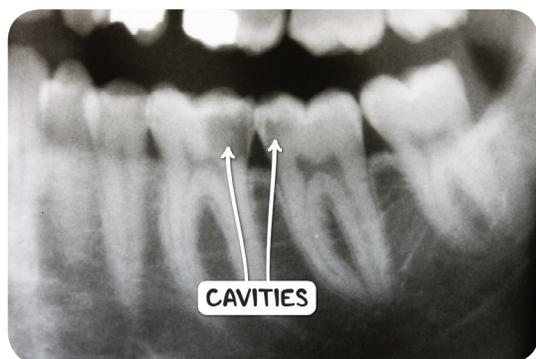


Figure 39.3 An orthopantomogram demonstrating dental cavities of the left mandibular second and third molar teeth.

GINGIVITIS

osms.it/gingivitis

PATHOLOGY & CAUSES

- Type of periodontal disease; inflammation of gums
- Pathogenic bacteria tunnel between microcolonies on tooth to surface in order to bring in steady supply of food → form hard mass (dental calculus) → bacterial plaque formation → enter gingival sulcus → gingivitis
- Immune response delivers blood to damaged tissue → provides nutrients for bacteria → immune response activates osteoclasts → dissolves bone → tooth loosening
- Non-infectious systemic factors → gingival overgrowth, inflammation
 - Hormonal shifts (e.g. during pregnancy)
 - Drug-induced (e.g. phenytoin, calcium channel blockers)
 - Malnutrition-induced (e.g. vitamin C deficiency)
 - Non-plaque-induced (rare, associated with genetics, allergy, trauma)

RISK FACTORS

- Poor dental hygiene, older age

COMPLICATIONS

- Periodontitis, tooth loss, receding gums

SIGNS & SYMPTOMS

- Redness, swelling, bleeding after brushing/flossing
- May be asymptomatic in early infection

DIAGNOSIS

DIAGNOSTIC IMAGING

X-ray

- Evaluate bone level, sulcus becomes deeper as periodontal pocket expands

OTHER DIAGNOSTICS

Physical exam

- Swollen/bleeding gums, probe gingival sulcus to determine depth



Figure 39.4 An individual with a severe case of gingivitis. The gums are swollen and hemorrhagic. There is visible plaque covering the free gingival margin of both maxillary incisors.

TREATMENT

MEDICATIONS

- Antibiotics for severe infections

SURGERY

- Removal of infected tissue if severe

LUDWIG'S ANGINA

osms.it/ludwigs-angina

PATHOLOGY & CAUSES

- Bilateral infection of submandibular space (sublingual, submylohyoid)

CAUSES

- Spread from infection of 2nd/3rd mandibular molars, pericoronitis, parotitis, peritonsillar abscess
- Mandibular fracture, piercings
- Causative agents polymicrobial from mouth flora, dominated by *Streptococcus viridans*; *staphylococci*, *bacteroides* also common

RISK FACTORS

- DM, hypertension, HIV infection, immunosuppression

COMPLICATIONS

- Airway obstruction, mediastinitis, necrotizing cellulitis, sepsis, asphyxia

SIGNS & SYMPTOMS

- Infection
 - Fever, chills, malaise, pain
- Stiff neck, dysphagia, individual leans forward to expand airway, no lymphadenopathy, bilateral, sudden aggressive spread, enlarged tongue, drooling
- Critical symptoms
 - Stridor, cyanosis
- No abscess formation

DIAGNOSIS

DIAGNOSTIC IMAGING

CT scan

- Rule out abscess formation (occurs late in disease)
- Chest CT scan
 - Mediastinitis

LAB RESULTS

- Blood culture

OTHER DIAGNOSTICS

- Ultrasound-guided needle aspiration

TREATMENT**MEDICATIONS**

- Empiric broad-spectrum antibiotics with beta-lactamase activity

SURGERY

- Surgical drainage, if abscess identified on CT scan

OTHER INTERVENTIONS**Airway management**

- Fiberoptic nasal intubation, emergent tracheostomy may be necessary

ORAL CANDIDIASIS

osms.it/oral-candidiasis

PATHOLOGY & CAUSES

- Opportunistic infection of oral mucosal membranes by *Candida* spp. (e.g. *Candida albicans*)
- AKA thrush

TYPES**Pseudomembranous**

- Whitish plaques on oral mucosa (most common); can be scraped off to reveal erythematous surface

Atrophic (denture stomatitis)

- Red lesions without plaques

Hyperplastic (rare)

- Non-scrapable plaques

RISK FACTORS

- Young age, dentures, xerostomia, antibiotics, DM, malnutrition
- Immunosuppression due to corticosteroids, chemotherapy, HIV/AIDS

COMPLICATIONS

- Spread into pharynx, disseminated candidiasis

SIGNS & SYMPTOMS

- May be asymptomatic
- Cottony feeling in mouth; lesions
- Pain/tenderness in oral cavity
- Painful swallowing (odynophagia)
- Decreased sense of taste
- Angular cheilitis

DIAGNOSIS**LAB RESULTS**

- Microbiological analysis of scrapings; Gram stain; KOH preparation; biopsy

TREATMENT

MEDICATIONS

- Topical antifungal agents (e.g. nystatin suspension, clotrimazole troches, systemic fluconazole)



Figure 39.5 Oral candidiasis in a child who had taken antibiotics.

PAROTITIS

osms.it/parotitis

PATHOLOGY & CAUSES

- Parotid gland inflammation
- Salivary stasis → seeding of parotid (Stensen) duct by microorganisms → infection, inflammation

CAUSES

- **Bacterial:** *S. aureus*, most common
- **Viral:** mumps, influenza, coxsackie, Epstein-Barr virus (EBV)
- **Autoinflammatory:** sarcoidosis as part of Mikulicz syndrome

RISK FACTORS

- Surgery, dehydration, salivary gland stones, poor oral hygiene, medications that decrease salivation (e.g. anticholinergic,

sympathomimetics)

COMPLICATIONS

- Spread to deep head, neck structures; septic jugular thrombophlebitis; septic osteomyelitis; sepsis; respiratory obstruction; facial nerve palsy

SIGNS & SYMPTOMS

- Systemic manifestations
 - Fever, chills
- Periauricular, mandibular pain, swelling; trismus, dysphagia; purulent drainage
- Viral
 - No discharge, prodrome followed by swelling lasting 5–10 days

DIAGNOSIS

DIAGNOSTIC IMAGING

- Sample purulent exudate, ultrasound guided needle aspiration; culture, Gram stain

Ultrasound

- Increased blood flow through gland, enlargement, nodules

CT scan

- Extension of inflammation to surrounding tissues

LAB RESULTS

- Complete blood count (CBC)
- Increased amylase without underlying pancreatitis
- Viral shows leukocytosis, increased IgM against mumps



Figure 39.6 The clinical appearance of parotitis of the left parotid gland. There is a marked swelling just anterior to the left ear.

TREATMENT

MEDICATIONS

- Hydration; IV antibiotics
- Vaccination
 - Mumps prevention

PERIODONTITIS

osms.it/periodontitis

PATHOLOGY & CAUSES

- Inflammation, destruction of supporting structures around teeth, wasting of bone
- Dysbiosis (disturbed bacterial symbiosis) more extreme than in gingivitis
- Orange-complex of bacteria (*Fusobacterium nucleatum*, *Prevotella intermedia*), red-complex of bacteria (*Tannerella forsythia*, *Treponema denticola*, *Porphyromonas gingivalis*) → immune response → more blood flow to damaged tissue → provides nutrients for bacteria → more damage to gingiva, periodontal ligament → activated osteoclasts in bone
 - tooth loosening
- Severity based on ligament loss
- *Porphyromonas gingivalis* impairs immune cells, kills bacteria → pathogenic bacteria overgrow
- Necrotizing ulcerative periodontitis (NUP)
 - Extreme loss of periodontal attachment, alveolar bone; associated with immunosuppression (e.g. HIV/AIDS; chemotherapy, severe malnutrition); may be associated with enteric bacteria, yeast

CAUSES

- Poor oral hygiene; red-, orange-complex bacteria

RISK FACTORS

- DM, smoking, Ehler–Danlos syndrome

COMPLICATIONS

- Tooth loss, infection spread to soft tissues of head, neck, sinusitis; hematogenous dissemination to heart valves (prosthetic/native), joints, etc.

SIGNS & SYMPTOMS

- Redness, swelling, tender to palpation
- Halitosis
- Bleeding during teeth brushing
- Teeth loosening
- Periodontal pockets widen

DIAGNOSIS

DIAGNOSTIC IMAGING

Panoramic dental X-ray

- Bone loss around tooth

OTHER DIAGNOSTICS

- Clinical exam
 - Probe teeth pockets, test for bleeding, depth

TREATMENT

MEDICATIONS

- Systemic antibiotics (if severe)

SURGERY

- Removal of infected tissue (if severe)

OTHER INTERVENTIONS

- Prevent plaque formation
 - Daily brushing, flossing; antimicrobial agents (e.g. mouthwash)
- Scaling, root planing
 - Remove plaque
- Topical fluoride

SIALADENITIS

osms.it/sialadenitis

PATHOLOGY & CAUSES

- Inflammation of salivary glands
 - Parotid (most common), sublingual, submandibular; unilateral
- Decreased flow of saliva → deposits settle in walls of salivary duct → duct blocked → flow of saliva slowed → deposits of calcium, phosphorous, etc. precipitate → form small concretions (microsialoliths) → grow into sialoliths → stones block duct → bacteria moves from mouth up, around blockage, into salivary duct →

inflammation, tissue swelling

CAUSES

- Bacterial: *Staphylococcus aureus* (most common), *Streptococcus viridans*, *Haemophilus influenzae*
- Viral: mumps, HIV

RISK FACTORS

- Decreased salivary flow (dehydration, illness, anticholinergic medications, Sjogren's syndrome)
- Risk increases with age



Figure 39.7 An individual holding their own salivary duct stone following surgical removal. Salivary duct stones predispose individuals to sialadenitis.

SIGNS & SYMPTOMS

- Acute sialadenitis
 - Fever, chills, abscess formation
 - Pain, swelling, redness of skin overlying affected gland
 - Less saliva → dry mouth → bad taste (pus leaking out of affected duct)
 - Severe: painful to open mouth
- Chronic sialadenitis
 - Less painful, gland enlarges following meals, no overlying redness of the skin
 - Associated with conditions linked to chronic decreased salivary flow (e.g. Sjogren's syndrome), due to inflammation, salivary duct fibrosis, altering glandular tissue, composition of saliva

DIAGNOSIS

DIAGNOSTIC IMAGING

Ultrasound

- Abscess, salivary stone, tumor

LAB RESULTS

- Lab culture of pus
 - Gentle compression of gland

OTHER DIAGNOSTICS

- Clinical presentation

TREATMENT

MEDICATIONS

- Antibiotics

SURGERY

- Surgical gland removal
 - If disease recurrent

OTHER INTERVENTIONS

- Hydration, warm compress, glandular massage, sialogogues

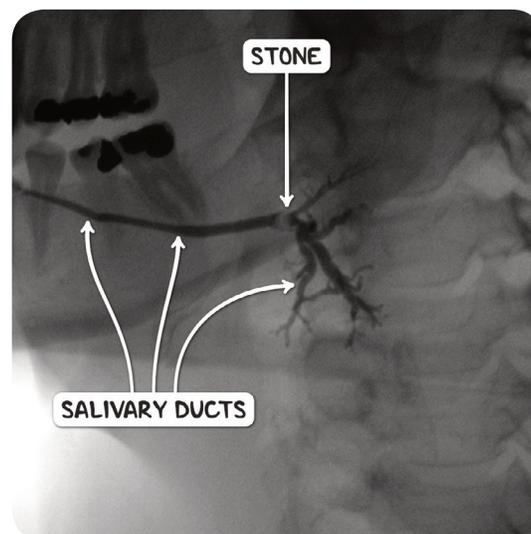


Figure 39.8 A submandibular sialogram demonstrating a salivary duct stone; a risk factor for sialadenitis.

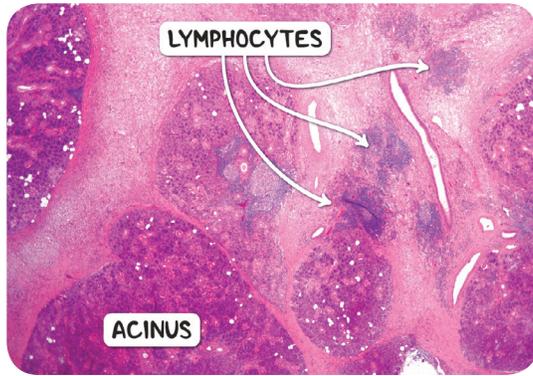


Figure 39.9 The histological appearance of sialadenitis at low power. The acini are surrounded by dense fibrosis and display patchy lymphocytic infiltrates.