Oral Pathology Clinical Review

4 Credit Hours (4 CEs)

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With updates from Oral Cancer Foundation from 2016

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Objectives

Upon completion of this course, the student will:

• Remember major examples of oral pathology included in the course survey.
• Recognize the major characteristics of the types of oral pathology as presented.
• Understand major aspects of differential diagnosis (DDX).
• Know major aspects of treatment, management, and prognosis.
• Improve diagnostic knowledge of radiographic interpretation.
• Know specific points to communicate to patients about prognosis.

Introduction

The practice of dentistry is an application of art and science. In everyday practice, the dental practitioner reviews many cases of suspected pathology, and while some case presentations are very common, others are not common. This course is a refresher course of fairly common oral pathology conditions. A very strong background in clinical dental practice is assumed for this course. The course is most suitable for dentists and dental hygienists who want a good review with clinical case presentations and radiographs to support the text. However, the course would be a useful summary and review for all members of the dental team who want to familiarize themselves with an overview oral pathology in clinical practice. This course is ADA CERP approved for 4 hours of continuing education credit, and fulfills credit hours toward license renewal. Individuals must check with their state licensing boards for exact requirements for dental license renewal.

2016 Statistics on Worldwide Occurrence

Citing the Oral Cancer Foundation:

Oral cancers are part of a group of cancers commonly referred to as head and neck cancers, and of all head and neck cancers, they comprise about 85% of that category. Brain cancer is a cancer category unto itself and is not included in the head and neck cancer group.

Historically the death rate associated with this cancer is particularly high not because it is hard to discover or diagnose, but due to the cancer being routinely discovered late in its development. Today, (2016) that statement is still true, as there is not a comprehensive program in the US to opportunistically screen for the disease, and without that; late stage discovery is more common. Another obstacle to early discovery (and resulting better outcomes) is the advent of a virus, HPV16, contributing more to the incidence rate of oral cancers, particularly in the posterior part of the mouth (the oropharynx, the tonsils, the base of tongue areas) which many times does not produce visible lesions or
discolorations that have historically been the early warning signs of the disease process.

Often oral cancer is only discovered when the cancer has metastasized to another location, most likely the lymph nodes of the neck. Prognosis at this stage of discovery is significantly worse than when it is caught in a localized intra oral area. Besides the metastasis, at these later stages, the primary tumor has had time to invade deep into local structures.

Oral cancer is particularly dangerous because in its early stages it may not be noticed by the patient, as it can frequently prosper without producing pain or symptoms they might readily recognize, and because it has a high risk of producing second, primary tumors. This means that patients who survive a first encounter with the disease, have up to a 20 times higher risk of developing a second cancer. This heightened risk factor can last for 5 to 10 years after the first occurrence. There are several types of oral cancers, but around 90% are squamous cell carcinomas.

Dental Professionals are often times the first line in defense, in simply recognizing abnormalities in a patient. Clearly, the earlier the detection, the higher the rate of survival for patients.

Oral Pathology Clinical Review

1. Fibroma

Fibroma Characteristics:

• “Irritation fibroma” “traumatic fibroma”
• Benign
• Reactive vs. Neoplastic
  o mature pyogenic granuloma?
• Buccal mucosa, labial mucosa, tongue, gingival
• Variant on lingual surface canine area of mandible
  o --Retrocuspid papilla
• Pink
- +/- pigmentation
- +/- hyperkeratosis
  - Sessile or pedunculated

Fibroma of left labial commissure in a 59 y/o man (Medscape, 2013)

**Differential Diagnosis (DDX)**

- Fibroma
- Neurofibroma
- Pyogenic granuloma
- Peripheral ossifying fibroma
- Benign mesenchymal neoplasm

**Management / Prognosis**

- Excision
- Removal of causative agents
- Recurrence rare

2. **Pyogenic Granuloma**

**Pyogenic Granuloma Characteristics**

- Tumor-like growth
- Non-neoplastic, typically reactive
- Response to irritation, trauma
  - -- antecedent history
  - -- oral hygiene
• --pregnancy
  • Gingiva, lips, tongue, buccal mucosa
  • Smooth or lobulated
  • Pink, red, purple
  • Ulceration
  • Vascular
  • --bleeding

**Differential Diagnosis (DDX)**

• Pyogenic granuloma
• Peripheral ossifying fibroma
• Peripheral giant cell granuloma
• Lobular capillary hemangioma
• Epulis granulomatosa
• Peripheral odontogenic neoplasm

![Pyogenic Granuloma Histology](image)

Typical appearance of a pyogenic granuloma involving the buccal gingiva of teeth numbers 20 and 21. Note the extreme vascularity. (Medscape, 2013)

**Diagnostic Aids**

• Radiograph
• Periodontal probing
• Vitality testing

**Management / Prognosis**

• Excision
• Excision to periosteum (gingival lesions)
• Removal of irritative sources
• Recurrence possible (inform patient)
• Pregnancy: defer until delivery if possible (recurrence common)

3. Peripheral Ossifying Fibroma

Peripheral Ossifying Fibroma Characteristics

- Common gingival growth
- Reactive
- Maturing pyogenic granuloma (?)
- Sessile or pedunculated
- Red to pink
- Ulceration
- Mineralization
  - bone, cementum, dystrophic calcifications
  - osteoid to mature lamellar bone

Differential Diagnosis

- Pyogenic granuloma
- Peripheral ossifying fibroma
- Peripheral giant cell granuloma
- Lobular capillary hemangioma
- Peripheral odontogenic fibroma
Management / Prognosis

- Excision
- Excision to periosteum (gingival lesions)
- Removal of irritative sources
- Recurrence 8-16% (inform patient)

4. PERIPHERAL GIANT CELL GRANULOMA

Peripheral Giant Cell Granuloma Characteristics

- aka ‘peripheral giant cell eupulis’
- Reactive
- Gingiva, edentulous ridge
- Red to re-blue ‘dusky’ mass
- Similar in appearance to pyogenic granuloma
- Cupping resorption of underlying bone
- Multi-nucleated giant cells
• Mitoses
• Hemorrhage
  o --hemosiderin
• Ulceration

DDX

• Pyogenic granuloma
• Peripheral giant cell granuloma
• Central giant cell granuloma
  o hyperparathyroidism
  o renal osteodystrophy
  o cherubism

Management and Prognosis

• Excision
• Excision to periosteum (gingival lesions)
• Removal of irritative sources
• Recurrence ~10% (inform patient)
• Consider ruling out:
  o Central giant cell granuloma
  o hyperparathyroidism

5. Gingival Overgrowth

Associations

• Medications
  o Calcium channel blockers (i.e. Nifedipine, Amlodipine)
  o Dilantin
  o Cyclosporine
  o Cyclophosphamide
• Hereditary fibromatoses
• Hyperplastic gingivitis
Also consider plasma cell gingivitis (cinnamon)
  • Leukemic infiltrate

Management
  • Alter medication if possible
  • Remove causative agents
  • Remove and avoid irritants (cinnamon)
  • Genetic counseling
  • Management of malignancy (leukemia)

6. Gingival Cyst

Gingival Cyst Characteristics
  • Developmental
  • Usually mandibular anterior / bicuspidd
  • Usually facial gingiva
  • Painless, dome-like swelling

DDX
  • Lateral periodontal cyst
  • Epithelial inclusion cyst

Management and Prognosis
  • Excision
  • Recurrence rare
7. Benign Migratory Glossitis

Benign Migratory Glossitis Characteristics

- aka “geographic tongue”, erythema migrans
- Greater incidence in females
- Unknown pathogenesis
  - may be associated with psoriasis
- Well demarcated zones of erythema
- Atrophy of filiform papillae
- Yellow-white serpentine border
- Patient describes healing, then migration
- Fissured tongue

DDX

- Benign migratory Glossitis / erythema migrans
- Burning tongue / burning mouth
- Nutritional deficiencies
- Radiation – chemotherapy stomatitis

Management and prognosis

- No treatment generally required
- Patient reassurance
- Avoidance of irritating agents
- Topical steroids
- Fluocinonide (Lidex) 0.05% gel, 30 gm tube, applied 2x/day if symptomatic
8. (Pseudomembranous) Candidiasis

Pseudomembranous Candidiasis Characteristics

- Considerations
  - oral inhaled steroids
  - systemic antibiotics
  - immune suppression
- Diabetes mellitus
- Cytology / histology
- Infectious
  - candida species
  - normal oral flora
  - Cytologic smear using periodic acid Schiff (PAS)
  - candidal hyphae

Forms

- Pseudomembranous
  - rubs off, leaving erythematous base
- Erythematous
  - red, burning sensation
- Median rhomboid Glossitis
  - midline tongue, red, papillary atrophy
- Angular cheilitis
  - denture wearers, reduced VDA
- Hyperplastic
  - white, corrugated, does not rub off
- Others
DDX

• Varies according to type
• Frictional irritation / hyperkeratosis
• Squamous cell carcinoma
• Materia alba **ck this

Anti-fungal Strategies

Systemic

• Nizoral (ketoconazole 200mg) 200-400mg/ day
  o metabolized in liver, take with food, better absorption with acidic pH
• Diflucan (fluconazole 100mg/200mg) 100-200 mg/ day
  o metabolized in kidney, food elective

Topical

• Mycelex (clotrimazole 10 mg), dissolve 1 troche 5x/day
• Mycostatin (nystatin)
  o suspension, swish and swallow 500,000 U, 3 tsp/day
  o vaginal troche, dissolves 100,000 U, 3-5x/day
  o Mycolog cream for angular cheilitis
• Mycostatin Powder 100,000U/gm, for dentures
  o Sporanox (itraconazole 10mg/ mL) 10mL 2x/day
  o Fungizone (amphotericin 100mg/ mL) 1 mL 4x/day
  o Oravig (myconazole 50mg tablet) 1x/day 14 days

9. Apthous Ulcer

Types

• Pathogenesis uncertain
  o T-cell mediated
  o Immunodysregulation
Variants

Minor
- Non keratinized mucosa
- Small (3-10mm) fibrinopurulent membrane
- 1-5 lesions
- Fewest recurrences

Major
- Larger (1-3cm)
- Longest duration
- Labial, soft palate, tonsillar, oropharyngeal
- Scarring

Herpetiform
- Multiple lesions (up to 100)
- 1-3mm
- Lesions coalesce
- Recurrences closely spaced

Corticosteroids for Apthous Ulcers

Systemic
- Prednisone = Tapering Dose:
  - 25mg / day x 15 days
  - 12.5 mg / day x 15 days
  - 6.25 mg / day x 15 days
  - 6.25 mg / every other day
- Medrol DosePak as directed

Topical
- Lidex (fluocinonide 0.05%) Apply 3x / daily
- Triamcinolone 0.01% susp Rinse 1 tsp 4 x / day, hold 3 mins, expectorate, NPO x 30 mins
- Kenalog in Orabase Apply 3x / day
10. HERPETIC STOMATIS

Herpes Simplex Characteristics

Human herpesviruses #1-8

HSV1, HSV2, V2V, EBV, CMV, HHV-6/7, HHV-8

HSV1 = spread by saliva

Primary Infection / Primary herpetic gingivostomatis

- HSV-1
- Age range 6 mo – 5 year
- Lymphadenopathy, chills/ fever, nausea, vomiting, anorexia, irritability, oral lesions, pharyngotonsillitis
- Vesicles which rupture
- Gingival edema, erythema, enlargement
- Self-innoculation
- Contagious (hand hygiene)

Secondary Infection

- Vermillion border / skin of lips
- Herpes labalis
- Prodrome
- Vesicles which rupture and crust
- Herpetic whitlow

Latency

Antivirals for Herpetic Lesions

Topical

- Zovirax (acyclovir 5% ointment) Apply q3hr x 7 days
- Denavir (penciclovir 1%) Apply q2hr x 4 days
Other agents:

- Abreva (docosanol 10% cream)  Apply 5x / day until resolution
- Dyclonine (0.5-1.0%)   Apply prn

Systemic

- Zovirax (acyclovir 200/400/800mg)
- Valtrex (valacyclovir 500mg)
- Famvir (famciclovir 125/250/500mg)

11. Herpes Zoster (Shingles)

Herpes Zoster Characteristics

- VZV (HHV-3)
- Latency in sensory nerves (CN-V)

3 phases:

1. Prodome (pain, itching, burning)
2. Acute:
   a. Exanthem / rash along dermatomes
   b. Vesicles
   c. Scarring, pigmentary changes
   d. Ocular involvement
   e. Facial paralysis / auditory loss  (RAMSAY HUNT)
3. Chronic (post-herpetic neuralgia)
Antivirals for Herpes Zoster

Systemic

- Famciclovir
- Valacyclovir
- Acyclovir
- Prednisone

Topical

- Zostrix (capsaisin)
- Lidocaine patch

12. Lichen Planus

Lichen Planus Characteristics

- Dermatologic disease
- Variety of causes:
  - T-cell mediated
  - medication induced
  - foreign body
  - stress / anxiety
- Female predominance
- Skin lesions
  - purple, pruritic, polygonal
  - lace-like striations (Wickham's striate)
  - dysplastic nails
- Oral lesions
  - reticular form
  - erosive form
• Hyperkeratosis
• Acanthosis (epithelial thickening)
• “Sawtooth” rete ridges
• Degeneration of basal layers
• Band-like lymphocytic infiltrate

DDX
• Lichen planus
• Lichenoid drug / amalgam reaction
• Graft versus host disease
• Oral mucosal cinnamon reaction

Management and Prognosis
• Reticular form usually requires no treatment
• Erosive forms use topical corticosteroid
  o -- Fluocinonide (Lidex)
  o -- Clobetasol (Temoval)
  o -- Steroids may be compounded with OraBase or methylcellulose

13. Pemphigus Vulgaris

Pemphigus Vulgaris Characteristics
• Blistering disease
• Autoimmune reaction
  o desmosomes
• Intra-epithelial split
• Other etiologies
  o medication induced
  o paraneoplastic pemphigus
  o familial forms (Hailey-Hailey)
• Often manifest first intraorally
  o precedes skin lesions up to 1 yr
• Soreness, erosions, ulcerations
  o desquamative gingivitis
• Vesicles, bullae rupture quickly
• Nikolsky sign
  o rubbing / pressure to mucosa induces blisters
• Peri-lesional tissue important
• Separation between cells
  o intraepithelial clefting
• Acantholysis (lysis between the cells)
  o desmosomal attachments lost

Management and Prognosis
• Before treatment, cases were routinely fatal
• With treatment, cases are less commonly fatal
• Usually systemic corticosteroids and other immune suppressive agents required
• Manage in consultation with physician
• Prednisone high initial dose (40-80mg daily) tapered over several weeks once patient responds.

14. Pemphigoid

Pemphigoid Characteristics
(benign mucous membrane pemphigoid, cictricial pemphigoid)
• Most common auto-immune blistering disorder
• Autoantibodies vs. basement membrane
• Usually older patients (60-80 y/o)
• Pruritis
• Multiple tense skin bullae
  o rupture / heal w/o scarring
• Oral uncommon
  o bullae
  o shallow ulcerations
• Ocular involvement
  o symblepharon (adhesions)
  o cicatrix (scarring)
  o blindness
• Peri-lesional tissue important
• Separation at basement membrane
  o subepithelial clefting
• Acute / chronic inflammation

Management and Prognosis
• Refer to ophthalmologist (important!)
• For oral mucosal lesions, start with topical steroids

Medications for Oral Mucosal Diseases

Systemic
• Prednisone 40-80 mg / day
• Prednisone 40 mg / 30 mg (am/pm)
• Dapsone 50-300 mg / day
• Doxycycline 20 mg 2x / day
• Niacinamide 0.5-2.0 mg daily (w/TCNs)

Topical
• Lidex
• Temovate
• Ultravate
• Dexamethasone
• Triamcinolone
15. Benign Mixed Turmor (pleomorphic adenoma)

Benign Mixed Turmor (pleomorphic adenoma) Characteristics

- Most common salivary neoplasm
- Parotid gland most common site
- Upper lip, buccal mucosa, posterior palate
- Painless, slow growing mass
- Young to middle age
- More females than male
  - Most salivary neoplasms show female predilection
- Circumscribed to encapsulated
- 3 major elements:
  1. Glandular epithelium (ducts)
  2. Myoepithelial cells
  3. Hyalinization
- Other elements
  - Keratinizing squamous cells
  - Mucous producing cells
  - Chondromyxoid (loose cartilaginous) background

DDX

- Pleomorphic adenoma
  - Other benign salivary neoplasms
- Malignant salivary neoplasms
  - Mucoepidermoid carcinoma
- Lymphoma

Management and Prognosis

- Excision
- For superficial parotid: superficial parotidectomy
- For deep parotid or other gland: excision of entire gland sparing facial nerve
- Cure rate = 95%
• Recurrence possible, especially more myxoid / genetainous tumors (may result in tumor seeding during surgery)
• Carcinoma – mixed tumor possible

16. Mucoepidermoid Carcinoma

Mucoepidermoid Carcinoma Characteristics

• Most common salivary malignancy
• Considered a low grade malignancy
• Parotid, palate
• Lower lip, floor of mouth, tongue, retromolar pad
• Mass
• Pain
• Facial palsy
• Asymptomatic swelling
  o fluctuance
  o blue / red color (clinically mistaken for mucocele)
• Cystic spaces
• Mucous-producing cells
• Squamous elements
• May see keratinization

DDX

• Mucoepidermoid carcinoma
• Squamous cell carcinoma
• Mucocele
• Salivary duct cyst
Management and Prognosis

- Determined by histologic grade and tumor stage
- Subtotal / total parotidectomy +/- facial nerve sparing
- Total excision of other glands with margin of normal tissue for minor glands
- Radical neck dissection for invasive / high grade tumors
- Post-operative radiation therapy for high-grade, invasive, or incompletely / non-excisable tumors
- 90-98% cure for low grade tumors; 30-54% for high grade tumors

17. Mucocele / Ranula

Mucocele / Ranula (=Mucocele on floor of mouth) Characteristics

- Mucous retention
  - sometimes called 'salivary duct cyst'
- Mucous extravasation
  - mucous escapes from ruptured duct
- Dome shaped mucosal swelling
- Flutuant to firm
- Recurrence / association with eating
- Sialoliths / sialolithiasis (salivary stones)
- Spilled mucin
- Granulation tissue
- Inflammation, foamy macrophages
- Chronic inflammation, fibrosis
- Sialoadenitis (inflamed salivary glands)

DDX

- Mucocele
- Salivary duct cyst
- Dermoid cyst

Management and Prognosis

- Some mucoceles / ranulas rupture and self-heal
- Excision usually required
• Remove adjacent glands (total sublingual or submandibular) to minimize recurrence
• Prognosis excellent, other than recurrence

18. Dermoid Cyst

Dermoid Cyst Characteristics

• Developmental
• Benign cystic form or teratoma
• Commonly midline floor of mouth
• Histology
  o squamous cell epithelium
  o keratin
  o adnexal (skin) structures in the wall: sebaceous, hair follicles, sweat glands
• Histologic variation:
  o Epidermoid (1 germ layer)
  o Dermoid (2 germ layers)
  o Teratoma (3 germ layers)

DDX

• Dermoid cyst
• Thyroglossal duct cyst

Management and Prognosis

• Excision
• Intraoral approach if above mylohyoid
• Extraoral approach if below mylohyoid
• Recurrence uncommon
19. Labial Melanotic Macule

Labial Melanotic Macule Characteristics

- Benign
- Flat brown mucosal discoloration
- Increased melanin deposition
- Increased melanocytes
- Not related to sun exposure
- Solitary, well-demarcated
- Tan to dark brown (blue, black)
- Non-enlarging

DDX

- Melanotic macule
- Amalgam tattoo
- Rule out the following features of melanoma: A B C D E:
  - A  Asymmetry
  - B  Borders irregular
  - C  Color variation
  - D  Diameter > 6mm
  - E  Evolution

Management and Prognosis

- Benign lesion
- Biopsy to rule out melanoma
- Often no further treatment required unless cosmetic
20. Racial Pigmentation

Racial Pigmentation / Management and Prognosis

- No treatment required

21. Minocycline Staining

Minocycline Staining Characteristics / DDX

- Medication-induced pigmentation
  - anti-malarials (cloroquinine et al.)
  - antibiotics (tetracyclines, et al.)
  - tranquilizers (chlorpromazine)
- Racial pigmentation
- Addison’s disease
22. Medication Induced Pigmentation

Management and Prognosis

- No treatment required
- Removal or alteration of medication may result in long term partial or complete resolution of pigment
- Consult physician before considering alteration of medication

23. Amelogenesis Imperfecta

Amelogenesis Imperfecta Characteristics

- Defect in enamel development
- Complex multiple forms:
  - hypoplastic
  - hypocalcification
  - hypomaturatation
  - association with taurodontism
- Complete genotypic and phenotypic profile
- Complex classification
Management and Prognosis

- Treatment focused on dental rehabilitation
- Caries control
- Management of loss of vertical dimension
- Fixed or removable dental prosthesis

24. Opalescent Teeth

Opalescent Teeth Characteristics

- Rule out dentinogenesis imperfecta
- Rule out osteogenesis imperfecta

Dentinogenesis Imperfecta

- Defect in dentin and pulp development
- Multiple forms
  - dentinogenesis imperfect
  - dentin dysplasia
  - opalescent teeth
  - pulp malformations
  - root malformations
Management and Prognosis

- Treatment focused on dental rehabilitation
- Caries control
- Teeth are poor candidates for fixed prostheses (poor dentin characteristics)
- Removable / complete dental prostheses often required
- Pulpal pathology common, endodontic therapy is challenging (pulp canal alteration or obliteration)

25. Buccal Bifurcation Cyst

Buccal Bifurcation Cyst (Inflammatory Buccal Cyst) Characteristics

- Inflammatory odontogenic cyst
- Locations:
  - buccal aspect, mandibular first molar
  - distal buccal, third molars (paradental cyst)
- Associations:
  - cervical enamel extensions
  - eruption-associated inflammation
- Radiographic:
  - unilocular radiolucency
  - buccal bifurcation and roots
  - occlusal radiograph: tipping of root apices toward lingual cortex
DDX

- Buccal bifurcation cyst
- Inflammatory cyst (periodontal, endodontic)
- Other odontogenic cyst or tumor

Management and Prognosis

- Debridement – treat as a periodontal pathology
- Removal of cervical enamel projections and enamel pearls / beware of pulp horns
- Extraction usually not necessary

26. Dentigerous (Follicular) Cyst

Dentigerous (Follicular) Cyst Characteristics

- Developmental
  - most common developmental odontogenic cyst
- Associated with follicle / crown of developing tooth
  - usually unerupted third molars, canines
- Radiographic
  - unilocular radiolucency
  - crown of developing tooth
  - well defined sclerotic border

DDX

- Dentigerous cyst
- Glandular odontogenic cyst
- ameloblastoma
- Odontogenic keratocyst

Management and Prognosis

- Careful cyst enucleation with extraction of tooth
- Submit entire cyst for evaluation
Tooth may be left if eruption anticipated
- Marsupialization / decompression of large cysts may be considered
- Recurrence rare with adequate enucleation
- Malignant transformation and development of odontogenic tumors are rare

27. **Ameloblastoma**

**Ameloblastoma Characteristics**

- Most common odontogenic tumor
- Odontogenic epithelial origin
- Benign but aggressive
- 3 variants
  - conventional solid / multi-cystic (most common type)
  - unicystic
  - 3. peripheral / extraosseous
  - Mandible, usually molar-ramus
  - Painless swelling and expansion
- Radiographic
  - --multiocular (or uniocular)
  - --“soap bubble” or “honeycomb” appearance on radiograph
  - --associated with unerupted tooth

**Histology (follicular or variant)**
• Islands of odontogenic epithelium
• Mature fibrous stroma
• Tall columnar cells
• Palisading (nuclei line up)
• Reverse polarity (nuclei away from basement membrane)
• Stellate reticulum (loose cellular architecture in the center of the nests / islands of epithelium.

DDX
• Ameloblastoma
• Dentigerous cyst
• Odontogenic keratocyst
• Ameloblastic fibroma / fibro-odontoma (child)
• Central giant cell granuloma

Management and Prognosis
• Wide excision usually recommended: 1-1.5 cm margin

Recurrence
• Enucleation: 50-90% recurrence
• Excision: 15%

28. Odontogenic Keratocyst (Odontogenic Tumor)
Odontogenic Keratocyst Characteristics

- Posterior body / ramus of mandible

Radiology

- well defined uni / multilocular radiolucency
- defined borders
- unerupted tooth (25-40%)

Histology

- Clear/ cheesy fluid within cyst lumen
- Epithelium 6-8 cell thickness
- Flat epithelium, lack rete ridges
- Wavy parakeratosis
- Palisaded basal layer
- Inflammation may mask features

DDX (often based on location)

- Dentigerous cyst (with unerupted tooth)
- Radicular cyst (at root apex)
- Residual cyst (in site of extracted tooth)
- Lateral periodontal cyst (along side of root)
- Nasopalatine duct cyst (mid-line, anterior palate)
- Glandular odontogenic cyst (often in anterior mandible)
- Odontogenic keratocyst (any location)
- Ameloblastoma (any location but usually posterior)
- Consider nevoid basal cell carcinoma syndrome (Gorlin Syndrome) if there are multiple cysts

Management and Prognosis

- Enucleation, or enucleation with ostectomy
- En bloc or wide excision
- Recurrence
  - --enucleation: 5-62% in numerous case series
• --excision: less likely to recur

29. Stafne Defect

Stafne Defect Characteristics
(static bone cyst / lingual mandibular salivary gland depression)

• Developmental
• Focal concavity, lingual mandible
  o usually posterior near angle
  o may be seen anterior
• Submandibular, sublingual gland
• Asymptomatic radiolucency
• Well-circumscribed, sclerotic border
• Usually diagnosed clinically / radiographically
  o biopsy shows salivary gland tissue or muscle, connective tissue, fat, lymphoid elements

Management and Prognosis

• No treatment required if radiologic diagnosis is likely / certain
• Usually static

30. Periapical Cemento-Osseous Dysplasia (PCOD)
Periapical Cemento-Osseous Dysplasia (PCOD) Characteristics

- Benign fibro-osseous lesion
- focal
- periapical
- florid
- PCOD:
  - anterior mandible
  - usually solitary
  - Black > White, Female > Male, 30-50 y/o
  - vital teeth
- Radiographic:
  - Early: radiolucent, mature / opacify over time
  - Late: radiopaque, narrow radiolucent rim
- Diagnostic tests suggested:
  - vitality testing
  - serial radiographs

31. Idiopathic Osteosclerosis

![Image of dental X-rays]

Idiopathic Osteosclerosis Characteristics

- Focal area of increased radio-density
- Asymptomatic
- No expansion
- 90% mandible / first molar area
- Usually solitary
  - multiple lesions: suspicious for osteomas / rule out Gardner Syndrome
- Well-defined radiopaque mass
- Usually associated with root apex
- Dense lamellar bone

DDX

- Condensing osteitis (focal chronic sclerosing osteomyelitis)
• Osteoma
• sialolith

Miscellaneous

• Condensing Osteitis
  o association with non-vital teeth / pulpitis
  o premolar, molar
• Osteomyelitis
  o usually mandible
  o immuno-compromised considered
  o malignancies considered

32. Bisphosphonate Related Osteonecrosis of the Jaw (BRONJ)

**BRONJ**

Bisphosphonates are a class of drugs that prevent the loss of bone mass. High-potency intravenous bisphosphonates have been shown to modify the progression of malignant bone disease in several forms of cancer, especially breast and frequently prostate cancer. Oral bisphosphonates are used to treat osteoporosis, osteitis deformans (Paget’s disease of the bone) and other conditions that lead to bone fragility.

**Stage 1**

Stage 1 - is characterized by exposed bone, that shows no indication of disease or inflammation of the soft tissue around the bone.

**Stage 2**

Stage 2 - is distinguished by painful areas of exposed bone accompanied by soft tissue
Stage 3

Stage 3 - is the most advanced stage of BRONJ. One of the most significant features is a fracture of bone that has been weakened by the disease. In addition, there is an extensive amount of exposed bone, soft-tissue inflammation and infection.

(American Academy of Oral and Maxillofacial Surgeons, 2013)

Panoramic radiograph revealing a segment of detached necrotic bone in the lower left jaw of a patient receiving oral bisphosphonates for osteoporosis.

- Greatest Risk / IV bisphosphonates
  - Zometa
  - Reclast
  - Aredia
- Lower Risk / Oral bisphosphonates
  - Fosamax
  - Boniva

Treatment

- Daily irrigation and anti-microbial rinses
- Antibiotics to control infection
- Surgical treatment to remove necrotic bone may be advisable in more advance cases

Oral Cancer and Technology in Research
In 2016, mechanical engineers at the University of Washington, have constructed a pen-size cancer cell detecting microscope, which is a huge breakthrough for surgeons removing cancer! As Dennis Wise at the U of W explains, “The miniature microscope uses an innovative approach called ‘dual-axis confocal microscopy’ to illuminate and more clearly see through opaque tissue. It can capture details up to a half millimeter beneath the tissue surface, where some types of cancerous cells originate.” It was originally intended for surgeons during cancer removal, to be able to differentiate between healthy tissue versus cancer-laden tissue while excising cancer out of the body, to not only save healthy tissue (especially brain,) but also to ensure no cancer cells are left behind. If successful in the trials, this differentially-diagnosing tool could be so impactful in the dental office, too, as the first defenders against head and neck cancer, highlighting cancerous oral lesions more easily!

Citing the Oral Cancer Foundation:

*Important research into oral and oropharyngeal cancer is underway in many university hospitals, medical centers, and other institutions around the country. Each year, scientists find out more about what causes the disease, how to prevent it, and how to improve treatment.*

*DNA changes: A great deal of research is being done to learn what DNA changes are responsible for causing cells of the oral cavity and oropharynx to become cancerous. One of the changes often found in DNA of oral cancer cells is a mutation of the p53 gene. The protein produced by this gene normally works to prevent cells from growing too much and helps to destroy cells with DNA damage too extensive for the cells to repair. Damage to p53 DNA can lead to increased growth of abnormal cells and formation of cancers. Recent studies suggest that tests to detect these p53 gene alterations may allow very early detection of oral and oropharyngeal tumors. These tests may also be used to better define surgical margins (check to see if all cancer cells have been removed) and to determine which tumors are most likely to respond to surgery or radiation therapy.*

*Another DNA change found in some oral cancers is that DNA from a papillomavirus (HPV) becomes mixed together with the patient’s own DNA. Some parts of the HPV DNA instruct the cells to produce proteins that inactivate the p53 protein. Studies are underway to determine whether tests to detect HPV DNA may help in diagnosing these cancers.*

*Tumor growth factors: Researchers have discovered naturally occurring substances in the body that promote cell growth. These hormone-like substances are called growth factors. Growth factors activate cells by attaching to growth factor receptors, which are present on the outer surface of the cells. Some cancer cells grow especially fast because they contain more growth factor*
receptors than normal cells do. One of the growth factors that has been linked to oral and oropharyngeal cancers is called epidermal growth factor or EGF. Oral and oropharyngeal cancers with too many EGF receptors tend to be especially aggressive. New drugs that specifically recognize and cells with too many EGF receptors are now being tested in clinical trials. These drugs work by preventing EGF from promoting reproduction of cancer cells, and may also help the patient’s immune system recognize and attack the cancer. Preliminary studies indicate that at least one such drug, called C225, makes radiation therapy more effective in killing head and neck squamous cell cancers.

New chemotherapy: Researchers continue to develop new chemotherapy drugs that might be more effective against advanced oral and oropharyngeal cancer. Intraarterial chemotherapy (injection of drugs into arteries feeding the cancer) is being tested in combination with radiation therapy in an attempt to improve their effectiveness. Another new approach to treating head and neck cancers is intralesional chemotherapy (injecting the drug directly into the tumor). Until recently, success with this approach was limited because the drug tended to spread to nearby tissues and the rest of the body quite quickly. Recent advances in preparing the drug solution, so that it remains localized in the tumor, have renewed the interest in intralesional chemotherapy, and preliminary results have been promising.

New radiotherapy methods: Several clinical trials have been conducted to test the effectiveness of new radiation regimens delivering twice-a-day irradiation in the treatment of oropharyngeal cancer. Higher cure rates have been obtained with a couple of these new regimens. Clinical trials are ongoing to confirm these initial findings. There has also been progress in reducing xerostomia (dry mouth), one of the most important side effects of head and neck radiation therapy. Recent research suggests that amifostine can help reduce this side effect by limiting radiation damage to salivary glands. The drug is given into a vein a few minutes before each radiation treatment. Side effects of amifostine include low blood pressure, nausea, and vomiting. Also, a new protocol for radiating an area from multiple angles, and controlled by new software and blocking shutters, seems to be useful in avoiding the radiation induced destruction of the salivary glands by targeting multiple beams around them, removing the gland from the radiation field.

Vaccines: Most people think of vaccines as a way to prevent infectious diseases such as polio or measles. However, vaccines are being studied as a way to treat people with cancer by helping their immune to recognize and attack the cancer cells. Since some oral and oropharyngeal cancers contain DNA from human papillomaviruses, vaccines against these viruses are being studied as a treatment for these cancers.
Gene therapy: New discoveries about how changes in the DNA of cells in the oral cavity and oropharynx cause these cells to become cancerous are being applied to experimental treatments intended to reverse these changes. For example, clinical trials are testing whether it is possible to replace abnormal tumor suppressor genes (such as the p53 gene) of oral cancer cells with a normal copy, to restore normal growth control. Gene therapies to interfere with growth-stimulating effect of certain papillomaviruses are being developed. Another type of gene therapy adds new genes to the cancer cells to make them more susceptible to being killed by certain drugs.

Conclusion

All clinical practitioners are encouraged to use a variety of references in their office libraries and online for additional case presentations, and to continuously upgrade and maintain a knowledge base about oral pathology for best practices management for their patients. This course is meant to be a survey of relatively common oral pathology conditions and is not a substitute for academic instruction. Dentists should refer complex cases to the appropriate specialist after initial consultation and diagnosis.

References


Medscape.com; various clinical definitions in reference section; Reviewed January 2017.

Oral Cancer Foundation, http://oralcancerfoundation.org/facts/whats-new-oral-cancer-


Course Exam: Oral Pathology Review

1. A fibroma’s characteristics include all of the following except:
   a. Generally benign
   b. Also known as a “vascularized fibroma”
   c. Recurrence is rare.
   d. Excision is effective

2. Pyogenic granuloma is generally a neoplastic lesion.
   a. True
   b. False

3. Which of the following is NOT true about a peripheral ossifying fibroma?
   a. Red to pink in color
   b. Reactive
   c. Stimulated by pregnancy
   d. Common gingival growth

4. A giant cell granuloma is similar in appearance to a pyogenic granuloma.
   a. True
   b. False

5. Gingival overgrowth is associated with all of the following medications, except:
   a. Dilantin
   b. Calcium channel blockers
   c. Minocycline
   d. Cyclosporine

6. A gingival cyst usually appears in the retromolar pad area.
   a. True
   b. False

7. Which of the following is true about benign migratory Glossitis?
   a. Greater incidence in females
   b. Also known as geographic tongue
   c. A & B
   d. B only
8. Pseudomembranous Candidiasis is associated with oral inhaled steroids and systemic antibiotics.
   a. True
   b. False

9. Anti-fungal strategies for candidiasis include topical medications only.
   a. True
   b. False

10. Apthous ulcers include a herpetiform variant.
    a. True
    b. False

11. A characteristic of Secondary Herpes Simplex includes:
    a. Herpes labalis
    b. Prodrome
    c. Herpetic whitlow
    d. All of the above

12. Herpes zoster is commonly referred to as shingles.
    a. True
    b. False

13. Lichen planus is primarily a systemic and auto-immune disease.
    a. True
    b. False

14. In a case of pemphigoid, the most important patient referral is:
    a. Internal medicine
    b. Immunologist
    c. Ophthalmologist
    d. Pulmonologist

15. A benign mixed tumor (pleomorphic adenoma) is characterized in patients as extremely painful.
    a. True
    b. False
16. The most common salivary malignancy is:
   a. Mucoepidermoid carcinoma
   b. Squamous carcinoma
   c. Primary parotid carcinoma

17. Mucocoeles are associated with sialoliths and sialoadenitis.
   a. True
   b. False

18. Dermoid cysts may contain skin structures like hair follicles and sweat glands.
   a. True
   b. False

19. Medication induced pigmentation includes all of the following medications except:
   a. Anti-malarials
   b. Anti-fungals
   c. Antibiotics
   d. Tranquilizers

20. Amelogenesis imperfecta is associated with all except:
   a. Defect in enamel development
   b. Hypocalcification
   c. Hypomaturation
   d. None of the above
   e. All of the above

21. A dentigerous or follicular cyst is the most common developmental odontogenic cyst.
   a. True
   b. False

22. An odontogenic keratocyst is not well-defined on a radiograph, and therefore difficult to diagnose.
   a. True
   b. False

23. A Stafne defect usually required aggressive treatment including excision.
   a. True
   b. False
   a. True
   b. False

25. BRONJ, is a condition related to use of steroids in women.
   a. True
   b. False