ORAL PATHOLOGY

-OUTLINE-

I. SOFT TISSUE LESIONS

A. Warm up: Interesting-looking tongues
1. Geographic tongue
2. Hairy tongue
3. Fissured tongue
4. Median rhomboid glossitis
5. Bifid tongue

B. Common oral lumps and bumps
1. Fibroma
2. Epulis fissuratum
3. Peripheral ossifying fibroma
4. Pyogenic granuloma
5. Peripheral giant cell granuloma
6. Lipoma
7. Nerve sheath neoplasms
    a. Neurofibroma
    b. Schwannoma
    c. Neurofibromatosis
    d. Multiple neuroendocrine syndrome, type 3
8. Salivary gland neoplasms
    a. Pleomorphic adenoma
    b. Monomorphic adenoma
    c. Mucoepidermoid carcinoma
    d. Adenoid cystic carcinoma
9. Papilloma

B. Common non-neoplastic oral conditions
1. Sialolithiasis
2. Mucocele
3. Ranula
4. Nicotine stomatitis
5. Xerostomia
6. Sjögren's syndrome
7. Drug-induced gingival hyperplasia
8. Leukemia
9. "Meth mouth"

C. White, red, and pigmented lesions
1. Oral leukoplakia
2. Proliferative verrucous leukoplakia
3. Candidiasis
4. Lichen planus
5. Oral erythroplakia

6. Amalgam tattoo
7. Oral melanotic macule
8. Melanoacanthoma
9. Oral nevi
10. Melanoma

D. Oral ulcerative conditions
1. Aphthous stomatitis
2. Herpes simplex

E. Oral cancer
1. Squamous cell carcinoma
2. Verrucous carcinoma
a. Smokeless tobacco use
4. Oropharyngeal carcinoma

II. BONY LESIONS

A. Radiolucent lesions
1. Cysts of the jaws
a. Radicular cyst
b. Dentigerous cyst
c. Eruption cyst
d. Lateral periodontal cyst
e. Gingival cyst
f. Odontogenic keratocyst
i. Basal cell nevus syndrome
g. Traumatic bone cyst
2. Multilocular neoplasms
a. Ameloblastoma
b. Central giant cell granuloma
3. Destructive radiolucencies
a. Metastatic cancer
b. Bisphosphonate-induced osteonecrosis

B. Radiopaque lesions
1. Odontomas
2. Osteitis deformans (Paget's disease of bone)

C. Mixed radiolucent/radiopaque lesions (fibro-osseous lesions)
1. Osseous dysplasia
a. periapical
b. Focal
c. Florid
2. Fibrous dysplasia

DIFFERENTIAL DIAGNOSIS OF ORAL LESIONS

NAME OF DISEASE	DEFINITION/ETIOLOGY	EPIDEMIOLOGY	CLINICAL FEATURES	MICROSCOPIC FEATURES	TX/PROGNOSIS
Fibroma	Reactive proliferation of collagen in response to trauma	Common - the most commonly biopsied oral lesion! Usually adults	Buccal mucosa, lateral tongue… Normal colored, smooth surfaced, firm sessile nodule	Nodule of dense fibrous connective tissue	Conservative surgical excision is usually curative
Peripheral ossifying fibroma **Peripheral odontogenic fibroma**	Reactive? Neoplastic? Proliferation of gingival collagen	Most common in young adult black females	Gingiva. Normal colored, smooth surfaced, firm sessile nodule	Nodule of dense collagen containing bone and/or odontogenic epithelium	Conservative excision. Recurs in 25% of cases
Pyogenic granuloma	Reactive proliferation of granulation tissue in response to local factors on teeth (plaque/calculus) or trauma	Most common in young adult females. AKA "pregnancy tumor"	Gingiva. Red lobulated mass with superficial ulceration. Often bleeds	Mass of granulation tissue with young collagen, capillaries, and inflammatory cells	Surgical excision and removal of local factors on adjacent teeth; may recur
Peripheral giant cell granuloma	Reactive? Neoplastic?	Uncommon. Most occur in first 5 decades.	Gingiva. Purple nodules which grow rapidly; may ulcerate and erode underlying bone.	Vascular spindle-cell stroma containing numerous multinucleated giant cells	Surgical excision. May recur. Rule out hyperparathyroidism if bone is involved.
Lipoma	Benign neoplasm of adipose tissue	Uncommon. Usually adults	Subcutaneous tissue of neck; buccal mucosa and vestibule	Encapsulated mass of mature adipose tissue	Surgical excision is usually curative
Nerve sheath tumors	Neoplasms of the nerve sheath – neurofibroma and schwannoma May be associated with genetic syndromes.	Uncommon. Usually adults	Tongue, lips, palate… fibroma-like nodules	Neurofibroma – admixture of nerve sheath elements with axons Schwannoma – Schwann cells with formation of verocay bodies	Solitary lesions are cured by excision. Those associated with genetic syndromes – see below!

Neurofibromatosis	Autosomal dominant mutation of NF1 gene	Uncommon. Cases are mild early in life but become progressively more severe	Multiple neurofibromas both external and interna; café-au-lait spots of skinl; wide mandibular neurovascular bundle; enlargedcircumvallate papillae	See above	No cure other than to manage the various manifestations. Lesions cause paralysis and other neurological symptoms. Risk of malignant transformation.
Multiple endocrine syndrome, type 3	Autosomal dominant trait of RET gene	Rare. Manifestations accumulate throughout life	Multiple mucosal neuromas (100%), Marfanoid habitus, risk of developing medullary thyroid carcinoma and pheochromocytoma	Neuromas are similar to neurofibromas	No cure. Important to recognize and treat cancers early
Granular cell tumor	Benign neoplasm of Schwann cells	Uncommon. Usually adults	Most common on dorsal tongue	Polygonal granular cells; overlying epithelial hyperplasia may mimic cancer	Conservative surgical excision is curative
Pleomorphic adenoma	Benign salivary gland neoplasm	Uncommon; usually adult females	Parotid gland, hard palate, upper lip	Encapsulated mass of myoepithelial cells in various patterns with ductal formation…	Complete surgical excision; long-standing lesions may undergo malignant transformation – carcinoma ex pleomorphic adenoma
Monomorphic adenoma	Benign salivary gland tumor	Uncommon, adults	Canilicular type occurs almost exclusively in the upper lip as a well-circumscribed, movable nodule	Encapsulated mass with a monotonous pattern of ductal formation	Conservative surgical excision is curative
Mucoepidermoid carcinoma	Malignant salivary gland tumor	Uncommon; adults	Parotid gland, hard palate; nodular mass with cystic areas	Unencapsulated admizture of epidermoid (squamous) cells and mucous cells.	Complete surgical excision; prognosis determined by differentiation (more mucous cells best)
Adenoid cystic carcinoma	Malignant salivary gland tumor	Uncommon, adults	Usually oral and on hard palate; asymmetrical mass	Unencapsulated atypical myoepithelial cells forming back-to-back ductal structures (cribiform pattern)	Radical surgery, irradiation, chemotherapy; very poor ultimate prognosis

Sialolithiasis	Formation of a salivary stone; calcium carbonate salts deposit around organic nidus	Fairly common; adults	65% submandibular duct; 20% parotid duct, 1% minor glands; pain and swelling in gland at mealtimes	Concentric rings of calcification around a central nidus	Surgical removal of stone; may cause fibrosis of gland
Mucocele	Submucosal accumulation of saliva due to traumatic rupture of a salivary gland duct	Common – The most commonly biopsied oral lesion in children! Children and young adults	80% occur on mucosal aspect of lower lip as a red-purple fluctuant mass	Pseudocyst with pool of inspissated mucin surrounded by a granulation tissue reaction	Surgical removal is usually curative
Ranula	Submucosal accumulation of saliva; unknown etiology	Uncommon; usually adults	Large fluctuant swelling in the floor of the mouth	Like a mucocele	Marsupialization and allow lesion to heal by second intent (“granulate in”)
Xerostomia	Symptom of having a dry mouth; Salivary gland aplasia, irradiation, diabetes mellitus, medications, nutritional deficiencies, Sjogren syndrome	Common; usually females; incidence increases with age;	Dry mouth with resulting fragile mucosa; often associated by drying of eyes, vagina…	See below	Depends on etiology
Sjogren syndrome	Autoimmune disease against salivary and lacrimal glands; antibodies against ribonucleoprotein antigens	Uncommon; 90% middle-aged females	Xerostomia with secondary caries and periodontal disease; dry eyes; bilateral parotid and lacrimal gland enlargement common; many also have rheumatoid arthritis	Biopsy of minor salivary glands shows lymphocytic replacement of acini	No cure; immunosuppression and pilocarpine; complications of long-term steroid use (infections and osteoporosis); risk of lymphoma
Oral Leukoplakia	White patch on the oral mucosa which cannot be diagnosed as anything more specific; often due to trauma and tobacco use; some associated with sanguinaria extract, cinnamon reaction…	Common; usually adult males	Most common on alveolar ridge, mucobuccal fold, buccal mucosa…; lesions on tongue and floor of the mouth are considered “high risk”; white-gray patch (red implies high risk)	Hyperkeratosis and epithelial hyperplasia; may show varying degrees of dysplasia	Low risk lesions: remove etiology and watch for resolution – biopsy if persists. High risk lesions should be biopsied immediately. Small percentage will become malignant
Proliferative verrucous leukoplakia (PVL)	Leukoplakia with a high risk for malignant transformation; unknown etiology – not associated with tobacco, alcohol…; possibly viral (HPV?)	Uncommon overall but more common in southeast U.S.; usually middle-aged, adult females	Buccal mucosa, gingival, tongue – often involved multiple sites; white, exophytic, verrucoid plaque	Verrucoid hyperplasia with hyperkeratosis and dysplasia of varying degrees	Complete surgical excision and close followup; 70% progress to SCC and 40% die of PVL-associated cancer!

Candidiasis	Superficial fungal infection predisposed by diabetes mellitus, nutritional deficiencies, antibiotic therapy immunosuppression, radiotherapy.	Common – The most common opportunistic infection in the entire body!	Multiple white patches throughout the mouth which burn slightly and wipe off with a gauze sponge/tongue blade leaving a raw base of inflamed tissue	Fungal hyphae in superficial layers of epithelium – need PAS stain to see organism clearly; can be recovered with cytology smear	Antifungal drugs are usually effective – nystatin chlortrimazole, ketaconazole… May be refractory in immunosuppressed patients
Lichen planus	Inflammatory disease of skin and mucosa; probably autoimmune in nature	Common – Affects 1% of population; usually onset in young adult females	Oral lesions are white papules which interconnect to form a lacy pattern (Wickham striae); Skin lesions are pruretic, purple, polygonal papules	Hyperkeratotic and hyperplastic epithelium with necrosis of basement membrane; intense lymphocytic infiltrate in lamina propria	Establish diagnosis; classic reticular lesions rarely go away completely; erosive lesion treated with anti-inflammatory medications; Debatable association with oral cancer
Drug-induced gingival hyperplasia	Enlargement of the gingival due to ingestion of a drug (and often poor oral hygiene); many drugs possible: dilantin, calcium channel blockers, cyclosporine…	Fairly common; usually adults	Generalized gingival hyperplasia with secondary inflammation due to poor oral hygiene	Non-specific epithelial and fibrous hyperplasia	Discontinue or change medication; periodic recontouring gingivectomies may be necessary
Leukemia	Malignant neoplasm of blood and bone marrow; Many genetic alterations have been identified, some of which are specific to various type of leukemia; predisposed by irradiation…	Fairly common; all ages affected	Systemic: symptoms of anemia, infection, and bleeding; organ failure due to infiltration may occur Oral: gingival bleeing, mucosal ulceration, gingival enlargement, formation of mucosal masses	Infiltration of gingival by atypical mononuclear cells	Chemotherapy, total body irradiation with bone marrow transplant. Prognosis much improved with modern chemotherapy but still a serious disease
Amalgam tattoo	Traumatic implantation of amalgam particles during restorative dentistry	Very common – The most common oral pigmented lesion! Usually adults	Gingiva, buccal mucosa… Black macule. Amalgam particles may appear on x-ray	Particles of amalgam in lamina propria with minimal inflammatory reaction	None if diagnosis can be substantiated clinically; excise if unsure
Oral melanotic macule	Melanocytic oral pigmented lesion; often associated with sunlight exposure	Fairly common; usually adults	Lower lip most common, sometimes on gingival; brown macule	Increase in melanin production by melanocytes	Surgical excision to rule out melanoma

Melanoacanthoma	Melanocytic oral lesion; may be caused by trauma	Rare; almost always young adult black male	Black-to-brown macule which rapidly enlarges	Proliferation of melanocytes with prominent dentritic processes	Biopsy to establish diagnosis – lesion often regresses rapidly following biopsy
Oral melanocytic nevi	Hamartomatous pigmented lesions of neural crest origin	Very common on skin – Average Caucasian in U.S. has 17 nevi Oral lesions uncommon and usually occur on palate or gingiva	Junctional nevus – flat, light brown and hairless Compound nevus – nodular, dark and hair-bearing Intradermal nevus – same as compound Blue nevus – Dark indigo nodule Oral lesions are usually either intramucosal or blue and appear as brown or blue-gray macules	Nevus cell proliferation in theques (junctional) or in the lamina propria (compound and intramucosal).	Biopsy to rule out melanoma
Oral melanoma	Malignant neoplasm of melanocytes; associated with a variety of genetic abnormalities; skin lesions are associated with sublight exposure	Oral melanomas are rare. Skin lesions are uncommon but increasing in frequency; usually blond, blue-eyed, caucasians	Pigmented mass with ulceration; often on palate and/or gingival; ABC's of melanoma: Asymmetry Borders irregular Coloration variable Diameter enlarging Evolution of nevus	Malignany melanocytes invading both down into the lamina propria and up into the epithelium	Surgery, chemotherapy, radiotherapy – someday maybe immunotherapy Very poor prognosis for oral melanomas; skin lesion have a five year survival of around 70%
Aphthous ulcers	Mucosal ulceration due to a localized immune derangement – either hypersensitivity or autoimmune	Very common; usually females; onset in 2nd decade	Loose movable mucosa – labial, buccal, and lingual mucosa; small, shallow yellow ulcers with red inflammatory halo; very painful	Non-specific ulcer	Solitary lesions best treated with cautery or topical anti-inflammatory agent; Multiple lesions managed with steroid rinses.
Herpes simplex	Viral infection by HSP, type 1 (usually) or HSV, type 2 (sometimes).	Very common; primary infection in 1st decade; secondary infections thereafter	Primary infection: generalized gingovostomatitis Secondary infection: usually herpes labialis or lesions on mucosa over bone Vesicles… painful erosions	Intraepithelial vesicles with ballooning degeneration of epithelial cells; Cowdry inclusions seen in nucleus	Supportative therapy for primary infection. Secondary infections may be treated with antiviral agents if necessary

Oral squamous cell carcinoma	Malignant neoplasm of oral mucosa; associated with tobacco use, alcohol abuse, sunlight exposure (lip), nutritional deficiency, trauma (debatable), and possibly viruses; p53 and other mutations are common	Uncommon but the most common form of oral cancer; typically patient is a white male, over age 55 who smokes and drinks.	Lower lip, lateral and ventral tongue, floor of the mouth, buccal mucosa, gingival, palate… Early lesions may be areas of leukoplakia or erythroplakia… later lesions are ulcerative Any mucosal ulcer that does not heal with two weeks should be considered cancer until proven otherwise!	Invading islands of dysplastic squamous epithelium with keratin pearl formation.	Radical surgery, irradiation, chemotherapy or a combination. 50% overall five year survival rate. Lateral tongue worse followed by floor of mouth… Most cases fail due to lymph node or distant metastasis.
Oropharyngeal carcinoma	Malignant neoplasm of oropharyngeal structures; majority associated with HPV infection	Uncommon but increasing in incidence; usually middle-age men	Soft palate, tonsils, base of tongue, posterior pharyngeal wall. Primary lesions are often small ulcers which are difficult to visualize. A metastasis to a lymph node in the neck is often the initial presenting sign.	Poorly differentiated squamous cell carcinomas	Irradiation and chemotherapy. Debulking surgery in some cases. 25% five year survival rate
Papilloma	Benign epithelial neoplasm caused by HPV	Common; usually adults but seen in children too	Soft palate, tongue… Small, exophytic, white papillary lesions	Papillary proliferation of stratified squamous epithelium	Surgical excision is usually curative
Meth Mouth	Dental disease associated with methamphetamine use. Damage caused by acidic nature of drug, xerostomia, heavy sugar comsumption, poor oral hygiene, and bruxism	Uncommon but meth addiction is increasing rapidly. Also all addicts have oral manifestations. Usually young adults.	Drug causes extreme euphoria, insomnia, and obsessive behavior. Addicts experience rapidly progressive and severe dental caries and periodontal disease; attrition and fracture of teeth; tendency to form abscesses…	NA	Drug rehabilitation and restorative dental treatment. Multiple teeth are usually lost.
Mucosal cyst of the maxillary sinus	Retention of mucus beneath the Schneiderian membrane; often associated with upper respiratory infection (cold)	Uncommon; usually adults	Asymptomatic dome-shaped opacity on one of the walls of the maxillary sinus	Pseudocyst with mucus retention beneath the sinus epithelium	Inconsequential; most resolve with URI

Lingual mandibular salivary gland depression	AKA Stafne defect; anatomic depression in lingual mandible by submandibular gland	Common; usually adults	Asymptomatic discrete radiolucency inferior to the mandibular canal in the posterior mandible	Composed of normal submandibular gland	Inconsequential; no treatment other than establish diagnosis
Simple bone cyst	AKA traumatic bone cyst…; might be caused by trauma Some associated with fibro-osseous lesionsof the jaws	Fairly common; typically in males in 2nd decade	Asymptomatic discrete radiolucency which tends to dome up between the roots of vital teeth; almost always in mandible	Technically composed of air since it is an empty hole	Open lesion to establish diagnosis, and to stimulate bleeding and healing
Radicular cyst	Cyst associated with the root of a non-vital tooth; a sequelae of dental caries… pulpitis… periapical inflammation stimulated proliferation of rests of Malassez… cystification	Very common – The third most commonly biopsied lesion (behind fibromas and periapical granulomas). Any age but usually adults	Discrete radiolucency classically at the apex of the root of a non-vital, carious tooth; some arise from accessory root canals on the lateral aspect of the root; usually painless	Lumen filled with necrotic debris, lined with SSE, wall of inflamed collagen	Endodontics or extraction of the tooth
Dentigerous cyst	Cyst associated with the crown of an unerupted tooth; arises from cystification of the reduced enamel epithelium	Very common – The second most common cyst of the jaws behind radicular cysts. Usually diagnosed in 2nd-3rd decades	45% associated with unerupted mandibular third molars, 15% with maxillary cuspids, 10% with maxillary third molars, 30% with the rest of the teeth; present as a well-circumscribed radiolucency around the crown of the tooth; may displace teeth and expand bone	Lumen filled with necrotic debris, SSE lining, and wall of dense collagen	Removal of cyst and unerupted teeth; rarely ameloblastomas and other neoplasms may arise from epithelial lining
Eruption cyst	Cyst forming around the crown of an erupting tooth; arises from cystification of the dental follicle	Common; children who are erupting teeth	Usually affects permanent first molars or incisors; fluctuant, dome-shaped swelling	Roof of normal mucosa; cystic space lined with reduced enamel epithelium	Inconsequential; unroof lesion and allow tooth to erupt
Lateral periodontal cyst	Cyst occurring between the roots of adjacent vital teeth; arises from rests of Malassez	Uncommon, adults	Small, well-circumscribed radiolucency; usually in the mandibular premolar area; may cause slight alveolar expansion; multilocular variant is the botryoid odontogenic cyst	Lumen contains necrotic debris; SSE lining with focal nodular areas; wall of dense collagen	Conservative surgical excision

Gingival cyst of the adult	Cyst located in the gingival mucosa; arises from rests of Malassez	Uncommon; adults	Small, normal-colored nodules in the attached gingival; usually in the premolar region of the mandible; typically do not show up on x-day	Identical to lateral periodontal cyst	Conservative surgical excision
Odontogenic keratocyst	Aggressive odontogenic cyst which tends to mimic other cysts clinically and radiographically; arises from remnants of the dental lamina	Relatively common – The 3rd most common cyst of the jaws behing the radicular and dentigerous cysts. Usually adults	Discrete radiolucent lesions which are usually unilocular but may become bilocular or multilocular; most occur in the posterior mandible; they may get very large and cause jaw expansion	Lumen is typically filled with keratin; lining if parakeratinized SSE with a prominent basal cell layer of polarized columnar cells; wall is composed of dense collagen. "Daughter" cysts are often seen	Complete surgical excision; up to 40% recur so followup is mandatory; may be associated with Gorlin syndrome
Gorlin syndrome	AKA: basal cell nevus syndrome. Autosomal dominant trait due to mutations in the PTCH tumor suppressor genes	Rare but entire families tend to be affected to varying degrees; congenital but manifestations become more obvious with age	Major manifestations include multiple basal cell carcinomas of skin, multiple keratocysts of the jaws, and skeletal anomalies such as bifid ribs. Minor manifestations include hypertelorism, mild cognitive dysfunction, hypogonadism…	Typical basal cell carcinomas and OKC's	No cure; manage skin cancers and jaw cysts
Ameloblastoma	Technically benign but locally aggressive epithelial odontogenic tumor; etiology unknown	Rare; peak age between 20-40 years	Classically a multilocular ("soap-bubble") radiolucency in the posterior mandible; often associatyed with unerupted tooth; tooth resorption and/or displacement is common; may achieve large size and expand jaw	Islands of odontogenic epithelium resembling enamel organ (peripheral columnar cells with reverse nuclear polarization and central stellate reticulum) in a stroma of dense collagen	Complete surgical excision, usually jaw resection; tend to recur
Cystic ameloblastoma	Cystic variant of the ameloblastoma; unknown etiology	Uncommon; usually 2nd-3rd decades	Classically presents as a dentigerous cyst-like radiolucency around the crown of an unerupted tooth; usually mandibular third molars	Dentigerous cyst with an ameloblastoma arising from the epithelial lining; may be luminal, mural or a hybrid	Conservative surgical removal; rarely recur

Odontoma	Odontogenic hamartoma arising from tooth bud	Common – The most common odontogenic tumor. Usually in 1st-2nd decades	Two types: Compound odontoma: most common, a sac of small toothlets surrounded by a dental follicle, usually in the anterior maxilla Complex odontoma: an amorphous mass of dental hard tissue surrounded by a dental follicle, usually in the posterior jaws	Composed of enamel, enamel matrix, dentin, cementum, pulp, and follicular tissues	Conservative surgical excision
Osteitis deformans	AKA Paget disease of bone. Metabolic disorder of bone deposition and resorption; possibly a myxovirus infection	Common in mild form, affecting about 1% of the elderly	Early lesions are osteolytic but rarely diagnosed. Later lesions produce a “ground glass” opacity and enlargement of affected bones; usually polyostotic affecting vertebrae, parietal bones, long bones, jaws (maxilla > mandible); pathologic fractures, bowing of long bones, obliteration of nerve foraminae (blindness and deafness) may occur	Late stage bone shows both osteoblastic and osteoclastic activity; bone is laid down in irregular increments producing a “jigsaw puzzle” pattern in the bone	No cure; manage problems as they arise; risk of osteosarcoma
Periapical osseous dysplasia	Benign fibro-osseous lesion of the jaws; unknown etiology	Common; classically occurs in middle-age black females	Almost exclusively the periapical region of the mandibular incisor teeth as multiple lesions; early lesions are lytic, middle stage lesions are mixed, mature lesions are radiopaque; lesions are asymptomatic and caries is usually absent	Admixture of poorly formed woven bone in a cellular collagenous matrix	Inconsequential

Focal osseous dysplasia	Benign fibro-osseous lesion of the jaws; unknown etiology	Common – The most commonly biopsied fibro-osseous lesion; usually in middle age white females	Tend to occur in edentulous areas of the mandible as a solitary lesion less than 1.5 cm in diameter without jaw expansion; radiolucent early, mixed later, opaque when mature	Admixture of poorly formed woven bone in a cellular collagenous matrix	Usually excised to rule out other lesions; inconsequential
Florid osseous dysplasia	Benign fibro-osseous lesion of the jaws; unknown etiology	Common; classically middle aged black females	Multiple lesions, often in the tooth-bearing areas of all four quadrants of the jaws. May be associated with simple bone cysts; lucent early… opaque later; does not expand jaws	Admixture of poorly formed woven bone in a cellular collagenous matrix	Inconsequential unless secondarily infected
Fibrous dysplasia	Benign fibro-osseous lesion which often affects the jaws; etiology unknown	Uncommon; usually diagnosed in 2nd decade	Four forms: Monostotic: involved one lesion and usually involves one bone Craniofacial: most common form of monostotic FB which involves the face/jaws Polyostotic: multiple lesions in multiple bones without endocrine abnormalities McCune-Albright syndrome: polyostotic FB with precocious puberty and other endocrine abnormalities	Stroma of cellular, dense collagen containing spicules of woven bone resembling Chinese letters	Diagnose with clinical/radiologic/histologic correlation. Try to wait until after growth has stopped before surgical removal or recountouring of lesions. Early intervention may stimulate growth.
Metastatic cancer to the jaws	Hematogenous spread of a malignant neoplasm from a primary site to the jaws; Most common primary sites are breast, prostate, lung, and kidney	Uncommon; usually adults who may or may not know they have cancer	Usually posterior mandible as a destructive lytic lesion; almost always painful; may cause pathologic fracture	Malignant cells mimicking the primary neoplasm	Very poor prognosis since metastasis to jaws is often accompanied by widespread dissemination; palliative chemotherapy…

Bisphosphonate-associated osteonecrosis of the jaws	Complication of bisphosphonate therapy for a systemic disorder (severe osteoporosis, metastatic cancer…); drugs block osteoclastic activity and inhibit hydrxyapatite dissolution; precipitated by tooth extraction, periodontal disease, dentures, implant placement…	Uncommon but increasing in incidence; typically elderly patients who are receiving IV administration of bisphosphonates; much lower risk with oral bisphosphonates	65% mandible, 25% maxilla, and 10% both jaws; lesions range from a “dry sock” or massive osteonecrosis with exposure of dead bone; irregular radiolucency; often painful	Necrotic bone and soft tissue; secondary inflammation is often present	Refer to ADA/AAMOS guidelines. Patients should be referred to dentist before being placed on bisphosphonates to allow for existing dental disease to be minimized. Minor dentoalveolar surgery can usually be performed on patients who have been on oral bisphosphonates less than 3 years. Patients on oral bisphosphonates longer than 3 years should be taken off for 3 months prior to oral surgery and allowed to heal before continuing drug therapy Contact with bone ahould be avoided in patients on IV bisphophonates unless absolutely necessary. Staging: I – Exposed necrotic bone which is asymptomatic and without secondary infection – treat with chlorhexidine rinses II – Exposed necrotic bone with pain and infection – treat with antibiotics and oral antimicrobial rinses III – Stage II with fistulation, pathologic fracture, or necrosis to inferior border – surgical debridement with Stage II tx

XEROSTOMIA

DEFINITION: THE CLINICAL MANIFESTATION OF DRYNESS OF THE MOUTH

ETIOLOGY: DECREASED SALIVARY FLOW

1. SALIVARY GLAND APLASIA
2. IRRADIATION OF THE SALIVARY GLANDS
3. DIABETES MELLITUS
4. INGESTION OF VARIOUS MEDICATIONS
5. AUTOIMMUNE DISORDERS
   - SJOGREN'S SYNDROME
6. VITAMIN DEFICIENCIES – A, RIBOFLAVIN, NIACIN

CLINICAL FEATURES:

1. EPIDEMIOLOGY
   - A. INCIDENCE – RELATIVELY COMMON
   - B. AGE – INCREASES IN INCIDENCE WITH AGE
   - C. GENDER – FEMALE PREDILECTION
2. ORAL SIGNS AND SYMPTOMS
   - A. DRY ORAL MUCOSA
   - B. BURNING SENSATION OF THE MUCOSA
   - C. PALE, TRANSLUCENT MUCOSA
3. EXTRA-ORAL SIGNS AND SYMPTOMS
   - A. DRY THROAT
   - B. DRY EYES AND BLURRED VISION
   - C. DRY SKIN
   - D. DECREASED VAGINAL SECRETION WITH ITCHING AND SUSCEPTIBILITY TO CANDIDIASIS

TREATMENT:

1. ELIMINATE CAUSE, IF POSSIBLE
2. SIP WATER, CHEW GUM, ARTIFICIAL SALIVA
3. MEDICATIONS TO STIMULATE SALIVARY FLOW
   - SIALOGOGUES SUCH AS PILOCARPINE, CEVIMELINE, BETHANECHOL, ANETHOLE-TRITHIONE, BROMHEXINE AND MUCOLYTIC AGENTS
4. DISEASE ALTERING AGENTS SUCH AS ALPHA INTERFERON, HYDROZYCHLOROQUINE, AND ANTI-INFLAMMATORY DRUGS

XEROSTOMIA

PROGNOSIS:

1. MOST CASES ARE PERMANENT

2. PREDISPOSES TO RAMPANT CARIES AND PERIODONTL DISEASE

3. DIFFICULTY WITH DENTAL PROSTHESES

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SJOGREN'S SYNDROME

DEFINITION: AUTOIMMUNE DISEASE PRIMARILY AFFECTING THE FUNCTION OF THE SALIVARY GLANDS.

ALSO KNOWN AS SICCA ("DRY") SYNDROME AND **MILKULICZ' DISEASE**.

ETIOLOGY: THE PRECISE MECHANISM OF THIS ATTACK BY T-LYMPHOCYTES ON EXOCRINE GLANDS IS UNKNOWN ALTHOUGH A RETROVIRUS HAS BEEN IMPLICATED.

CLINICAL FEATURES:

1. EPIDEMIOLOGY

   A. INCIDENCE - UNCOMMON BUT STILL TO SECOND MOST COMMON AUTOIMMUNE DISEASE (TO SYSTEMIC LUPUS ERYTHEMATOSUS).

   B. AGE - PEAK IN 5TH-7TH DECADES.

   C. GENDER - FEMALE 10:1 MALE.

2. TYPES AND SYMPTOMS

   A. PRIMARY SJOGREN'S SYNDROME
   - XEROSTOMIA WITH RESULTING CARIES, PERIODONTAL DISEASE...
   - BILATERAL PAROTID ENLARGEMENT (LYMPHOEPITHELIAL LESION)
   - DRY EYES (KERATOCONJUNCTIVITIS SICCA)
   - LACRIMAL GLAND ENLARGEMENT

   B. SECONDARY SJOGREN'S SYNDROME
   - ALL OF THE MANIFESTATIONS OF PRIMARY SJOGREN'S SYNDROME, PLUS
   - ANOTHER AUTOIMMUNE CONDITION WITH RHEUMATOID ARTHRITIS BEING MOST COMMON

MICROSCOPIC FEATURES:

BIOPSY OF THE MINOR SALIVARY GLANDS OF THE LOWER LIP IS OFTEN USED TO CONFIRM THE CLINICAL DIAGNOSIS OF THIS CONDITION.

THE SECTION SHOWS LOBULES OF MINOR SALIVARY GLANDS WITH ATTACHED COLLAGEN AND STRIATED MUSCLE. THE SALIVARY GLAND LOBULES ARE INFILTRATED WITH LYMPHOCYTES WHICH ARE ARRANGED BOTH DIFFUSELY AND INTO PATCHES. A FEW SCATTERED DUCTAL STRUCTURES ARE PRESENT WHICH EXHIBIT METAPLASIA AND FORM **EPIMYOEPITHELIAL ISLANDS**.

TREATMENT: ESSENTIALLY SYMPTOMATIC

1. ARTIFICIAL TEARS AND SALIVA.
2. SYSTEMIC CORTICOSTEROIDS.
3. PILOCARPINE TO STIMULATE SALIVARY FLOW.
4. ASPIRIN FOR RHEUMATOID ARTHRITIS.

PROGNOSIS:

1. TENDS TO BE A CHRONIC, DEBILITATING DISEASE WHICH IS RARELY FATAL ITSELF.
2. TENDS TO WAX AND WANE IN SEVERITY.
3. XEROSTOMIA CAUSES CARIES AND PERIODONTAL DISEASE (0.4% STANNOUS FLUORIDE GEL)
4. SUSCEPTIBLE TO ERYTHEMATOUS CANDIDIASIS AND ANGULAR CHEILITIS.
5. ORAL TISSUES ARE EASILY TRAUMATIZED.
6. LYMPHOMA HAS BEEN REPORTED TO DEVELOP IN SOME CASES.

DRUG-INDUCED GINGIVAL HYPERPLASIA

DEFINITION: OVERGROWTH OF GINGIVAL TISSUES DUE TO INGESTION OF VARIOUS DRUGS

ETIOLOGY:

1. MECHANISM - UNKNOWN ALTHOUGH POOR ORAL HYGIENE MAY BE A COFACTOR IN SOME CASES

2. SECONDARY TO INGESTION OF

   A. **PHENYTOIN (DILANTIN)**

   B. **CYCLOSPORIN**E (SANDIMMUNE)

   C. SODIUM VALPROATE (DEPAKENE)

   D. ERYTHROMYCIN (E-MYCIN, ERYBID, ERYTHROMID, ETC

   E. PRIMIDONE (MYSOLINE)

   F. PHENOBARBITAL (PHENOBARBITONE)

   G. **CALCIUM CHANNEL BLOCKERS**
   - DILTIAZEM (CARDIZEM, DILACOR)
   - NICARDIPINE (CARDENE)
   - FELODIPINE (PLENDIL)
   - NIMODIPINE (NIMOTOP)
   - NIFEDIPINE (ADALAT, PROCARDIA)
   - NITRENDIPINE (BAYPRESS)
   - DEPRIDIL (VASCOR)
   - VERAPAMIL (CALAN, ISOPTIN, VERELAN)

CLINICAL FEATURES:

1. EPIDEMIOLOGY

   A. INCIDENCE - UNCOMMON BUT INCREASING AS MORE PEOPLE TAKE CALCIUM CHANNEL BLOCKERS FOR HYPERTENSION

   B. AGE - NO PREDILECTION

   C. GENDER - NO PREDILECTION

2. DISTRIBUTION - USUALLY GENERALIZED IN GINGIVA

3. MORPHOLOGY - VARIES FROM SLIGHT HYPERPLASIA TO COMPLETE COVERAGE OF THE TEETH (**PSEUDOANODONTIA**)

4. SYMPTOMS - SECONDARY INFLAMMATION

MICROSCOPIC FEATURES:THE SECTION SHOWS A WEDGE OF MUCOSA WHICH IS SURFACED WITH STRATIFIED SQUAMOUS EPITHELIUM EXHIBITING ACANTHOSIS AND HYPERPARAKERATOSIS. THE LAMINA PROPRIA SHOWS MILD INFLAMMATION.

TREATMENT:

1. INSTITUTE GOOD ORAL HYGIENE

2. CONSIDER SWITCHING TO ANOTHER MEDICATION

3. GINGIVECTOMY - LASER WORKS WELL

PROGNOSIS: MOST CASES RECUR SLOWLY AFTER SURGERY

REFERENCES:

1. Harel-Raviv M et al: Nifedipine-induced gingival hyperplasia. Oral Surg Oral Med Oral Pathol 1995;79:715-722.

2. Montebugnoli L, Bernardi F, Magelli C: Cyclosporin-induced gingival overgrowth in heart transplant patients. J Clin Perio 1996

"METH MOUTH"

DEFINITION: ADVANCED TOOTH DECAY RESULTING FROM HEAVY METHAMPHETAMINE USE

ETIOLOGY: METHAMPHETAMINE USE

1. AKA ICE, SPEED, CRYSTAL
    a. BURST OF DOPAMINE (NEUROTRANSMITTER) RELEASE
    b. EXTREMELY ADDICTIVE – YOU CAN GET HOOKED ON 1ST TRY!
    c. CAN BE INJECTED, SMOKED, SNORTED OR TAKEN ORALLY
2. ADDICTS ARE KNOWN AS "TWEAKERS"
3. PEOPLE WHO MAKE IT ARE KNOWN AS "COOKERS"
4. MECHANISM
    a. ACIDIC INGREDIENTS IN DRUG (COMPONENTS INCLUDE BATTERY ACID, ANTIFREEZE, HYDROCHLORIC ACID, DRAIN CLEANER, LYE, RED PHOSPHORUS, LITHIUM, MURIATIC ACID, SULFURIC ACID, ETHER, LANTERN FUEL…)
    b. DRUG INDUCED XEROSTOMIA
    c. HEAVY CONSUMPTION OF CARBONATED BEVERAGES
    d. POOR ORAL HYGIENE
    e. TENDENCY TOWARDS BRUXISM

CLINICAL FEATURES:

1. EPIDEMIOLOGY
    a. 12 MILLION USERS IN U.S. AND INCREASING
    b. TEENAGERS AND YOUNG ADULTS
    c. MALES = FEMALES
2. MORPHOLOGY AND SYMPTOMS
    a. SYSTEMIC
        i. EXTREME EUPHORIA
        ii. DON'T SLEEP – IRRITABLE
        iii. OBSESSIVE BEHAVIOR
        iv. JERKY MOVEMENTS
        v. RAPID EYE MOVEMENT (10X NORMAL)
        vi. QUIVER IN VOICE
        vii. APPEAR TO AGE RAPIDLY
        viii. SKIN ULCERATIONS
    b. ORAL
        i. RAPID AND SEVERE DENTAL CARIES
        ii. PERIODONTAL DISEASE
        iii. ATTRITION/FRACTURE OF TEETH
        iv. TENDENCY TO FORM ABSCESSES…
        v. TOOTH LOSS

TREATMENT:

1. DRUG REHABILITATION PROGRAMS
2. RESTORE DENTITION

PROGNOSIS:

1. VERY POOR – ADDICTS ARE RARELY REHABILITATED
2. TOLL ON FAMILIES AND SOCIETY IS SEVERE
3. DENTAL WORK REQUIRED IS EXTENSIVE AND EXPENSIVE – TAXING PRISON BUDGETS/FACILITIES
4. TEETH ARE OFTEN LOSS WITH RESULTING MORBIDITY
5. EVENTUAL NEUROLOGIC DETERIORATION

PROLIFERATIVE VERRUCOUS LEUKOPLAKIA

DEFINITION: PROGRESSIVE ORAL EPITHELIAL PROLIFERATION WITH A HIGH DEGREE OF MALIGNANT TRANSFORMATION

ETIOLOGY: UNKNOWN

CLINICAL FEATURES:

1. EPIDEMIOLOGY
   - A. INCIDENCE - UNCOMMON
   - B. AGE - WIDE RANGE WITH PEAK IN 6th-7th DECADES
   - C. GENDER - 80% FEMALE
2. DISTRIBUTION - ANY SITE MAY BE AFFECTED
   - A. BUCCAL MUCOSA - 57%
   - B. GINGIVA - 54%
   - C. TONGUE - 54%
   - D. MULTIPLE SITES ARE OFTEN AFFECTED
3. MORPHOLOGY - WHITE, EXOPHYTIC, VERRUCOID PLAQUE
4. SYMPTOMS - MOST ARE ASYMPTOMATIC OTHER THAN THE PRESENCE OF THE LESION

MICROSCOPIC FEATURES:

THE SECTION SHOWS A WEDGE OF MUCOSA SURFACED WITH STRATIFIED SQUAMOUS EPITHELIUM. THE EPITHELIUM EXHIBITS A VERRUCOID PATTERN WITH HYPERPLASIA AND HYPERKERATOSIS. EPITHELIAL DYSPLASIA IS PRESENT. THE UNDERLYING LAMINA PROPRIA IS COMPOSED OF INFLAMED COLLAGEN.

TREATMENT: AGGRESSIVE SURGICAL EXCISION

PROGNOSIS:

1. 70% UNDERGO TRANSFORMATION TO CARCINOMA WITHIN 10 YEARS
2. LESIONS ON THE GINGIVA (29%) AND TONGUE (26%) WERE MOST LIKELY TO UNDERGO TRANSFORMATION
3. VERY PERSISTENT WITH HIGH RATE OF RECURRENCE
4. 40% DIE OF PVL-ASSOCIATED CARCINOMA

REFERENCES:

1. Silverman S, Gorsky M: Proliferative verrucous leukoplakia. A followup of 54 cases. Oral Surg Oral Med Oral Pathol 1997;84:154-157.
2. Fettig A et al: Proliferative verrucous leukoplakia of the gingiva. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;90:723-30.

OROPHARYNGEAL CARCINOMA

DEFINITION: MALIGNANT NEOPLASM OF THE OROPHARYNGEAL MUCOSA

ETIOLOGY:

1. TOBACCO USE
2. ALCOHOL USE
3. HUMAN PAPILLOMA VIRUS (HPV) INFECTION
    a. HPV-16
    b. 60% OF CASES
    c. E6 AND E7 PROTEINS INACTIVATE p53 AND pRB genes
    d. INCREASING IN FREQUENCY
    e. USUALLY IN NON-SMOKERS/DRINKERS
    f. CORRELATES WITH:
        i. ↑ NUMBER OF SEXUAL PARTNERS
        ii. ORAL-GENITAL SEX
        iii. FEMALE PARTNERS WITH ABNORMAL PAP SMEARS
        iv. GENITAL WARTS

CLINICAL FEATURES:

1. EPIDEMIOLOGY
    a. INCIDENCE - ↑ IN PAST DECADE
    b. AGE – 5^{TH}-7^{TH} DECADES (YOUNGER IF HPV)
    c. GENDER – M > F
2. DISTRIBUTION
    a. TONSILS – USUALLY ANTERIOR PILLAR
    b. BASE OF TONGUE - SILENT AND DEEP
    c. SOFT PALATE
    d. POSTERIOR LATERAL PHARYNGEAL WALL
3. MORPHOLOGY
    a. OFTEN HARD TO SEE CLINICALLY
    b. ENDOSCOPY OFTEN NECESSARY
    c. IMAGING ESSENTIAL
    d. FUNGATING, ULCERATIVE MASSES
    e. OFTEN LARGER THAN EXPECTED
4. SYMPTOMS
    a. ASYMPTOMATIC EARLY
    b. SORE THROAT
    c. OTALGIA (EAR ACHE)
    d. DYSPHAGIA (DIFFICULTY SWALLOWING)
    e. PAIN AND TRISMUS LATE
    f. METASTASIS TO LYMPH NODE IS OFTEN THE FIRST SIGN

MICROSCOPIC FEATURES:

1. 90% SQUAMOUS CELL CARCINOMAS
2. SOME ADENOCARCINOMAS
3. FEW LYMPHOMAS AND OTHER SARCOMAS

TREATMENT:

1. STAGING ESSENTIAL
2. EARLY STAGES – SURGERY AND/OR RADIATION

3. LATER STAGES – RADIATION AND/OR CHEMOTHERAPY

OROPHARYNGEAL CARCINOMA

PROGNOSIS:

1. OVERALL 25% SURVIVAL RATE
2. LYMPHATIC SPREAD GOES FROM SUPERIOR TO INFERIOR
3. UPPER PARAJUGULAR LYMPH NODES INVOLVED FIRST
4. HPV-RELATED TUMORS HAVE BETTER SURVIVAL
5. MORBIDITY FROM SURGERY, RADIATION AND CHEMOTHERAPY IS CONSIDERABLE

BISPHOSPHONATE-ASSOCIATED OSTEONECROSIS OF THE JAWS

DEFINITION: ORAL COMPLICATION OF BISPHOSPHONATE THERAPY

ETIOLOGY:

A. ADMINISTRATION (USUALLY IV – 93%, RARELY ORALLY – 7%) OF BISPHOSPHONATE DRUGS

B. EXAMPLES INCLUDE ETIDRONATE, CLODRONATE, TILUDRONATE, PAMIDRONATE (AREDIA), NERIDRONATE, OLPADRONATE, ALENDRONATE (FOSAMAX), IDANDRONATE (BONIVA), RESEDRONATE, AND ZOLEDRONAGE (ZOMETA)

C. BISPHOSPHONATES ARE USED FOR TREATMENT OF OSTEOPOROSIS AND BONE-DESTROYING CONDITIONS SUCH AS MULTIPLE MYELOMA, METASTATIC CANCER AND PAGET'S DISEASE

D. PRECISE MECHANISM IS UNKNOWN BUT APPARENTLY THEY BLOCK DISSOLUTION OF HYDROXYAPATITE, INHIBIT OSTEOCLAST FUNCTION, AND ARE CYTOTOXIC TO OSTEOBLASTS.

E. MOST CASES FOLLOW TOOTH EXTRACTION BUT SOME ARE ASSOCIATED WITH PERIODONTAL DISEASE, WEARING DENTURES AND IMPLANT PLACEMENT; 17& OCCUR SPONTANEOUSLY

CLINICAL FEATURES:

A. EPIDEMIOLOGY

1. INCIDENCE – UNCOMMON BUT INCREASING IN INCIDENCE; AFFECTS ABOUT 1/10,000 ON BISPHOSPHONATES

2. AGE – CHIEFLY ELDERLY ADULTS

3. GENDER – MALE = FEMALE

B. DISTRIBUTION

1. MANDIBLE = 65%

2. MAXILLA = 25%

3. BOTH JAWS = 10%

BISPHOSPHONATE-ASSOCIATED OSTEONECROSIS OF THE JAWS

C. MORPHOLOGY

1. IRREGULAR RADIOLUCENCY WITH SEQUESTRUM FORMATION
2. SLOUGHING OF OVERLYING MUCOSA

D. SYMPTOMS

1. MAY BE ASYMPTOMATIC FOR WEEKS TO MONTHS
2. PAIN
3. SECONDARY INFECTION
4. EXPOSURE TO SHARP EDGES OF EXPOSED BONE
5. LOOSENING OF TEETH
6. DRAINAGE
7. SINUS INVOLVEMENT AND PATHOLOGIC FRACTURE HAVE BEEN REPORTED

E. CLINICAL STAGING

1. STAGE 1 – EXPOSED/NECROTIC BONE IN ASYMPTOMATIC PATIENT WITHOUT INFECTION
2. STAGE 2 – EXPOSED/NECROTIC BONE IN PATIENT WITH PAIN AND INFECTION
3. STAGE 3 – EXPOSED/NECROTIC BONE IN PATIENT WITH PAIN AND INFECTION, AND ONE OR MORE OF THE FOLLOWING: PATHOLOGIC FRACTURE, EXTRAORAL FISTULA, OSTEOLYSIS EXTENDING TO THE INFERIOR BORDER

MICROSCOPIC FEATURES:

THE SECTION SHOWS A FRAGMENT OF NON-VITAL BONE. OSTEOCYTES, OSTEOBLASTS, AND OSTEOCYTES ARE ABSENT. HOWSHIP'S LACUNAE ARE ABSENT.

BISPHOSPHONATE-ASSOCIATED OSTEONECROSIS OF THE JAWS

TREATMENT: BASED ON RECOMMENDATIONS BY THE AMERICAN ASSOCIATION OF ORAL AND MAXILLOFACIAL SURGEONS:

A. INDIVIDUALS ON IV BISPHOSPHONATE THERAPY

    1. AVOID PROCEDURES INVOLVING DIRECT OSSEOUS CONTACT

    2. TREAT NONRESTORABLE TEETH WITH ENDODONTICS

    3. AVOID PLACEMENT OF IMPLANTS

B. INDIVIDUALS ON ORAL BISPHOSPHONATE THERAPY

    1. ELECTIVE DENTOALVEOLAR SURGERY IS NOT CONTRAINDICATED

    2. INFORM PATIENT ABOUT RISKS OF COMPROMISED BONE HEALING

    3. NO ALTERATION IN TREATMENT IF PATIENT HAS BEEN TAKING BISPHOSPHONATES LESS THAN 3 YEARS

    4. IF THERAPY IS MORE THAN 3 YEARS AND/OR PATIENT IS ALSO TAKING CORTICOSTEROIDS, CONSULT WITH PHYSICIAN AND CONSIDER DISCONTINUING THERAPY FOR 3 MONTHS AND RESUME AFTER BONE HEALING IS COMPLETE

C. PATIENTS WITH ESTABLISHED OSTEONECROSIS

    1. STAGE 1 – CONSERVATIVE CHLORHEXIDINE RINSES

    2. STAGE 2 – ANTIBIOTICS AND ANTIMICROBIAL ORAL RINSES

    3. STAGE 3 – SURGICAL DEBRIDEMENT/RESECTION WITH ANTIBIOTIC THERAPY

BISPHOSPHONATE-ASSOCIATED OSTEONECROSIS OF THE JAWS

D. PREVENTION

1. PHYSICIANS SHOULD REFER PATIENTS FOR A DENTAL EXAM PRIOR TO THERAPY
2. RISKS OF BISPHOSPHONATE THERAPY SHOULD BE DISCUSSED WITH THE PATIENT
3. THOROUGH DENTAL EXAM TO RULE OUT SOURCES OF INFECTION
4. INVASIVE PROCEDURES SHOULD BE COMPLETED PRIOR TO INITIATING THERAPY
5. PREVENTIVE DENTAL PROCEDURES SHOULD BE INSTITUTED
6. FOLLOW-UP EXAMS SHOULD BE DONE EVERY 3 MONTHS

PROGNOSIS: SOME RESPOND TO THERAPY WHILE SOME PROGRESS

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4. Guttenberg SA: Bisphosphonates and bone… what have we learned? Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;106:769-72.