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| **Bisphosphonate Therapy** | |
| **Bisphosphonates** | **Oral Bisphosphonate Theraphy:**  **Osteoporosis** |
| - **Inhibit osteoclast differentiation & recruitment**  - Induce osteoclast apoptosis  - May inhibit angiogenesis  - BON: First Case reports in 2003  - Rare; Most cases are in the jaws  - No clinical trial data  - Can be PO or IV  - Can be first or second generation drugs  - **Currently only the 2nd gen drugs are assoc. w/ BON**  2nd gen get incorporated into the skeleton, are very potent and have extended half-life (eg. Fosamax half life is 12 yrs) | - Fosamax (alendronate) po, 2nd, weekly  - Actonel (risedronate) po, 2nd  - Boniva (ibandronate) po or iv, 2nd, mo.  - Didronel (etidronate) po 1st  - Skelid (tiludronate) po 1st  **- Reclast**(zoledronate) –once yearly iv, 2nd, FDA approved 2007, infusion takes 15 minutes  - Incidence of BON in pts treated for osteoporosis is low esp. 1st gen & esp. po  🡪 ~0.7 per 100,000 pt-years use of Fosamax est by drug industry  -Increased risk of BON with PO drug with:  - Dental surgery (extractions most cases)  - Dental disease/infections  - Oral trauma  - Periodontitis  - Concomitant chemotherapy or corticosteroids  - Over 65 yo  - Tori or bony exostoses  - Diabetes  - Smoking alcohol use  - Poor OH  - Duration of drug over 3 years |
| - Normally:  - Osteoclasts repair microfractures and resorb foci with nonviable osteocytes  - With Bisphosphantes:  - Decrease in osteoclast fnct which allows microfractures to accumulate.  - Bone turnover slows so osteocytes that have exceeded their 150 day lifespan (ie. Die) are allowed to remain |
| - Indications:  - Osteoporosis  - Treat Paget’s disease  - Cancer: Slow metastases of several cancers (breast, prostate, multiple myeloma), and prevention of cancer treatment induced bone loss  - On the horizon: prevent metastases  - may have antitumor activity & may prevent bone metastasis  - mechanisms may include induction of apoptosis, inhibition of tumor cell invasion and angiogenesis, and tumor growth reduction | **IV Bisphosphonate Therapy:**  **Cancer** |
| - Treatment for Myeloma, breast and prostate cancer    - **Zometa**(zoledronic) iv, 2nd gen (same drug as Reclast)  - Aredia (pamidronate) iv, 2nd gen  - Bonefos (clodronate) po or iv, 1st gen    **- 94% of cases of BON assoc. w/ Zometa and Aredia**  - 85% of cases in myeloma pts  - Incidence for pts taking 2nd gen for cancer: 6-10% |

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|  | **Bisphosphonate Associated Osteonecrosis (BON)** |
| **Definition** | Etio:  - (60%)Exposed bone from invasive procedure  - (40%)Spontaneous  - (Small Number) After minor trauma to bony protuberances like torus |
| **Clinical Feat.** | - 2/3 in mandible, but maxilla or both jaws simultaneously  - 2/3 painful  - Exposed bone  - +/- pain  - +/- infection  - Infection can lead to fistula formation or fracture |
| **Radio** | - Early: before clinical, increased radiodensity of ALVEOLAR bone (crestal)  - Late: “**moth-eaten**” (ill defined) radiolucency or mixed (opaque sequestra in center if any) |
| **Histology** | - Necrotic bone is sclerotic w/ loss of osteocytes from the lacunae and peripheral resorption w/ bacteria  - Trabecular bone pattern can look like Paget’s disease  - Ostoclasts big and vacuolated |
| **Tx/Prognosis** | **- Conservative management: the goal of therapy is to stop pain and infection**  - ASx pts: Chlorhexidine rinse daily, smooth rough edges of exposed bone  - Sx pts: Chlorhexidine and Antibiotics  - Amoxicillin +/-metronidazole  - Clindamycin, azithromycin  - Iv antibiotics in hospital if the po fail  -Analgesics  -Minimal surgical debridement  -No role for HBO  -No real role for d/c drug in short term because of half life, but it is commonly done in practice |

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|  | **Osteogenesis Imperfecta**  **(OI)** | **Cleidocranial Dysplasia**  **(CD)** | **Osteopetrosis (Osteopetrosis)** |
| **Definition** | - Disorder of **collagen** maturation  - **Most common inherited bone disease but still rare**  **(1/8000 live births)**  - Spontaneous/Sporadic possible  **- A group of diseases (4 types)**  - Abnormal maturation of type I collagen (chromosome 7 or 17) | - **Osteoblastic** differentiation effected  - Genetic defect on chromosome 6  - Autosomal dominant  - 40% spontaneous  - Membranous bones predominately effected (ie. clavicles and skull), but also long bones (endochondral) | - **Osteoclasts** not function  - Rare, genetic  - group of diseases  - 2 major forms (infantile & adult) vary by severity & genetic origin |
| **Clinical** | - Bone fragility, blue sclera, altered teeth, hearing loss, long bone&spine deformities, joint hyperextensibility  - Craniofacial findings:  - Class III malocclusion due to maxillary hypoplasia , sometimes florid osseous dysplasia-like bone changes  - Teeth:  - dentinogenesis imperfecta primary & permanent teeth (opalescent teeth)  - Type I:  - Most common form and mildest  - Autosomal dominant  - Preschool fractures, adult hearing loss, joint hypermobility, bruising, blue sclera, - +/-opalescent dentin  - Type II: most severe form, often stillborn  - Type III: most severe form in live births surviving beyond perinatal period  - Type IV: mild –moderate form | - Clavicles:  - hypoplastic or aplastic, uni or bilaterally, increased shoulder mobility  - Skull bones:  - sutures and fontanels remain open, wormian bones develop in the suture lines, frontal and parietal bossing, large heads  - Hypertelorism  - High palate, depressed nasal bridge  - Absent maxillary sinus  - Teeth:  - Many unerupted perm & supernumerary teeth (often misshapen)  - Primary teeth may be retained into adulthood  - Absent cementum | - InfantileType:  - auto recessive  - marrow failure  - facial deformity (frontal bossing, broad face, hypertelorism, delayed tooth eruption, deafness, facial paralysis)  - no distinction btwn cortical & medullary bone on radiograph  - Adult Type:   - auto dominant  - axial and craniofacial skeleton  - may discover on dental radio  - bone pain - common |
| **Radio** | - thin cortex  - fine trabeculation  - osteopenia  - bowing of long bones  - multiple fractures  - wormian bones of skull |  | - infantile: Dense bone, sclerotic marrow spaces replaced w/ bone or fibrous tissue  - Adult: generalized increase in bone density, reduced sinus cavities in size, unerupted teeth are common |
| **Histo** | - Immature bone architecture  ie. Woven bone does not mature to lamellar bone |  | - sclerotic bone – NO marrow spaces |
| **Tx/Prog** | - Tx: None  - Manage fractures  - tooth attrition and tooth loss  - Prognosis: variable w/ type of OI | - Tx: none  - Function well  - Extrusion of perm. teeth orthodontically to increase fnct, encourage growth of the alveolus, and correct vertical growth of the jaws to prevent class III occlusion | - Tx: none  - Tx complications of osteomyelitis and fracture, bone marrow obliteration, etc. |

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|  | **Paget’s Disease of Bone** | **Idiopathic Osteosclerosis** | **Focal Osteoporotic**  **Bone Marrow Defect** |
| **Def** | - Common (1% of men over 45 yo), esp in England  - Acquired bone metabolism problem of **increased bone turnover** (increased resorption and deposition)  - Etiol: ? | - common (5% of population)  - Focal radiodensity of the jaw which is **not** inflammatory, dysplastic, neoplastic, nor manifestation of a systemic disease  - Etiol: ? | - Area of normal bone marrow in the jaws which is big enough to produce a radiolucency on plain film  - Importance: can confuse w/ pathology  - Etiology: ?? Marrow hyperplasia secondary to healing/bone fill |
| **Clinical Feat.** | - Over 40 yo, white men  - Sx vary from subclinical to pain esp. near joints, bowing of long bones, enlarging skull, some monostotic (malignant), most polyostotic  - 17% of Paget’s pts have  - jaw (body and alveolus) expansion  - max enlarges>mand (lion-like)  - sinus obstruction  - chronic and slow progression  - Dx:  - Blood tests: inc alkaline phosphatase (marker of osteoblast activity), normal calcium, normal phosphorous  - Urine: inc hydroxyproline, and other markers of bone resorption   - Biopsy usually unnecessary | - Teens and 20s  - Asx: Incidental finding on radio  - No jaw expansion  - Dx:  - radio, hx  - biopsy **rarely** needed  - follow with radio periodically –should have minimal growth and sometime even regression | - 70% posterior mand  - Women 3:1  - Asx: Incidental finding on radio  - No jaw expansion  - Freq at healed exo site  - Dx: radio, +/-biopsy |
| **Radiographic** | - Early phases “osteoclastic”(osteopenia): decreased density and lucencies esp. of skull (osteoporosis circumscripta)  - Later “osteoblastic” stages: patchy sclerosis becomes confluent (“**cotton wool**”), teeth hypercementosis | - Well defined  - Rounded or triangular  - Uniformly Radiopaque (no lucent)  - mm-cms  - Rt apex or inter-radicular area 80%  - **Rare** rt resorption/tooth movement (but documented) | - ill-defined borders  - Lucency  - mm to cm(s)  - Fine trabeculations (decreased bone density, not “lesion”)  - Biopsy proved it to be filled w/ normal bone marrow |
| **Histology** | - Very vascular fibrous CT replaces marrow  - Basophilic reversal lines=junction btwn alternating resorptive and formative phases of bone (A.K.A. “jigsaw” or “mosaic” pattern)  - Active phase: Alternating resorption (osteoclastic) & formation (osteoblastic) activity on same bone trabeculae  - Less active phases (final stage): more sclerotic , dense bone w/ prominent reversal lines | - Lamellar bone and marrow | - normal hematopoietic bone marrow |
| **Tx/Prognosis** | - None  - If severe course, parathyroid hormone antagonists (blocks osteoclastic activity)  - Bisphosphonates (eg.Skelid, 7 Bisph. drugs currently indicated)  - Osteosarcoma may develop (1-13%)—usually lower extremity or pelvis  - Benign & malignant giant cell tumors – rare but jaws most commonly involved | - none | * None (no assoc w/ systemic disorders) |

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|  | **Langerhan’s Cell Histiocytosis**  **= Histiocytosis X**  **= “Eosinophilic granuloma”** | **Central Giant Cell Granuloma (CGCG)** | **Cherubism** |
| **Def** | - Monoclonal proliferation of histiocyte-like cells (Langerhans cells w/ a polyclonal infiltrate of multinucleated giant cells, eosinophils, and other chronic inflammatory cells)  - Langerhans cells are antigen presenting mononuclear cells  - Spectrum of diseases | - Tumor? “Reparative”?  - Osteoclast 🡪 tumor 🡪 over-resorption of bone | - Genetic disease; **syndrome**  - autosomal dominant  - chromosome 4  - variable expressivity  - Spontaneous/Sporadic possible |
| **Clinical** | - Children (50% < 10 y.o)  - Bone lesions most common manifest.  - Jaw lesions (10 -20%) in pts over 20 y.o  - **Eosinophilic granuloma of bone** (mono- or polyostotic): radiolucency in bone  - **Chronic disseminated histiocytosis:** bone, skin, viscera (“**Hand-Schuller-Christian disease”-** triad of bone lesions, exophthalmos, diabetes insipidus)  - **Acute disseminated histiocytosis**: prominent visceral, bone marrow, skin involvement—usually **infants**(“**Letterer-Siwe disease**”); Leukemia-like clinical feature | - Mand anterior to 1st perm M  - Frequently cross the midline  - 60% < 30 y.o but, wide age range  - Female often  - Asx, slow growth  - Aggressive lesions:  - rapid growth, expand bone, pain, rt resorption, paresthesia, perforate cortical plate, and produce an associated soft tissue lesion (ulcer or mass), high recurrence rate | - Posterior body and ramus  - Begin to see changes 2-5 y.o and then stabilize at puberty  - **Bilaterally** develops bone lesions  - Painless expansion  - Expanded mandible & orbital bone produced cherubic like facial appearance (angelic chubby cheeks, eyes turned twd heaven)  - Severe cases can expand alveolus and displace teeth or block eruption  - maxilla involved in severe cases as other craniofacial, long bones  - DX: radio, family hx, biopsy, rule out hyperparathyroidism and other syndromes with CGCG |
| **Radiographic** | - Punched out radiolucencies  - well defined w/o corticated rim  - Teeth may appear "floating in space"  - absolutely no bone around a tooth  - sometimes ill-defined esp. in the jaws  - alveolar bone destroyed from crest 🡪mimicking severe perio +/-gingival ulcers or masses, **or**  **-** body of mandible/maxilla 🡪 mimicking periapical inflammation | - Unilocular or multilocular radiolucencies  - Well defined, corticated or non-corticated  - If multifocal: rule out cherubism and hyperparathyroidism | - **bilateral**  - multifocal, multilocular radiolcencies (unilocular possible) |
| **Hist** | - Langerhans cells (CD1a+, S100 +, peanut agglutinin +, Birbeck granules) plus eosinophils, MNGC, plasma cells and lymphocytes | - MNGC (probably osteoclasts)  - Spindle cell background  - Hemorrhage | - **Central giant cell granulomas (CGCG)** |
| **Tx/Prognosis** | - Bone lesions:  - surgically removed (curetted) if accessible  - if not RT or steroid injections 🡪 very good prognosis  - Chronic disseminated: chemo  - Acute disseminated: chemo w/ more guarded prognosis | - Surgery (curettage 🡪20% recur and respond well to curettage)  - Aggressive lesions: intralesional or systemic chemotherapy w/ corticosteroids or calcitonin respectively  - Very good prognosis overall | - ?  - Often remission and involution after puberty, sometime grotesque deformities remain  - Surgical curettage good for some/aggravating to others |

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|  | **Traumatic Bone Cyst**  **= simple bone cyst**  **= solitary bone cyst**  **= “Idiopathic bone cavity”** | **Aneurysmal Bone Cyst (ABC)** | **Hemangioma of Bone**  **(Intrabony vascular malformation)** |
| **Def** | - Empty or fluid filled cavity in bone  - Misnomer: not true cyst  - Etiology? Trauma-hemorrhage theory  - trauma without fracture  - hemorrhage that does not reorganize into bone  - Reported in almost all bones of body | - Intrabony accumulation of blood.  - Reactive fibrous CT and bone  - Misnomer: not true cyst  - Etiol: ? Primary lesion or disrupted vascular dynamics in a preexisting bony lesion? |  |
| **Clinical** | - Mandible>>>>mx  - Pm/molar body of mand  - 10-20 y.o, rare over 35 y.o  - slight male > female  - painless, no expansion usually  - Rarely, pain, paresthesia, cortical expansion  - DX: radio, cl, and “biopsy” – empty bone cavity found at surgery | - Posterior mandible  - Children and young adults  - More common in long bones, vertebrae  - Pain, rapid swelling, ballooning or “blow out” of bone  - Rarely paresthesia, teeth mobility and root resorption, maxilla involving sinuses | - Mandible  - Usually 10-20 y.o, (HOWEVER, **adults can manifest vascular Malformations!!**)  - Females  - Sx variable (asx, pain, swelling)  - Tooth mobility  - Can expand jaw  - Spontaneous bleeding from sulcus, venous or a/v malformation  - if high flow, may feel pulse  |
| **Radiographic** | - Unilocular Radiolucency  - variable definition (well to poorly defined)  - 1-10cm  - **scallops** btwn rts of teeth w/o devitalizing or resorbing or moving  - Very very rare multilocular | - Uni or multilocular  - well defined to poorly defined  - Non-corticated Radiolucency  - Cortical expansion and thinning  - +/-small calcifications | - Multilocular or less often unilocular radiolucency  - **Sunburst** periosteal reaction (cross-section) |
| **Hist** |  | - Blood filled spaces lined by fibrous CT and MNGC  - Calcifications in wall |  |
| **Tx/Prognosis** | - Surgical exploration of cavity (curettage; bleeding into cavity) often causes repair within 6 months  - Rare recurrence or persistence | - Surgery -“blood soaked sponge” curetted out and dark venous blood fills the cavity  - Surgical defect heals within 6 months  - Cryosurgery may be needed | - MAKE THE DX!!!! Pose tremendous surgical risk!!  - Reason to aspirate all intrabony lesions prior to surgery  - No extractions in the area  - No extractions without a radiograph  - Angiography delineated flow and feeder vessels  - May need embolization prior to resection |

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|  | **(Benign) Fibro-osseous Lesions of the Jaws (BFOL)**  - Def: group of pathologic processes in which fibrous CT and mineralized product replaces bone  - Clinical and radiographic features +/-histology is required to make a specific diagnosis | | |
|  | **Fibrous Dysplasia (FD)** | **Cemento-osseous Dysplasias (COD)** | **Ossifying Fibroma (OF)** |
| **Def** | - **Sporadic postzygotic gene mutation** causes tumorous swellings of bone(s) and possible skin & endocrine abnormalities  - Severity of disease likely depends on the point in fetal life that the mutation occurs (undifferentiated stem cells vs. skeletal progenitor cells, etc.) | - All types occur **only** on the jaws and in the tooth bearing areas (alveolus, not body of jaws, etc.)  - **3 Types**: Periapical, Focal, Florid COD  - Represent variants of the same process, and probably a progression of disease | - Similar in radiographic and histologic appearance to CODs but OF is a **true neoplasm**  - Historically reported w/ focal COD |
| **Clinical Feat.** | - **Monostotic** FD (85%):  - single bone or craniofacial bones  - jaws most common bone  - mx>md  - Dx in teens  - slow growing, painless swelling  - mx often involves zygoma, sphenoid, etc=craniofacial FD  - teeth displaced but **not** mobile  - **Polyostotic** FD:  - 2 or more bones  - long bones subject to fracture and resultant deformity  - **Jaffe-Lichtenstein syndrome**:  PolyFD +café au lait (skin lesion)  - **McCune-Albright syndrome**:  PolyFD +café au lait + multiple endocrinopathies | - Asx, no expansion  - **Periapical COD(PACD)**  - **Anterior mand only**  - **ave 40 yo AA women (30-50 y.o)**  - **Periapical area**  - **Focal COD**  - Posterior mand often  - ave 40 y.o AA (+ some Caucasian) women  - Any area of the jaw  - **Florid COD**  - Multifocal involvement  - **ave 50 yo AA female (40-60 y.o)**  - often all 4 quadrants  - Bilateral and symmetric  - **VERY** radiopaque  -Dx: hx, radio findings, and pulp testing  - **AVOID biopsy**! (b/c infection of bone) | - Posterior mandible (PM-M region)  - Females in 20s - 30s  - jaws and craniofacial bones  - expand bone  - grow slowly in a centrifugal way  - Rare, pain and paresthesia |
| **Radiographic** | - Poorly defined uniformly radiopaque  - “Ground glass” trabecular pattern (poorly calcified & haphazardly arranged)  - Pdl narrowed, lamina dura blends  - Mx: floor of sinus displaced superiorly or sinus obliterated, extension into adj bones (occiput, sphenoid, orbit, frontal, etc.)  - Md: inf alv canal displaced superiorly, lower border of md bowed | - Early lesions: radiolucency at the apex of vital teeth (radiographically identical to pa granuloma/cyst)  - Later lesions: adj teeth develop radiolucencies and the lucencies fuse to form a continuous lucency at apex of several teeth  - Mature lesions: mixed w/ radiolucent rim and a radiopaque center, w/ PDL intact & no fusion to the tooth 🡪 **“cotton wool”** | - Early: well defined unilocular radiolucency or mixed lesion, varying amounts of radiopacity, some have sclerotic border  - Later: become large, cause rt divergence and bow the inferior border of mandible, increasing radiopacity in center? |
| **Histo** | - Haphazard mix of immature (woven) bone in fibrous ct stroma.  - **Metaplastic bone**—arises directly from stroma w/o osteoblastic rimming  - **“Chinese character-like” trabeculae**: irregularly shaped trabeculae that do not connect w/ each other | - All three CODs have same histology  - “Fibro” fibrous connective tissue  - “osseous” metaplastic immature woven bone (**not** rimmed by osteoblasts)  - ”cemento” cementum-like droplets (parvicellular)  - Proportion of each element relates to the degree of maturity of the lesion | - very similar if not identical to cods  - “Fibro” fibrous connective tissue  - “osseous” immature woven bone  (**may be** rimmed by osteoblasts)  - ”cemento” cementum-like droplets (parvicellular)  -Usually submitted to lab in one piece b/c at surgery it shells out (potato-like) mass |
| **Tx/Prognosis** | - Cosmetic shave down; best left until skeletal maturity when lesions usually stop enlarging  - Contraindication: Radiation due to risk for osteosarcoma | - Tx: none  - Contraindication: surgery of all kinds due to risk of osteomyelitis, esp. Florid COD  - Dental care aim to avoid tooth loss & surgical needs  - May develop traumatic bone cysts in lesions | - Enucleate, rare recurrence  - **Must treat** because it is a neoplasm with capacity to destroy large segments of bone |

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|  | **Osteoma** | **Gardner Syndrome** | **Osteoblastoma** | **Cementoblastoma** |
| **Def** | - True neoplasm?  - **Restricted to craniofacial skeleton** | - Genetic  - Auto Dom  - chromosome 5  - 33% spontaneous, rare  - Multiple osteomas  - Part of “**familial colorectal polyposis”** | - Rare benign bone neoplasm | - Neoplasm of cementum (really an odontogenic tumor)   |
| **Clinical Feat.** | - Body of mandible or condyle or paranasal sinus  - Young adults  - Asx until large  - Periosteal (on bone) or endosteal (in bone)  (can cause bone expansion) | - GI polyps: colon mostly  - Osteomas: At puberty craniofacial begin, any part of jaws but often **angle of mandible**  - Dental: odontomas, supernumerary teeth, & impacted teeth  - Skin: epidermoid cysts | - Post. Md  - often < 30y.o  - Slight male > female  - **VERY painful**, pain usually is not relieved by aspirin  - Extragnathic and jaws | - Most perm posterior MD  - < 30 yo  - **pain and swelling (66% pts)** |
| **Radiographic** | - Well defined radiopacity  - Small lesions indistinguishable from idiopathic bone sclerosis or sclerosis secondary to inflammation |  | - Mixed w/ varying amount of patchy opacity  - Well ill-defined  - **> 2cm** | - Radiopacity fused to root(s)  - some **root resorption**  - thin radiolucent rim at periphery  - loss of normal PDL |
| **Histology** | -Normal bone with variable amount of fibro-fatty marrow |  | - Bone formation from large osteoblasts  - Very vascular | - Very similar to osteoblastoma, but cells are cementoblasts and lesion is fused to cementum of tooth |
| **Tx/Prognosis** | - Small: watch. **Slow** continued growth is normal  - Large: cosmetic or functional problems are surgically removed | - Prophylactic colectomy b/c colonic polyps are pre-malignant | - Local excision  - Rare recurrence | - Removal of lesion and root(s) or tooth  - Does not recur if completely removed |

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|  | **Osteosarcoma (OS)** | **Chondrosarcoma (CS)** | **Metastatic**  **Malignancies**  **to the Jaws** | **Ewings Sarcoma** |
| **Def** | - **The most common primary of malignancy of bone under 40 yo**  **(vs. >40 y.o. multiple myeloma)** | - Half as common as osteosarcoma | - The **most common cancer** found **in bone** is **metastatic carcinoma**  - Jaws: most common carcinomas mets are breast, lung, thyroid, prostate and kidney | - Relatively common primary malignancy of bone (1=os, 2=cs, 3=Ewings) |
| **Clinical Feat.** | - md=mx  - long bones: 10-20 y.o  - jaws ave 33 y.o.  - Sx of swelling and pain  (also loose teeth, paresthesia) | - mx and maxillary sinus>>md  - long bones: over 50 yo  - jaws: wide age range  - Sx of swelling  - Pain is **unusual**!  (also loose teeth, nasal, sinus or ocular symptoms) | - MD>>>MX  - elderly  - sx vary from Asx to pain, swelling, paresthesia, loose teeth | - md>mx  - teens  - Whites>>>>Blacks  - **VITAL teeth!!!**  - any bone, jaws are uncommon  - paresthesia, loose teeth, pain, swelling, sometimes soft tissue breakthrough, fever, elevated white cell count and ESR (mimicking infection, osteomyelitis on radio) |
| **Radiographic** | - **poorly** **defined borders**  - vary in density (radiolucency, radiodensity or mixed – “**Moth eaten**”)  - Spiking root resorption  - **sunburst** periosteal rxn to cortical infiltration  - Symmetric **widening of pdl** around involved teeth | - same as Osteosarcoma | - Most often ill defined radiolucency  - Sometimes resembles perio dx or cyst or widens pdl  - Some present with mixed lucent/opaque (breast and prostate) | - Ill defined radiolucency  - **onion skin** periosteal reaction |
| **Hist** | - pleomorphic round or spindle cells producing bone  - May also produce fibrous ct or chondroid | - cartilage (condyle only, if somewhere else, malignant)  - amount of cellular atypia and pleomorphism varies w/ grade of tumor |  | - undifferentiated round cells  - dx usually requires special studies for immunophenotype (e.g.CD99) or genetic alteration (t 11,22) |
| **Tx/Prognosis** | - Improved survival w/ pre and post op chemotherapy  - Radical surgical resection  - 30-50% survival  - Local disease control is usually the problem (local recurrence) more often than distant mets (lungs and brain) | - Radical surgical resection  - Chemo **only used as last resort** in unresectable tumors  - Prognosis depends on size, location and tumor grade  - Survival data poor – but sure of late recurrence (15 yrs or more), so the pt must be followed for lifetime  - local disease control is usually the problem (local recurrence) much more often than distant mets | - poor prognosis  - stage IV disease  - <1 year survival unless solitary met (tx with surgery or RT) | - surgery and chemo, +/-RT  - The more distal the tumor(s) the better the prognosis  - Overall prognosis much improved w/ multimodality tx to 40-80% survival |