sheet:#14

oral pathology

heba alkhwaldeh& rana selehat

Last lecture in this semester no oral pathology FOREVER :D

**Secondary Hyperparathyroidism:**

As a result of chronic renal failure, rickets and osteomalacia that stimulates parathyroid hormone secretion and lead to adenoma in the parathyroid gland

**Paget's disease of bone:**

Increased activity of osteoclasts followed by increased activity of osteoblasts with abnormal and disorganized bone remodeling leading to enlargement of bones

- affecting one or more bones

Etiology:

Unknown, but there is genetic susceptibility

More common in north Europe and Britain than in our region, it is thought that we have slow viral infection, and some studies suggested that measles and respiratory syncytial virus are responsible for the disease

3 Phases:

1- Osteolytic activity: bone resorbtion

2- Mixed Osteolytic and osteoplastic

3- osteoplastic or sclerotic: born formation

- Usually males above 40 years of age

- Predominant in the axial skeleton: spinal cord, femurs, ribs and skull

- lead to deformities in the spine, legs, joints "arthritis"

- Deformities in bones with susceptibility to fractures in the Osteolytic activity phase

- Enlargement of the skull and facial bones, in 20% of patients jaws affected specially the maxilla more than the mandible

- Thickening of the cortical plates

- The foramina of the cranial nerves and vessels will be compressed and patients will complain of headaches, visual and hearing loss, facial paralysis and anesthesia, spinal cord compression affecting lower extremities

Radiographically:

Cotton-wool appearance

- happening in the maxilla more than the mandible (2:1)

- Alveolar process enlargement, maxilla is protruded, retroclination of teeth and malocclusion

- bone prominence lead to incompetent lips ,illfiting the dentures

- Spacing of teeth

- flattening of the palate

Early stage:

Osteolytic activity and vascularity is increased: extraction of teeth at this stage will result in hemorrhage

Latter stages:

Sclerotic: difficulty in extraction that may result in fractures, dry socket, osteomyelitis

Radiographically:

Generalized hypercementosis

Clubbing of the roots sometimes root resorption

Loss of lamina dura (susceptible for infection )

Ankylosis

Histopathology:

Bone resorption and replacement or formation, where the remodeling activity is **repeated**

-Early osteoclastic phase: fibrous vascular stroma

-mosaic of bone "reversal lines"

Reversal lines: Indicate junctions where there has been reversal of osteoclastic resorption to osteoblastic deposition

* bone scan can help in the diagnosis
* biochemistry: increase in the alkaline phosphates with normal calcium

Complications:

In the initial stage, vascularity is high and when multiple bones are affected this will increase the load on the heart and arteriovenous shunts may form and tachycardia will follow

1-15% could develop osteosacoma

The giant cell lesions that were discussed before are the aneurysmal bone cyst , and Cherubism and brown tumers,, and today we will continue and talk about another giant cell lesions which are the Central giant cell granuloma lesion and the giant cell tumers.

 🞾 **Central giant cell granuloma**
This lesion is *less aggressive and destructive* in when in happens in the maxilla or the mandible compared to when it happens in another bony parts and it happens in the maxilla and mandible *more* than it happens in other parts in the body, so this lesion does not only affect the maxilla or the mandible it can also affects other parts in the body .
The majority occurs in young patients *less than 30* years of age and more in females , and affects the *mandible more* (75% of the cases ) than it affects the maxilla ,and especially in the anterior region of the mandible – anterior to the first molar - .less common than peripheral.
❖**Clinically** , we should remember the counter part of this lesion which is the peripheral giant cell granuloma , the Central is *less common* from the Peripheral giant cell granuloma , but its one of the lesions that oral surgeons can see as a cause for an expansion in the maxilla or the mandible .
It causes painless expansion with possible spacing between the teeth in the affected area .
🟋 In central giant cell granuloma when you take a radio graph you will usually see a multiloculer radiolucency with bone expansion , it can also be uniloculer, and you also might see in the aggressive cases a resorption in the roots of the teeth or a perforation of the bone or displacement of teeth .

The dr talked about an x-ray in the slides and he asked when you see such a case what would you think ? , the answer was that you might think that it is a periapical lesion with external root resorption so maybe its a radiculer cyst and you don’t think of central giant cell granuloma , that’s why you always have to check the vitality of the tooth – the central giant cell the teeth will be vital - and explain the radiolucent lesion and take a biopsy if needed ,, in the case of this x-ray we don’t take a biopsy immediately rather we make a root canal treatment and then wait if the lesion did not resolve and the size of it increased then you take a biopsy . "We always have to put in mind that we have another diagnosis "

**❖Histopathology** of central giant cell , we will see multiple multinucleated giant cells osteoclast like distributed in background of a fibrovasculer stroma -fibroblasts ,endothelial cells and blood vessels- , and those giant cells are variable in size ,shape, color and number.

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🞾 **Tori or torus**
They are localized benign hyperplasia of the bone or what so called localized exostosis , so its not something abnormal because if we took this torus and examine it under the microscope we will see a normal bone histology but the onlt thing is that it is an excess bone .
 Tori can happen in the palate and we call it **Toru palatinus** or in the lingual part in the mandible and we call it **torus mandibularis** .

**❖Etiology** of tori is unclear , one of the causes is the *genetic factors* and *environmental factors*, the environmental factors include the malocclusion , bruxism because these can cause stresses on the bone and stimulate bone formation or exostosis .

**🞛Torus palatinus** , we usually see it in the second decade of life ,and gradually increase in size with age – some books say that when the teeth are lost the size of the torus will decrease- , they can be rounded or multinodular , pedunculated or sessile and it covered with a thin mucosa , they can become very large , about 15 % of our patients might have the torus palatinus with different sizes .
**🟉The complications** of the this tori is when we want to make a denture like we took in prostho , it can also interfere with speech if the size was large , sometime the torus might have trauma from the mastication process and since it covered by a thin mucosa so we will have an ulcerations that are difficult to heal , it can also be a site where food collect so it can cause Halitosis and interfere with the oral hygiene.
**🟉The management** is that it can be easily surgically removed by simple excision , and when you give it to the lab the results will be a normal boney tissue.
**🞛Torus mandibularis** is *more common* than the palatinus about 25% , it happens lingual to the premolars and usually bilateral , but if you saw a unilateral lesion then you should be afraid a little bit because it can be something else not a torus maybe it can be a tumor or a cystic lesion or giant cell lesion , But when you see a bilateral lesion you think of torus mandibularis immediately.
🟉Torus mandibularis has different sizes and shapes it appears the occlusal radiographs and can be easily diagnosed because we will see a normal bone , usually multiple lobules will appear on the surface,, and it has the some complications, like causing a problem when we try to make a denture , it can also restrict the tongue movement of it's size was large , it can also interfere with the oral hygiene and causes halitosis and its also susceptible to trauma and ulcerations as in torus palatinus and its *management is the same* for the torus palatinus .

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🞾  **Exostosis**
It is a bone over growth that can be seen on other sites – not the site of torus palatinus or torus mandibularis - , it can be seen in the buccal side of the maxilla and the mandible ,and sometimes can be seen in the palatal side especially of the molars. The percentage of exostosis among our patients is 10%.
❖**The etiology** of the exostosis in unclear , so says its from the stresses from the teeth so the bone grows more , and other say that its due to gingival inflammation and due to that inflammation the periosteum form a new bone .
❖**Histopathology** , is a normal bone that is either all cortical bone if the exostosis is small in size or cortical and cancellous bone if it was large in size .

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🞾**Bone tumors** ,are of many types and we will not discuss them all , these bone tumors include , *osteoma ,osteosarcoma , chondroma , chondrosarcoma , myeloma , hemangioma, or metastasis of other tumors to the maxilla or the mandible.*

🞾 **Osteoma**
 is a benign slowly growing bone tumor , it can be single or multiple , if it was multiple we should think of Gardner's syndrome , the osteoma can be superficial (tebrez la barra el bone) or intraosseous ( tedkhol la jowwa el bone ) , it appears on the x-ray as a radio-opaque protrusion .

🞛**Gardner's Syndrome** : is an autosomal dominant disease, the patient will have supernumerary teeth and unerupted teeth , and have multiple osteomas , colon polyps and skin lesions (including [epidermoid cyst or sebaceous](http://en.wikipedia.org/w/index.php?title=Epidermoid_cyst%3D_sebaceous&action=edit&redlink=1) cyst, fibromas ) .. the colon polyps in these patients are hundreds of polyps and are potentially malignant and they say that if you leave the patients with these polyp then all of them will have colon cancer (100%), so these patients must have colectomy.
🟉So if a patient came to you with multiple bony hard swellings and on the x-ray they appear radio-opaque , and upon biopsy they where normal bone – because osteomas are benign tumor so we will have compact with cancellous bone that is normal - then you have to think in Gardner's syndrome and do further investigations and ask the doctors to do colescopy to make sure that the patient does not have colon polyps .

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🞾**Osteoblastoma** , remember that this tumor is painful and resembles the cementoblastoma .not attached to bone

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🞾**Osteosarcoma**
is a malignant tumor , remember that this tumor makes widening of the periodontal ligament , and on radiographs it makes what so called **"Sun-ray "**appearance , and since it is malignant then we'll have the malignant features from pain ,loosing , paresthesia, trismus and if it was in the maxilla it can make nasal obstruction, in the radiograph some can be osteolytic or osteoclastic or sclerolytic , is the Sun-ray appearance "ya3ni el bone trabecule bekoono wa2feen "and also the widening of the periodontal ligament .
🟋So if you saw a widening of the periodontal ligament in more than one tooth you write the osteosarcoma as one of the differential diagnosis even if the other symptoms did not appear yet ,, of course there are other causes for the widening of the PDL like the periodontitis , periapical lesion like abscess …. (diseases that causes widening of the PLD : scleroderm )
❖**The histopathology** of osteosarcoma pathology and this tumor is malignant so you know that .

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🞾**Chondroma and chondrosarcoma** it can happen in the maxilla or the mandible in the coronoid and the condyle region .

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🞾**Giant cell tumor**
It is one of the giant cell lesions , you have to differentiate between this tumor and the central giant cell granuloma and other giant cell lesions.. is there anything different in this tumor histopathologically or clinically **?** or in the places where is occur **?** .. the central granuloma happen in the maxilla and mandible more than other parts but can also occur in other parts and so does the giant cell tumor but more in other parts than in maxilla or mandible so both can happen anywhere so we don't depend on that .. But the giant cell tumor is *a malignant lesion* and does metastasize and its locally invasive and destructive, but the central giant cell granuloma does not metastasize it can be aggressive but not as much as the tumor if there was local invasion and destruction we should think more in the tumor ,, In histopathology there is no difference between them that we can rely on so don't depend on histopathology .
The patients in giant cell tumor are older age group while in the central they were young age less than 30 years .

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🞾**Multiple myeloma**
It is a tumor from the differentiated B-lymphocyte or the plasma cells , it can affect more than one site , but there are some cases where is occurred solitary-just one lesion- and in these cases in can happen in the maxilla or the mandible either in the bone or the soft tissue –gingiva- .
❖**The diagnosis** depends usually on radiographs where the dr see multiple fossi of bone destruction in the skeleton , they also analyze the blood and find out that it has a lot of immunoglobulin – antibodies which are products of the proliferating plasma cells - , in the urine they also find abnormal proteins are known as *‘monoclonal light chains*’ and are sometimes referred to as "**bence jones protein**" which are immunoglobulin light chains but in the urine.
🟋The patient will have multiple fossi of bone destruction in the skeleton like the skull and these will have the appearance of "**Pepper –pot skull** ") , they appear as well-defined round or oval punched out radiolucencies .This lesion is associated with pain , and since this destruction is within the bone marrow then we will have the same symptoms of leukemia like anemia and susceptibility to infections because the bone marrow is replaced with these plasma cells which are non-functional , and also we will have hypercalcemia – since we have bone destruction- so we will have strawns in the kidney , and we will also have proteinuria .
🟋This multiple myeloma happens especially in the bones with red marrow so we don't see it a lot in long bones rather in other bones like maxilla and mandible and might lead to pathological fracture.
🟋Multiple myeloma is associated with Macroglossia as an oral feature of this tumor and the reason behind that is amyloidosis – we will have amyloid deposition in different organs – including the tongue , macroglossia can be seen in 10-40% of patients having multiple myeloma.
❖**Histopathology** , is unique since we have sheets of plasma cells in the bone marrow or in the region affected .
Blood smear will show a lot of plasma cells.

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🞾**Amyloidosis**
It is extracelluer deposition of amyloid protein in different part of the body like the heart and the kidney and eventually leads to failure of these organs . It's of two types , primary and secondary ,, *primary* is the one that is associated with multiple myeloma ,, *secondary* is associated with chronic inflammatory diseases like the rheumatoid arthritis or malignancies .
🟋**Oral features of amyloidosis** is *macroglossia* ,, *petechia* as we saw in multiple myeloma there will be a problem in the blood cells including the platelets so they will have petechia, and amyloid might also deposit in the salivary gland and that result in *xerostomia* and dry mouth .

🟋**How to confirm diagnosis** **?** , If someone have macroglossia one of the differential diagnosis is the amyloidosis so we take a biopsy and in the H&E stain we will see eosinophilic hyaline homogenous material which is the amyloid ,, how to confirm that this is amyloid ? we stain it with a special stain called "**Congo Red** **"stain** and under polarized light the amyloid will get an apple green color , so if the patient was not diagnosed with multiple myeloma yet then it can help in the diagnosis process

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🞾**Metastatic tumors**
 We might have metastasis especially from breast , bronchus ,kidney to the maxilla or the mandible especially the mandible , we might have metastasis within the bone that will appear as radiolucency most of the time or as radio-opacities within the bone and make the features of a malignant tumor , like resorption of the bone resorption of the roots ,pain and paresthesia ,perforation and ulceration if it goes to the soft tissues .
🟉Sometimes metastasis tumors can make radio-opaque lesions – osteosclerolytic lesions-

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🞾**Hemangioma**
 It can affect the bone , its actually not hemangioma because it does not disappear with age so it actually a vascular malformation so its called **Arteriovenous malformation of the jaw** and another name is the Central hemangioma but the first one is better. The name means that there is a lesion that is composed of blood vessels and these vessels diameter is large "arteries and veins " and since we have arteries then the blood flow is high so "yemken ykoon feeha 5o6ora eza mathalan during extraction fata7na 3alaih w sar fe bleeding kteer "
🟋Arteriovenous malformation can affect the maxilla or the mandible and other bones , **Clinically** it is a painless swelling if you use a stethoscope you will hear pulses because we have arteries , and in the affected area the teeth will be loosened and we can see blood coming from the sulcus around the teeth, the answer is during extraction we will have very sever bleeding and the patient can die .
**🟋In** **radiograph** we will have osteolytic lesions that can be multiloculer or uniloculer ,we might see resorption of the roots in that area.
Management can be little bit difficult , so if you saw any radiolucent lesion under the teeth and you are not sure what that lesion is then don't extract the teeth.

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🞾 **Orofacial granulomatosis**
 A considerable amount of patients that we will see will have an enlargement of the lips or the cheeks for an unknown reason , at the beginning it is recurrent – come and go –and then is become persistent .
**❖Histopathology** ,if we took a biopsy then we will see granulomas ( non-caseating granulomas).
**❖Clinically** , it’s a swelling that is at the beginning is recurrent then it becomes persistent.
🟋When you these two features in a patient then you have to think of certain *systemic diseases* like **Crohn's disease**- which is a type of inflammatory bowl disease- so you have to exclude that disease so we ask another specialist to take further investigations and biopsy, also like **Sarcoidosis**
🟋If *you did not found any of these systemic diseases* in the patient then you might consider that as *an allergic reaction* form cosmetics ,lip stick ,certain food , oral hygiene products ,toothpaste especially one containing cinnamon or some restorative materials ,so we do the patch testing for the patient to exclude the possibility of allergy .
🟋 If the patient was *not allergic* to anything then we say that this Lip swelling is **Idiopathic orofacial granulomatosis** .
🟋We also have to take a biopsy and check that we don't have any *foreign bodies* because implanting of foreign bodies in the lip can cause chronic inflammation and enlargement or swelling of the lips ,, we also have to check that we don't have Infective granulomas in the biopsy like the one caused by *TB or leprosy* .

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🞛**Crohn's Disease**,Crohn's disease can affect any place from the mouth till the end of the colon and the infection might start in the oral cavity then appear in other places .
 Some symptoms of Crohn's disease is the abdominal pain, constipation, diarrhea, chronic obstruction in the colon or intestine, malabsorption so the patient might have anemia because he have ulcerations in the intestine so he might have iron deficiency anemia or B12 of folic acid deficiency anemia .
 ❖**Crohn's disease oral manifestations** might appear before anything happens to the colon or the intestine like the lip and cheek swelling "orofacial granulomatosis ", the gingiva might be red swollen and inflamed , mucosal tags, ulcerations like aphthous-like ulcers or linear ulcers in the sulci , inflammation of the tongue and glossitis so the tongue might be red or atrophic , and the feature that you always link to Crohn's disease is the" Cobblestoning "to the buccal mucosa and gingiva and this appears as small swelling with depressions in between, you might also see Candidal infection and Angular cheilitis because they have anemia .
 🟋If you saw all or some of these manifestations then you have to think of Crohn's disease, and how to make sure ?? ,, the answer is that you take biopsy from the buccal mucosa we will see non- caseating granuloma and this shall make you even think more that this is Crohn's and "t7awlo lal ba6eeni ".
❖**Histopathology** is non- caseating granuloma, fibrosis , lymphedema , chronic inflammatory cell infiltrate.

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🞛**Sarcoidosis** , it’s a multiple granulomas that affect the skin or usually lungs also it can affect lymph nodes ,salivary glands and mucous membranes including the oral cavity ,, its of unknown etiology and normally it affect the black people more .
🟋The special thing that you can link to Sarcoidosis is Bilateral hilar lymphadenopathy in the chest radiograph , so if the dr sees that in a radiograph he have to think in sarcoidosis and make other investigations including the angiogenesis converting enzyme level which are found to be elevated .
❖**Clinically** , there is difficulty in breathing ,lethargy , multiple macules on the skin , cervical lymphadenopathy , and there is a syndrome that can happen to a patient with sarcoidosis which is Heerfordt's Syndrome and here the patient develops xerostomia ,facial paralysis ,enlargement of the parotid gland and inflammation to the eyes (uveitis ) .
Oral manifestations , we will see red lesions ,firm nodules , enlargement of the lips or cheeks.
❖**Histopathology** , non- caseating granuloma .
🟋From the Blood tests in Sarcodosis we will see an increase in the ESR "erythrocyte sedimentation rate " and this can be seen in all chronic diseases ,there will be also hyperproteinemia , angiotensin converting enzyme levels will be high , calcium levels will be also high .

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🞾**Scleroderma**
"ta9allob el jeld " these patients suffer from extensive fibrosis in skin , GIT , lung, kidney ,heart , so if the heart or the kidney was affected this might lead to their failure , the **cause** might be an autoimmune disease ,, the **symptoms** might include dysphagia –difficulty in swallowing - if the fibrosis was in esophagus , difficulty in breathing ,pulmonary hypertension , renal failure , heart failure, what is important for us also the patient will have "Mask face " meaning the patient does not have facial expressions ,trismus –difficulty in mouth opening - , tongue movement is also restricted ,and in the x-ray there will be widening of the periodontal ligament spaces.

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🞾**Oral submucous fibrosis**, they consider it as a type of scleroderma that affects the oral cavity in areas where there is chewing habits like tobacco like India and Pakistan, this lesion is potentially malignant, there will be fibrosis in the buccal mucosa, the lips or the soft palate and sometimes the pharynx but does not affect other areas , the mucosa become firm ,pale and restricted mouth opening – trismus- ,so difficulty in eating and dental treatment is difficult , also there is restricted tongue movement so oral hygiene will be poor.
🟋If we take a biopsy we will notice that in about 13% or 10-15 % of patients will have dysplasia so this lesion can then turn into oral cancer, we will have few keratinization ,and the lamina propria is avasculer with fibrosis ,the mucosa and the epithelium are atrophic .