Sheet no.# 8
Refer to slide: Diseases of the peridontium
Corrected by: Dalin Jihad

Histologically:

When we take a crosssection, we can see that the radiolucency is filled with granulation tissues, this granulation tissue consists of immature fibrous connective tissue, so you'll find fibroblasts, collagen fibers, capillaries, endothelial cells and chronic inflammatory cells infiltrate (lymphocytes and plasma cells) and you can also find acute inflammatory cells. *(And because we have a cavity then bone resorption occurred. Granualation tissues are immature fibrous connective tissue, consisting of multiple dialated capillaries and chronic inflammatory cells, fluid/edema).*

One of the things that you can find in the periapical granuloma are the cholesterol clefts (spaces), which were originally cholesterol crystals (accumulations of cholesterol) and because of the processing in histopathological preparations they melt and spaces result in their place which are called cholesterol clefts.

Q) Where did the cholesterol come from?
From the breakdown of rbcs which are extravasated outside the blood vessels, they get damaged and cholesterol gets released and accumulates in the area.
In periapical granuloma you can also find Hemosiderin deposits *(pigments)*; granules of Hemosiderin.

Q.what is Hemosiderin and how did it reach this area?
You can also find foamy histiocytes which are macrophages, and foamy means that they have soaplike bubbles in their cytoplasm; they are distended, large macrophages with a foamy cytoplasm. They are phagocytes; they engulf materials, and as a result of engulfing cholesterol and lipid materials they will acquire a foamy cytoplasm.
You will also find in the periapical granuloma foreign body multinucleated giant cells; thought to be a group of histiocytes that got fused together and formed one large cell with multiple nuclei the aim of which is also to engulf foreign materials like cholesterol crystals that was resistant to the histiocytes, that's why they are most apparent around cholesterol. *(Their aim is to remove the foreign body).*

In the periodontal ligament you will find epithelial cell rests of Malassez *(their remanants is usually in the PDL space)*, in normal sections they are found like strands or small islands of inactive squamous cells in the normal periodontal ligament, if there was a periapical inflammation, they get stimulated again and start to grow and proliferate and they might form a mass of cells (later necrosis occurs in the center of this mass leading to the formation of cystic lesion in place of the periapical granuloma, and these are called radicular cysts).

So, if you left the periapical granuloma without interference what happens:

- Will increase in size, but will eventually stop growing *(after reaching a specific size)*

- Can transform into acute alveolar abscess.
- Can transform into chronic alveolar abscess *(if acute alveolar abscess was controlled by the body it will turn to chronic alveolar abscess)*.
- Radicular cyst can form (cystic lesion).
- In some cases bone formation might happen at its periphery *(instead of resorption)*, so the area around the radiolucency will appear radio dense, and this is called osteosclerosis.
- Periapical inflammation can lead to the resorption of the root cementum, or in some cases if it was chronic mild irritation, it can lead to hypercementosis (clubbing of the roots).

Acute periapical abscess:

Those are the patients that come to the clinic.
Think about the patients attending the clinics with severe, non tolerable pain, not sleeping the night and want an immediate action from you, those patients have acute periapical abscesses; which means there is pus formation around the apex.

> *(Mostly closed pulpitis [there's high pressure inside pulp due to fluid, the dentine and caries haven't been perforated yet, there's no direct communication with the pulp. While the open pulpitis is one where there's communication with the pulp])*

Microorganisms are virulent in these patients.

> *(upon touching it lightly or during mastication there's aggravating pain, there's pressure around the tooth, redness and swelling, inflammation reached falcum around the tooth. This inflammation had reached the blood vessels and so it turns to a systemic sign and symptoms {e.g fever and malase}. At this stage the patient may actually benefit from antibiotics. But there is no rationale in giving antibiotics in acute peridontitis or chronic but without systemic signs.*

Clinically:
- Patient with severe pain
- Uncontrolled inflammation resulting in systemic manifestations such as fever and malaise.
- Extrusion of the tooth; patient feels an elevation in the tooth and any pressure or percussion leads to severe pain.
- This pain is spontaneous and not related to temperature change.
- Doesn't respond to electrical or temperature stimulation because pulp is already necrotic.

Radiographically:
if it was an acute periapical inflammation and formed an acute abscess, changes might not be that detectable; just a slight widening in periodontal ligament and less clear lamina dura. Later on after spreading we will have faintness of trabecular pattern & ↑ radcy in the bone around it.

If it was acute alveolar abscess coming out from an already existing periapical granuloma

you'll definitely detect an illdefined periapical radiolucency in the radiograph *(because it's a chronic process).*

Acute alveolar abscess can transform into chronic abscess with time if you drilled into the tooth (opened the tooth), or the tooth itself opened spontaneously, *(through the pulp, if open pulpitis; it could spread from below producing an inflammation in the vestibule causing cellulitis [inflammation and swelling of the face]).*

The doctor shows a picture and adds that the abscess with time can open and the pressure decreases and transforms into chronic, or the patient can come to you to open an access for it to reduce the pressure and the lesion becomes chronic.
So as we said, as it might transform into chronic, it also might continue as acute if left, and pus may get through:

1. The root canal if the tooth was open, or
2. From the gingival sulcus through the periodontal ligament(around the tooth) or
3. It can distribute through the cancellous bone, reach facial spaces and cause swelling of the face.

It can reach facial spaces; buccal space, submandibular space and sublingual space and (can cause in some cases "Ludwig's angina; bilateral swelling of the facial spaces mentioned with severe consequences *read about it*).

Histopathology:
If we take a crosssection, we'll find pus, necrotic material, acute inflammatory cells, bacterial colonies, granulation tissue.
Microbiology: mixed bacterial infection.

3.Diseases of the periodontium

Periodontium: gingiva, cementum, alveolar bone, PDL

We're not going to get into this topic in details because there is a complete course regarding it.
If inflammation was in the upper part we call it gingivitis, if it was in the deeper part it's called periodontitis.

Healthy gingiva: pink, stippled, sharp appearance between teeth with interdental papillae, we have marginal and attached gingivae, no spontaneous bleeding or when brushing and no redness or swelling.

Healthy gingiva is occasionally found in individuals, the most common to find is **chronic gingivitis** which means redness, swelling and inflammation confined to the marginal gingiva.

We don't call it acute gingival, because it takes a lot of time to be inflamed and the cells inside are chronic inflammatory cells.
In a picture the dr. Showed > redness and swelling of the marginal (*free)* gingiva and interdental papillae, this is chronic gingivitis.

Chronic periodontitis:
It's an inflammation or destruction that is distributed beyond the marginal gingiva to the supporting structures; bone, periodontal ligament and the attachment of the gingiva to the tooth and this leads to bone loss, gingival recession *(attachment loss)* or pocketing, exposure to the roots and mobility of teeth.

Risk factors (of chronic gingivitis or periodontitis):

Gingivitis is a very very common disease, and so, these risk factors are for those susceptible to periodontitis.

- Plaque: like caries, which is caused by bacteria in plaque, there are specific bacteria in plaque that cause the periodontal disease. These bacteria are different than that causing the caries.The longer you leave this plaque, the higher the risk of developing a periodontal disease.

  In the natural formation of dental plaque the most common bacteria at first is gram positive cocci, but if plaque was left for a longer time (individual did not brush his teeth), the thickness of the plaque will increase and it will contain further more species (gram negative/ anaerobic/motile). The idea is not to leave the plaque for long periods, as doing so will increase the risk of developing a periodontal disease (chronic gingivitis or periodontitis).

As a conclusion, chronic gingivitis and periodontitis are <u>plaque induced</u>.

But there are other risk factors (local or systemic) that can enhance the progression of the disease:

Local factors:
- Alignment of your teeth; if you have good alignment, you have less risk of developing a periodontal disease, but those having crowding or problems in their teeth alignment and areas that are difficult to clean, probability of developing is higher
- Restorations; presence of restorations in general and those that are faulty or over hanged in specific may lead to collection of plaque in that area and develop a periodontal disease.
- Presence of appliances; partial dentures or orthodontic appliances can increase the progression of the disease.

Systemic factors:
Those are aggregating factors, and the source of inflammation is plaque; if you remove plaque there'll be no inflammation.

- **Pregnancy or puberty:**
  hormonal changes can increase the progression of the disease, pregnant ladies can sometimes have chronic gingivitis and not periodontitis, and this might be caused by the presence of the pregnancy hormone progesterone which leads to the increase in permeability of the blood vessels in the gingiva and increase in the local release of inflammatory mediators in that area which leads to the inflammation of the gingiva, we can also see an enlargement of the pregnant ladies' gingivae which we call "pregnancy epulis". تضخم اللثة

Diabetes:

- Diabetic patients have more risk of developing a periodontal disease, the increased risk is thought to be the result of the reduced vascularity, reduced phagocytic function and immunity and increased sugar level in saliva and crevicular fluid.

- **Malnutrition:**
- Protein or vitamin c deficiency for example.

- **Blood diseases**:
  which reduce the immunity like cyclic neutropenia; leads to more periodontitis.

- **Drugs can affect the periodontium**:
- Oral contraceptive pills can lead to gingivitis in ladies. Corticosteroids and NSAIDS can reduce the inflammation in the periodontium, cyclosporine, phenytoin, Ca++ channel blockers can cause gingival hyperplasia and enlargement of the gingiva.

- **AIDS** can cause periodontal disease in affected patients.

- **Smoking**; especially in relation to the progression of the periodontal disease and you need to know how.
- **Mouth breathing**

Q. Is it related to gingivitis? And what are other complications to mouth breathing? Bone changes, gingival effects, growth of the face ….

We said that systemic diseases can affect the periodontium, but do the periodontal diseases lead to systemic diseases?

Periodontal disease has shown in some studies to be an associated factor in coronary artery disease and in stroke. Periodontal disease in pregnancy has been associated in an increase in premature births and low birth weights. Control of diabetes in patients with periodontal disease is more difficult than those with no periodontal disease.

So some studies in the literature support the idea that periodontal diseases can lead to systemic diseases.

Chronic gingivitis:

Features: asymptomatic, red and swollen gingiva with plaque on the gingival margins. Brushing or minor trauma can lead to bleeding and patient feels or tastes the blood in saliva.

Etiology: ineffective teeth brushing.

For academic reasons this inflammation or gingivitis has 4 stages. If this inflammation was left for a long period, some patients may develop periodontitis. Not all patients with gingivitis develop periodontitis.

The 4 stages of periodontal disease (gingivitis and periodontitis) are:

1. Initial lesion 2. Early lesion and 3. Established lesion (3 stages of gingivitis)
4. Advanced lesion that is periodontitis

1. **Initial lesion:**

- Subclinical.
- Starts 24 days after exposure to plaque (if individual didn't brush for 4 days, initial lesion starts) and it's not apparent clinically.
- Localized around the base of the gingival sulcus; changes related to the area of the gingival sulcus, these changes are: vasodilatation of capillaries, fluids and neutrophils migration to that area.
- Because there is fluid extravasation, crevicular fluid amount will be more than normal but it isn't that visible clinically.
  Attached or junctional epithelium connecting the gingiva to the tooth starts to get disrupted.

2. **Early lesion:**

- If left more than 4 days till one week after exposure to plaque, neutophils will increase in number.
- Impairment in junctional epithelium increases.
- Acute inflammation with neutrophils strats to change into chronic, so lymphocytes start to appear instead of neutrophils.
- Damage to fibroblasts which synthesize collagen fibers.
- It could damage surrounding tissue
- Edema increases

3. **Established gingivitis:**

- Here symptoms start to appear, if left more than 23 weeks without tooth brushing.
- More damage to the junctional epithelium.
- Pocket or circular epithelium starts to get damaged or ulcerated, ulceration means beginning of bleeding, blood appears when brushing or when minor trauma occurs.
- Area becomes red in color.
- Inflammation involving plasma cells instead of lymohocytes.
- More destruction to the gingival connective tissue.

- Volume increases, so swelling occurs in gingival
- Sometimes repair occurs in the area because of chronic inflammation, repair leads to chronic hyperplastic gingivitis; so in some patients there's enlargement of the gingiva which is called "chronic hyperplastic gingivitis", clinically apparent as enlargement of the gingiva in area of marginal gingiva, it's not only edematous but also fibrous.

4. Advanced lesion/periodontitis:

Not all gingivitis cases progress into periodontitis.

Features:

- Destruction of the connective tissue attachment of the roots; damage to the gingiva attached to roots.
- Alveolar bone loss; bone resorption between teeth
- Junctional epithelium attachment will become lower, and the shallow sulcus becomes deep that you can insert a probe in that area, this process is called "pocket formation" *it could reach 56 mm*.

Earlier evidence:

chronic inflammation from the base of junctional epithelium, inflammation goes downwards leading to resorption of the bone and damage to the connective tissue attachments, this results in what is called "apical migration of the junctional epithelium" and pocket deepening.

Clinically how to distinguish periodontitis:

- Red, swollen gingiva; like those of gingivitis.
- Gingival bleeding.
- Pocketing; when using a probe and measuring the depth of the sulcus.
  (false pocketing: when the gingival is swollen and inflamed, it allows the probe to go deep making the dentist think there is a pocket)
- Recession of gingiva.
- Loosening of teeth; see if there is mobility; it's a sign of periodontitis because it means bone resorption.
- Unpleasant taste or halitosis; because there is pocketing which leads to bacterial accumulation, bad breath and an unpleasant taste.

Radiographically:

At the beginning, bone between teeth, instead of being triangular, it becomes blunt (blunting of the alveolar crests is an early change).

Later one there is horizontal bone loss and super bony pockets; all bone becomes uniformly resorbed, and this is called "horizontal bone loss". Bone is reaching cementoenamel junction, now it all got resorbed and this can be seen by radiographs. *This is usually associated with supra bon pocketing, since the pocket is still above bone*.

However, in some cases, in some teeth you don't have horizontal bone loss but instead "vertical bone loss". *This is associated with infra bony pocketing.*
Factors related to increase in the vertical bone loss:

- Malalignment.
- Restorations between teeth; class 2 overhang.
- Occlusal trauma.
- All these can lead to more vertical bone loss.

1) **Chronic gingivitis:** plaque induced, *patient has gingival redness but not only the margins it includes all the gingival. This is due to allergy due to e.g. Toothpaste.*

2) **Plasma cell gingivitis:**

Etiology:
Hypersensitivity to toothpaste, mint, cinnamon, MW. Allergy to certain materials is the cause.

Clinically:
Rapid onset, diffuse bright red enlargement of both free + attached gingiva.
Enlargement may extend to the palate or edentulous area.
Patient may also present with dry fissured lips, , Angular cheilitis, and swollen tongue.

Histologically:

- If we take a section through the gingiva we see:
- Diffused sheath of plasma cells.
- Hyperplasia, Intercellular edema (spongiosis), Neutrophilic infiltration in epithelium leading to the formation of micro abscesses.
- Lamina propria: numerous vascular spaces, dense chronic inflammatory cell infiltration composed of plasma cells.

Differential Diagnosis:
Histologically it can be mistaken with:
Acute leukemia infiltration of gingiva
Multiple Myeloma affecting gingival *malignant tumor*

Clinical forms of periodontitis:

1) **Chronic/Adult periodontitis**:

- Starts in early adult life, *it takes a long time of inflammation, generalized*
- Tooth loss after the age of 50
- Involve entire dentition
- Characterized radiographicaly by horizontal bone loss

2) **Aggressive Periodontitis**:

→Localized Aggressive Periodontitis:

- Starts around puberty.
- Rapid, sever vertical bone loss leading to the formation of infra boney pockets
- Mainly in upper incisors and first molars, *because they're the first to forms*
- Not directly related to plaque quantity, small amounts of plaque found compared to large amounts of bone loss.
- It is related to certain kinds of bacteria and the immune system defects to these bacteria [Genetic susceptibility and neutrophilic dysfunction].
- Bacteria: Gram –ve anaerobic rods Actinobacillus Actinomycetemcomitans.
- 35 times faster bone loss compared to chronic periodontitis. *It can spread to other teeth, depending on eruption.*

→Generalized Aggressive periodontitis:

- Some (1/3) of the localized Aggressive periodontitis may turn into
- Generalized aggressive periodontitis.
- Starts below the age of 30
- Sever rapid bone destruction occurs within weeks or months and is episodic sometimes high destruction and sometimes little destruction.
- Gingiva may be acutely inflamed or normal.
- Variable vertical bone loss between different patients or even different parts in the oral cavity of a single patient.

Etiology: defect in leukocyte function against certain bacteria

3) **Periodontitis as a manifestation of a systemic disease:**

- Mostly in children
- First affects primary dentition then may pass on to the permanent teeth
- Extensive, *rapid* destruction of alveolar bone
- *If the heavy destruction was to be found in a child, then we should look into the possibility of a systemic disease such as: Agranulomatosis, cyclic neutropenia, down syndrome, Langerhans cell histocytosis, Papillon Lefevre syndrome, Hypophosphatasia (early shedding of teeth), Juvenile diabetes mellitus .

Gingival enlargement:

1) Edematous: inflammation causing edematous enlargement like that occur in the usual chronic gingivitis. Also edematous enlargement from hormones → puberty, pregnancy, oral contraceptives. Vitamin C deficiency in patients → scurvy → edematous enlargement with haemorrhegica bleeding on minor trauma, also may lead to mobility of teeth.

2) Fibrous:

A) **Chronic Hyperplastic gingivitis**:
Chronic gingivitis causing the formation of granulation tissue→increase in fibrous tissue → enlargement of the gingiva.
Patient would have poor oral hygiene, calculus , inflammation of the gingiva and fibrous enlargement of the gingiva.

B) **Drug induced Gingival enlargement (D.I.G.E):**

- Enlargement is the best term since it is an increase in fibrous tissue (No hyperplasia, No Hypertrophy)
- Epilipsy, organ transplant patients, HTN
- Drugs: Phenytoins, Cyclosporins, Calcium channel blockers
- 50% of people taking phenytoin end up with gingival enlargement
- 25% Cyclosporins users have enlarged gingiva
- 25% CCB users also have enlarged gingiva

Starting in the interdental papilla as bulbous enlargement then reaches the marginal gingiva. *Pale upon palpation.* It is pale in color (not inflammatory).
Enlargement may even cover the crown.
Mostly generalized, but aggressively in the anterior region.

Etiology: Drug modifying factor, the plaque is the first factor but these medications increase the enlargement. Patients on these drugs should maintain good oral hygiene.

Q) Is the gingival affected in edentulous spaces? Implants?

Histological Section: a section from the gingival enlargement shows fibrous tissues (lamina propria increases). Chronic inflammatory cell infiltrate because the gingiva almost always has some inflammatory cells.
Surface epithelium also is hyperplastic, plus elongation and anastomosis of rete processes.

C) **Hereditary gingival fibromatosis**

- A.D
- Genetic disturbances in fibroblast function therefore producing high amounts of fibrous tissue in the gingiva.
- Localized: affecting mainly posterior upper region, may reach midline of palate if large.
- Generalized: affecting whole arch
- Can be seen very early; even before eruption of teeth, so may cause delay in eruption of primary or permanent teeth.

- May also cover the crowns
- Pale, firm, smooth.
- Other feature of the patient to help in diagnosis: mental retardation, epilepsy, hypertrichosis
- Histologically: Thick bundles of collagen with little or no inflammation and hyper plastic surface epithelium.

Other causes of gingival enlargement:
Puberty, Pregnancy, oral contraceptive pills are hormonal factors that cause edematour gingival enlargement.

3) Systemic causes:
Acute myelogenous leukemia → infiltration of leukemic cells in gingiva.
Wegner's Garnulomatosis→ strawberry gingivitis
Sarcoidosis

Periodontal Abscess:

Related to periodontal pockets around teeth

- Etiology:
- Acute infection of a pocket
- Patient with deep pockets especially with vertical bone loss, the pocket may close and then turns into an abscess, not seen in radiograph because it is beside the tooth
- Patient with periodontitis; or even trauma to the periodontal ligament from perforation through root canal treatment, may end up with periodontal abscess.

Features:

- Acute throbbing pain, gingival tenderness, teeth are vital compared to periapical abscess, but tender to lateral percussion while periapical abscess is tender to vertical percussion.
- Gingiva swollen red may have pus from sulcus
- Patient has generalized chronic periodontitis
- Radiograph: If acute → not visible
  If chronic →radiolucency lateral to the root

Pericoronitis:
Inflammation of gingiva around partially erupted teeth especially lower 3rd molar. Mesially erupted while distally still covered by operculum.

Plaque accumulates in the space (between the covered part and the crown below)→ chronic inflammation →edema → enlargement → trauma from opposing teeth during closure → inflammation.

Clinically: pain, tenderness, bad taste, difficulty in swallowing (Dysphagia), trismus, inflammation, and abscess may spread and reach into facial spaces.
