**Sheet #11**

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**Lecture topics:**

* Fibroepithelial polyp
* Denture induced (irritation) hyperplasia
* Papillary hyperplasia of the palate
* Epulides
* Pyogenic granuloma

❖ “Lymphoepithelial cyst”:

Pathogenesis: entrapped epithelium

Age: usually 2nd & 3rd decades

Clinically: usually shows as a painless swelling with or without a fistula, usually anterior to the sternocleidomastoid but can appear intraorally.

❖ “Dermoid & epidermoid cysts”:

Pathogenesis: development of branchial arches, usually crosses the midline, on the hyoid & jaw or just beneath the tongue above the mylohyoid.

Histology:

They both have a lining composed of stratified squamous epithelium however, the wall is different in the dermoid where it includes full skin histological features including the skin appendages, whereas the epidermoid cyst’s wall does not contain skin appendages histologically.

❖ “Thyroglossal cyst”:

It is basically composed of remnants of the thyroglossal tract & also usually appears in the 2nd or 3rd decades. Clinically, 70 to 80% of the cases appear below the hyoid, between the foramen cecum & the thyroid. It usually appears just at the midline of the neck. It sometimes opens a sinus tract & starts secreting secretions. It is usually asymptomatic & mobile. Histologically the lining is variable whereas the wall, as expected, is composed of thyroid tissue.

Connective Tissue Lesions

The connective tissue lesions are either hyperplastic lesions, or tumors (sarcoma).

Connective tissue hyperplasia is the most common. It is caused by a chronic infection or a chronic mild irritation (such as biting) of which the body reacts to “excessively” forming an abnormal granulation tissue. This abnormal granulation tissue is the lesion.

❖ “**Fibroepithelial polyp**”. The histopathology of it:

* The lesion comprises a core of dense, relatively avascular and acellular fibrous connective tissue that usually appears parallel to biting (buccal mucosa, lateral border of the tongue). It is a pale, firm, pedunculated/sessile, & painless lesion. There’s no ulcerations associated with it as it is a resisting trauma.
* The surface covered by stratified squamous epithelium. Sometimes, you can see the surface epithelium keratinized as a result of chronic trauma.
* Sometimes, you can see within the fibrous connective tissue esp. close to the surface epithelium large multinucleated fibroblasts. This variant is falsely called “Giant cell fibroma”. One of its wrong names is “Leaf fibroma” as well. However, using the term “fibroma” to describe this variant is incorrect as it is not a tumor. The difference between this type of polyps and a fibroma, is that as the polyp becomes larger, the cellularity of it decreases becoming an established lesion. However, as the tumor grows, as we all know, its cellularity increases. They both however, are similar in the clinical presentation & have similar prognosis (recurrence (for the polyp) vs. metastasis (for the tumor)).
* ❖ **Denture induced (irritation) hyperplasia**
* The same mechanism of fibroepithelial polyp; chronic irritation and formation of granulation tissue.
* Caused by the flanges or the periphery of ill-fitting denture. Over-extension of the flanges causes trauma to the sulcus, formation of chronic inflammation and granulation tissue. Thus, the same as fibroepithelial polyp but modified in shape; 2 linear folds found on the sulcus on both sides of the denture flange inner and outer (2 folds separated by the flange of the denture). We sometimes find ulcers between these folds. Also, it can be seen on the palate along the posterior edge of the upper denture.
* Can be single or multiple.
* *Clinically:*

🞊 More than one firm, broad base and leaf like.

🞊 Embracing the flange.

🞊 ± linear ulcer (linear ulcer can be found on the center).

🞊 More commonly found in the mandible than the maxilla, because in the mandible there is no good support to denture but in the maxilla there is good support.

🞊 More common in females than in males, because females wear the denture more because of their esthetic concerns

* *Histologically:* fibrous connective tissue (granulation tissue), chronic inflammation and covered by stratified squamous epithelium. Again, the same mechanism of fibroepithelial polyp and the only modification is the shape and that ulcer can be found in the area against the denture.
* **Papillary hyperplasia of the palate**

⚫ Found on the palate of edentulous patients in the area covered by the denture

⚫ Numerous, small and tightly packed papillary projections on the hard palate

⚫ 3-4 mm in size

⚫ Covering part or all of the denture bearing area

⚫ 2 variants can be seen; one with redness (thought to be caused by secondary bacterial or candidal infection under the denture), its appearance described as “field of confluent reddish mushrooms” and the other without redness.

⚫ *Etiology*: poor oral hygiene, poor denture hygiene, loose ill-fitting denture (causing trauma) and sleeping with the denture (mostly seen in females) + low grade infection by bacteria or candida (if the infection is found there will be redness).

⮱ **C**an we find **papillary hyperplasia** of the palate in **dentate patients**?

⚫ *Histologically*: granulation tissue (fibrous connective tissue) covered by stratified squamous epithelium. Sometimes, this surface epithelium is hyperplastic with elongated rete ridges (epithelial processes). This appearance may be referred to as “**pseudo-epitheliomatous hyperplasia**”. In some cases this may be so prominent as to mimic invasive squamous cell carcinoma; the appearance of long epithelial processes that appear as islands in cross section inside the connective. It must be distinguished from SCC to prevent an improper management (ex. Removal of the palate).

* **Epulides**

🙞 Singular: epulis

🙞 Epulis: reactive focal connective tissue proliferation confined to the gingiva.

🙞 Common; inflammation is common in gingiva (gingivitis).

🙞 Caused by **trauma** or **chronic irritation**. Chronic irritation: calculus, subgingival plaque, overhanged restorations, or appliance.

🙞 80% of cases anterior to molar region

🙞 Begins at the interdental papillae

🙞 Females more than males; because of hormonal modifications of gingival inflammations

🙞 The recurrence is common if we don’t remove the cause (ex. the surgical removal of the epulis without removing calculus or correcting the faulty restoration = recurrence of it). The recurrence rates however, are highly variable.

🙞 There are types of epulides according to the predominant component of the granulation tissue of the epulis; if the blood vessels and endothelial cells predominate, it is called “**Vascular epulis**”. If the fibrous tissue predominates, it is called “**Fibrous epulis**”. If the histocytes and giant cells (formed by fusion of histocytes) predominate, it is called “**Giant cell epulis**”.

1. ***Vascular epulis***
* Accounts for 28% of epulides, the recurrence rate is 14% (if 100 vascular epulides are removed, around 14 of them return).
* *Clinically:* Polypoid, soft (if traumatized, ulceration and bleeding will happen), bright red and rapid growth.
* Can occur in pregnant ladies “**Pregnancy epulis**” at the end of the 1st trimester, grows through pregnancy and following delivery it shrinks and becomes fibrous epulis. If it was small, it may disappear following the delivery. It is also called “Pregnancy tumor” as the term “tumor” means swelling, but it is better not to use this term to differentiate it from malignancy.
* *Management of pregnancy epulis*: If the patient has large epulis that causes bleeding, esthetic and eating problems, it should be removed during pregnancy. Otherwise, the management should be delayed after delivery and this is better, why? Because if we remove it during pregnancy, the bleeding and the recurrence rate will be more. So, as a rule it is better to delay the treatment after pregnancy. After pregnancy it will shrink and therefore the surgery will be easier and with less bleeding and recurrence.
* *Histopathology*: proliferation of endothelial cells (vascular proliferation) which may take the form of solid sheets of endothelial cells (immature) that form capillaries later on (mature). The result is the formation of numerous capillaries and large, dilated, thin walled vascular spaces containing RBCs. The vascular element is supported by a delicate fibrous connective tissue.
* The surface is soft and predisposed to trauma that may cause ulceration. So, it is common to see ulceration on surface.

⮱ The term “Vascular epulis” is limited to the gingiva, what about the same lesion occurring on other parts of the oral cavity (lateral border of the tongue, buccal mucosa…)? The general term is “Pyogenic granuloma”.

* **Pyogenic granuloma**

Any localized vascular proliferation on any site of the oral cavity or the skin. It is a wrong term to be applied here, the term “Pyogenic” is used to describe a reaction against bacterial infection that produces pus. Because historically they thought that pyogenic infection is the cause rather than chronic irritation. Also, the term “granuloma” is also misleading instead it is a granulation tissue.

⮱ So, pyogenic granuloma is a general term. Even if the lesion occurs on the gingiva, it can be called pyogenic granuloma, but vascular epulis is more specific for the gingival lesion.

⮱**W**hat is the **percentage of Pyogenic Granuloma** that occurs on the gingiva? And that occurs on other sites??

1. ***Fibrous epulis***
* Accounts for 65% of epulides (most common epulides) because part of the vascular epulides is converted into fibrous.
* The recurrence rate is 2%
* Pedunculated or Sessile.
* Firm (not soft as the vascular one), pink of similar color to normal gingiva or pale if there is a lot of scar tissue, it is rarely ulcerated because it is firm and can tolerate any trauma.
* *Histologically:* collagen bundles with diffuse sheets of fibroblasts, chronic inflammatory cell infiltrate (predominantly plasma cells), you can see also bone formation (bone trabeculae) in some cases and because of this in some text books, it is called **peripheral ossifying fibroma**. It is incorrect term because it is not a benign tumor.

⮱ **W**hat is the **origin of bone** that is deposited in the gingiva **in case of fibrous epulis**? (the cells of origin)?

1. ***Peripheral giant cell granuloma (giant cell epulis)***
* The least common epulis –only 7% of epulides.
* High recurrence rate (37%), so that is important to consider this in its management.
* Age groups are variable with gender: females -5th decade of life, males -2nd decade of life.
* Sessile or Pedunculated.
* Soft (like vascular epulis), reddish/**bluish** and ulcerated. Should think about when you see a soft bluish localized swelling of gingiva.
* Can reach a large size and extend interdentally with “Hour-glass” shape with buccal and lingual swellings connected by thin band between teeth.
* Complications: loosening & movement of teeth.
* Can be found on edentulous edge with dome shape (the difference between this epulis and the vascular and fibrous epulides).
* Radiographs may reveal in some cases where the lesion is large and causing pressure on underlying bone **superficial erosion** of the crest of interdental bone or in edentulous areas of the alveolar bone margin. The density of bone will be less.
* They are found on the gingiva, but some theories tell that they originate from the periosteum. The lesion may be deep to reach between teeth and periodontal ligaments. This may explain its high recurrence rate.
* *Histologically:*
* Polypoid lesion covered by stratified squamous epithelium. Under the epithelium, there is a zone free of giant cells called “giant cell free zone”. Then central to this zone, lobules of MNGCs proliferation are located.
* Nodular collection of multinucleated giant cells lying in richly cellular and vascular stroma composed of mononucleated cells or spindle-shape cells. (Numerous multinucleated giant cells close to capillaries between them mononucleated cells).
* The giant cells are variable in size, shape, number of nuclei and depth of staining of the cytoplasm.
* The mononucleated cells are thought to be a mixture of fibroblasts, endothelial cells, osteoblasts, histocytes and undifferentiated cells (have a potential to be any of the cells).
* Vascular channels, extravasated RBCs and hemosiderin deposits are found throughout.
* If there is ostoblasts, few trabeculae of bone or osteoid may be found within the lesion.

⮱**W**hat is the pathogenesis of giant cell epulis? **W**hat is the origin of the lesion? **W**hat is the origin of MNGCs??

Through the pathogenesis, we must know the cause of each one of the characteristics we have mentioned. Why it is a deep lesion? Causes pressure on the bone? Has high recurrence rate?