SHEET: LAB PATHOLOGY #1
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Normal lining of the oral cavity:
- Squamous epithelial lining (formed by maturation of basal cells)
- Basement membrane
- Followed by a layer of connective tissue (lamina propria)
- Submucosa
- Muscular layer

Usually there is no basal cells and few inflammatory cells
TYPES OF LESIONS in the oral cavity:

1- INFLAMMATORY:

Aphthous ulcer

Defect in the epithelium (no epithelium)

a- Necrotic tissue + inflammatory cells = active ulcer
b- Healing and angiogenesis (formation of granulation tissue)
c- Fibrosis = chronic ulcer
2- Reactive lesions:
   a- Fibrous proliferative lesion:
      - irritation fibroma = traumatic fibroma

Benign, well circumscribed, on the buccal mucosa (bite line)
Treatment: self-regression/surgical excision
Cell of origin: fibroblasts (produce collagen)
b-Leukoplakia:

Any whitish lesion that is not clinically or microscopically explained (no diagnosis)

Histology: severe dysplasia + loss of (polarity and maturation of basal cells) + mitotic figures + normal epithelial lining
Grades of dysplasia (one – involve the lower layer, two – lower and middle layers, three/carcinoma in situ-full thickness with intact basement membrane)

c- Erythroplakia:

Reddish lesion with no diagnosis

90% malignant (squamous cell carcinoma)

(SCC May occur in the oral cavity, skin, esophagus, stomach, small and large bowel, rectum, and anus)...same histologic appearance

2 criteria:

Keratin + dysplasia + break in basement membrane
At low power, there is normal squamous mucosa at the lower right, but a squamous cell carcinoma is infiltrating the submucosa and muscularis of the tongue. Risk factors for oral cancers include: use of tobacco, alcoholism, prolonged irritation from ill-fitting dentures or irregular teeth, and chewing betel nut.

At medium power, there is normal squamous mucosa at the right, with a squamous cell carcinoma is infiltrating the submucosa of the tongue at the left. Oral cancers can develop and spread quickly, but many can be detected early and excised.
Mucocele:
Most common in lower lips
Most common inflammatory lesion
Cause: obstruction of salivary gland duct
Size: 0.5-0.7 cm (change with meals)
Histology: pseudocyst (no epithelial lining, clear cavity) filled with saliva, mucin and inflammatory cells mainly macrophages

Duct
Acini
Granulation tissue
Types of salivary glands:
1- serous: parotid gland - pink
2- mucinous: sublingual gland - white
3- mixed/seromucinous: submandibular gland– pink and white

Causes of sialadenitis / inflammation in the salivary glands:
1- Nonspecific: caused by blockage of salivary gland duct

E.g.: sialolithiasis (blockage by stones)/ (most common in submandibular gland), superimposed by staphylococcus aureus or streptococcus viridians followed by duct dilation then recruitment of inflammatory cells

Histologically assembles calcification because it contains calcium...stains purple to red

Measured grossly no need for microscope
2-Specific: a-infection: e.g.: mumps in parotid gland /bilateral enlargement, most common in children

b- Autoimmune disease: e.g.: sjogren syndrome

- dry moth
- Auto immune antibodies that attack salivary gland (dry mouth), and lacrimal glands (dry eyes /keratoconjunctivitis sicca)
- Diagnosed by 1-lip biopsy 2- serology (detection of abs: SSA, SSE)
Salivary gland tumors:

1-pleomorphic adenoma:

- Mass in the oral cavity/upper neck/cheek, common in females (except for Warthin tumor (smoking)), the incidence of malignancy in salivary glands is inversely proportional with its size

- Unilateral mass in the cheeks
- Risk factor: radiological therapy to head and neck regions
- Grossly: benign, well circumscribed
- Cut surface: glistening and shiny (condroid material/cartilage)
- Treatment: complete excision of tumor + gland to avoid recurrence
Figure 16-15 Pleomorphic adenoma. A, Slowly enlarging neoplasm in the parotid gland of many years duration. B, The bisected, sharply circumscribed, yellow-white tumor can be seen surrounded by normal salivary gland tissue.

Figure 16-16 Pleomorphic adenoma. A, Low-power view showing a well-demarcated tumor with adjacent normal salivary gland parenchyma. B, High-power view showing epithelial cells and myoepithelial cells within a condroid matrix material.
Histology: 1) epithelial elements:
- (duct like structure, clusters)
- myoepithelial (spindle + contractile activity, and can be seen with smooth muscle immunomarkers)

2) Mesenchymal elements: condroid (cartilage like) material

From the book:
Rounded, well demarcated masses, encapsulated, but in some locations (palate): not well developed capsule+ expansile growth and protrusions into the surrounding tissues, in most cases: no dysplasia or mitotic activity
2-warthin tumor:

Smoking, males, 10% bilateral/multifocal,

Presence of lymphoid tissue (lymphocytes surrounded by epithelial cells (2 layers: columnar/cuboidal)) and many forms germinal centers.
3-mucoepidermoid carcinoma:

- 3 types of cells: mucinous cells, epidermoid/squamous cells, intermediate cells (have both: squamous features and mucous filled vacuoles)
Normal lining of the esophagus: squamous epithelium
And grossly: whitish mucosa and red glandular epithelium
Esophageal ulcers are caused by infections:

1) HSV esophagitis:

HSV causes formation of:
1) intranuclear inclusions which gives the glassy appearance
2) And giant cells (by cells aggregation)

=punched out ulcers + squamous epithelial cells become multinucleated with a glassy nucleus
2) Candida esophagitis

Fungal infection (yeasts that reproduce by budding), causes thrush/whitish lesions, in immunocompromised people, diagnosed by biopsy.

Sometimes there is formation of pseudo hyphae (septate hyphae) as a result of incomplete budding where cells elongate but remain attached.

Stain: PAS/AB stain (for fungal infections)

Fungi:
- yeasts (reproduce by budding)
- molds (reproduce by hyphae),
- dimorphic (yeast or mold depending on temperature)
Large numbers of eosinophils (red/bilobed nucleus) at upper + mid esophagus and is caused by:
1) food allergy (especially: cow milk with a history of eczema and allergic diseases in other parts of the body), 2) parasite, 3) procedures

Small number of intraepithelial eosinophils
Basal cell thickening
Lengthening of stromal papillae
+lower part of the esophagus

Figure 17-5 Esophagitis. A, Reflux esophagitis with scattered intraepithelial eosinophils and mild basal zone expansion. B, Eosinophilic esophagitis is characterized by numerous intraepithelial eosinophils. Abnormal squamous maturation is also apparent.
Barrett esophagus

It is the result of gastric content reflex (complication of 0.1 GERD) and chronic irritation that will cause metaplasia (appear as red lesions), and transformation of squamous cells into columnar epithelium (hallmark of barrette) with goblet cells and glands. It is followed by dysplasia and risk for adenocarcinoma if not treated.
Another cause for inflammation is a so-called "Barrett esophagus" in which there is gastric-type mucosa above the gastroesophageal junction. The metaplasia results from chronic gastroesophageal reflux disease (GERD). Note the columnar epithelium to the left and the squamous epithelium at the right. This is "typical" Barrett mucosa, because there is intestinal metaplasia as well (note the goblet cells in the columnar mucosa).

This is Barrett esophagus associated with gastroesophageal reflux disease (GERD) with some dysplasia of the columnar epithelium. There is a long-term risk for adenocarcinoma. The short term problem is inflammation and/or ulceration.
(Precancerous) Dysplasia in glandular epithelium will cause depletion of goblet cells and formation of cigar shaped, hyperchromatic, and elongated nuclei (arrow) + stratification.

**BOOK:** Goblet cells contain mucous vacuoles, stain pale blue by H&E

Dysplasia is classified as low or high grade on the basis of morphologic criteria.
Severe dysplasia /carcinoma in situ/no invasion
Squamous dysplasia with invasion

Resembles SCC in the oral cavity

BOOK: nests of malignant cells that recapitulate the stratified organization of squamous epithelium

Keratin pearl
VARICES: dilated blood vessels below the submucosal tissue if they become enlarged they can cause bleeding

Main cause: liver cirrhosis and increased pressure in portal vein and formation of collateral circulation between portal and caval systems

Figure 1 Large esophageal varices at EGD.
Chronic gastritis:

2 types of mucosa in the stomach: antral (foveolar cells), oxyntic (parietal and chief cells)

Normal lining: columnar epithelium and glandular epithelium (glands), NO goblet cells, NO lymphocytes.

If lymphocyte is present = chronic gastritis this can lead to thickened rugal folds

If neutrophils are present = acute gastritis

If both types of inflammatory cells are present = acute on top of chronic/acute active chronic gastritis

Chronic pan gastritis:

Antral and oxyntic mucosa are involved most common cause is H pylori
H pylori: (rod-like, stained by Warthin-starry silver stains and immunohistochemical stain) it affects antral type mucosa

Presence of germinal centers: lymphoid follicles indicates the presence of H pylori (BOOK: this is an induced form of MALT and could transform into lymphoma)
ACUTE GASTRITIS:

Only neutrophils
And with long term of gastritis ...
mucosal atrophy... loss of glands...
fibrosis

Atrophic gastritis: most common cause is: autoimmune gastritis that damage the oxyntic mucosa (body/fundus), and they become thinned+ rugal folds are lost (BOOK: if parietal and chief cells are loss extensively this could develop an intestinal metaplasia
Atrophic gastritis, intestinal metaplasia

As a result of chronic irritation: there will be intestinal metaplasia: (BOOK) (presence of goblet cells admixed with gastric foveolar epithelium), and is a risk factor for gastric adenocarcinoma.

PUD: complication of chronic gastritis, defect in the mucosa as a result of infection (H pylori: HCL, HCO3-(loss of protective layer) increasing the acidity and formatting an ulcer that can be replaced by fibrosis with time.

PUD occurs in specific area (antrum/first part of the duodenum), punched out ulcer with regular borders and rarely develop into cancer.

Cancer and tumors in gastric mucosa appear as heaped up ulcers with irregular borders and are malignant from the start.
The inside of the stomach where a peptic ulcer has formed.

Diagram shows Pept ulcer and duodeum ulcer.
Heaped up malignant ulcer with irregular borders
4 layers:
Ulcer on the surface (necrosis+fibrin+inflammatory cells) then granulation tissue formation (the ulcer base) then fibrosis.
Gastric polyps

Elevated part of the mucosa (small mass = 0.3-0.5 cm) + dilated glands

TYPES: 1) Hyperplastic/inflammatory polyp

Caused by H pylori, mass in antral type mucosa

BOOK: [grossly: multiple, ovoid, smooth surface/microscopically: irregular, cystically dilated, and elongated foveolar glands + edematous lamina propria with acute/chronic inflammatory cells and frequency of dysplasia is proportional to the size of the polyp.]
2) Fundic gland polyp

In fundus/body type mucosa (in parietal type cells)

Mass of glands lined by parietal cells

Can be caused by long term use of PPIs (TTT of GERD)

Gastric adenocarcinoma, intestinal type

Intestinal metaplasia is the precursor lesion

BOOK: Bulky, composed of glandular structures (neoplastic intestinal glands resemble those of colonic and esophageal adenocarcinoma)

And neoplastic cells contain apical mucin vacuoles and abundant mucin in the gland

Lumina

Cohesive cells (sticking together)
Gastric adenocarcinoma, diffuse type (signet ring cells)

No precursor lesion
Malignant cells filled with mucin and a peripheral nucleus
BOOK: grossly: linitis plastica (thickened gastric wall + loss of rugal folds //Discohesive cells

Lyomyoepithelial lesion in MALT lymphoma (the most common lymphoma)

Complication of chronic gastritis if not treated (caused by H pylori)
Lymphocyte accumulation and invasion of gastric glands and this represent a risk factor for the: development of lymphoma or translocate to form extranodal MALT lymphoma
(MALT are normally found in ilium as Peyer’s patches (innate immunity))
Gastric MALT lymphoma

Lymphocytes were first in the interstitium but now they have fully infiltrated/invaded the gastric glands and the glands can’t be seen anymore.
Tumor in the serosa/wall of the stomach (may occur in small bowel) caused by mutation in SYK gene (tyrosine kinase gene) and it arise from proliferation of interstitial cells of cajal (stromal tumor) (histogenesis is not yet determined: smooth muscle or neural)

GIST prognosis depend on: 1) site: stomach is better than in small bowel2) size: smaller the better3) presence of mitosis worsens the prognosis

Histologically: it is a proliferation of spindle cells and epithelioid like cells
Carcinoid tumor

Low grade tumor arise in GI (especially in small bowel), liver, pancreas in neuroendocrine cells (secretes endocrine material e.g.: serotonin)

GOOGLE: Neuroendocrine tumors (NETs) are neoplasms that arise from cells of the endocrine (hormonal) and nervous systems. Many are benign, while some are malignant. They most commonly occur in the intestine, where they are often called carcinoid tumors, but they are also found in the pancreas, lung and the rest of the body.
Organized (appear as: nests, stones, strands, islands...)

It is in the small intestine because there are villi (contain goblet cells and glands
(Brunner's glands in duodenum only)

[Duodenum, jejunum, and ilium differ in villi

Salt and pepper appearance (due to:
1) stippled nucleus because of the chromatin's texture (fine and coarse clumps)
2) The cytoplasm is granulated)
- Tumor cells are embedded in dense