

Pathology - GIT

29.9.2021
2nd Lecture

Cancer shows like mass (hard, soft, ...)
↳ Revolution with time

- ① surgery (tissue + normal tissue)
- ② chemotherapy, Radiotherapy (have ~~forex.~~ negative effect on bone marrow that contains RBC, WBC, Platelets)
↳ bleeding

→ molecular pathology → more specified and precise
→ new technology

↳ in SCC and oral cavity used

• TP53: the guardian of genome "مراقب"
always random mutation (normal) → repair mechanism → protection

↳ if → not repaired → apoptosis

→ importance: mistake isn't cared to second generation

- not specific for SCC, but all -

Cell cycle: moves between normal condition and not

↳ checkpoints at different points → proteins (cyclines, cycline dependent kinases and inhibitors)

→ CDKN2A → P16 → Not (its importance: inhibition of cell cycle)
PIK3CA → signaling pathway.

FAT 1 → cadherine family

→ work due calcium

TP53 → related HPV → negative tumors harbored more somatic mutations

RB → like "دفعه جريانه" in car

Retroviral → problem → cell defect

Inactivation of TP53 and RB pathways by HPV E6 & E7 in HPV positive tumor

- ① Growth factors → have receptors, that get affected (signal)
 - ② secondary messengers
- ③ Transduction proteins in the inside (other side of receptor)
 - every one has a specific job
- ④ Transcription factors inside
 - problem happens usually here.
 - no repair mechanism

Framework

HPV negative; classic

keratinizing

tonsils, ...

- does not
 - Older people
- Tobacco and alcohol exposure
 - anywhere
- Large primary lesion

HPV associated

- non keratinizing
- lateral border, hard palate (other than classic)
- goes to lymphoid rapidly
- Younger people
- Number of oral sex partners
- Oral pharynx and around
- Small primary lesion

Xerostomia & Sialostomia → dryness of eyes

dry mouth → no saliva

different etiology → Sjögren's syndrome

Auto immune disease

Antidepressant, anti psychotic → dry mouth

morphologically → dry mucosa

- chronic & long term atrophy of mucosa

- caries in teeth

- fissuring (fissuring)

- ulceration & secondary infection

↳ Superimposed infection

Sialadenitis

→ infection of glands

* duct blocking saliva → rupture → retention in gland
pus

Infections: Mumps

Morphologically: parotid, testis, pancreas

Bacterial Sialadenitis (S. aureus + S. viridans)

retrograde from saliva glands → severe dehydration

high amount of neutrophils → pus & abscess formation

Obstruction: sialolithiasis (stone)

→ proliferation of bacteria and infectious agents

Clinically: - swelling (painful) as mumps
→ sublingual (pus discharge)

microscopically: infiltrated salivary glands (tissue destruction)

granuloma → macrophages or epithelioid histocytes

(fungal) - mycological infections sometimes

Salivary gland tumors

- < 2% of all human tumors up to 8% within parotid
- most of them benign → but in other ^{salivary} glands not
- 5th and 7th decade of life
- male - female (percentage)

- In parotid ^{major tumors} not more than 20% are malignant
- mostly elderly

Pleomorphic adenoma

- in parotid gland most of time (60%)
 - Benign mixed tumor (first name)
 - Single origin of all components
 - 90% of benign salivary gland tumors
 - 2 types of cells →
 - epithelial tissue
 - mesenchymal tissue (bone, C.T.)

} closely intermingled
 - Pleu. Adenoma form 90% of Benign salivary cell carcinoma
 - Molecular
 - overexpression of **PLAG 1** (transcription factor)
 - Or **HMG A2** gene mutation in the cases lack the former
 - Benign tumor → capsulated
 - malignant → invaded in tissue
- } In pleomorphic adenoma
Finger like projection
↓
pleokinesis → come back

cancer

In testis $\downarrow \rightarrow$ tunica albuginea (cutting)

$\rightarrow$ increases diseases $\rightarrow$ cancer (testis) • has
to be removed completely

Malignant transformation

$\rightarrow$ genetic mutations form it from benign $\rightarrow$ malignant

- one mutation makes it easier for other mutations
 $\rightarrow$ faster $\rightarrow$ progressive

$\Rightarrow$ this happens 2-10% in <5 or >15 years
in pleomorphic cancers and large cell carcinoma respectively.

Under microscope =

fingerslike projection

(many little masses connected with initial one)

$\rightarrow$ recurrence

Warthin tumor

→ researchers showed that it's actually not a tumor more like a reactive process

other names (discriptive)

⇒ Papillary cystadenoma lymphomatosum

- commonly between smokers (8 fold)
- can be bilateral and multifocal (each 10%)
- common in between 5th - 7th century

Microscopically: projection like structure lined by double layer of epithelial cells (called oncocyte) rich in glycogen and myoepithelial cells

Mucocystic carcinoma

- most common primary malignant tumor
- most in parotid and rest in minor salivary glands
- Translocation between (11q21 → 19p13) in one half of cases
↳ part of DNA switches with another → genes have a lesser connection together
- ↳ **CTC1** } transcription, fusion (chimeric) protein → carcinoma
- ↳ **MALM2** } has role through NOTCH and cAMP dependent signaling pathways

(⇒ dependent on Stage again, as all cancers)

* **Desmoplasia**: reaction in C.T due to tumor cell invasion *

⇒ heterogeneous morphology

- intermediate cells = mucin
- squamous ^{like} cells

* Differences between different stages *

- Stroma → C.T rich in collagen called **Plastic Stroma**

↳ induces some reactions

↓
let Fibroblasts active, produces follicles
let inflammatory cells arrive

↓
Desmoplasia

sometimes stone hard in some tumors as in breast.

Adenoid cystic carcinoma

- aggressive and bad prognosis
- mostly seen in salivary gland (trachea, breast)
- likes perineural invasion (in perineural space)
↓ ↓
rarely rarely
between nerve and tissue

Acinar cell carcinoma

- compared to other carcinomas, relatively good prognosis
- looks like acinus in serous glands
- Bilateral and multifocal

Esophagus → non keratinizing squamous epithelium

Esophageal obstruction

① Functional obstruction

→ problem in delivering function → no food movement

nutcracker esophagus

Barium meal = x-ray picture showing lumen

↳ process done to see different types of esophagus.

Shapes for example.

→ high amplitude contractions in different places

(and between them no contraction at all)

→ Result = nutcracker shape

Diffuse esophageal spasm:

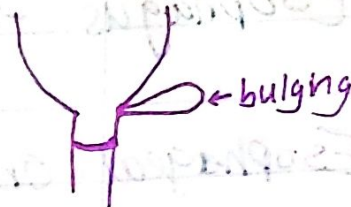
some disease

- Patient thinks here, usually it's a cardiac attack

Corkscrew esophagus

- Normal amplitude
- Lower esophageal sphincter dysfunction

Diverticulum: Bulging happens



Bulging → if it get large → a space for food → infections

recurgitation ← bad smell ← bacteria

at night

may cause also muscle → aspiration leading
relaxation food to lungs

② mechanical Obstruction:

→ something mechanically disturbing food movement, can be due to

→ Benign Stenosis

due to corrosive injury (خرب فاسد كلسه...)

Fibrosis

↓ leads to

stenosis

! لا تكونوا مبلين

→ **Radiation exposure** (or the presence of tumor inside mediastinum) → Radiotherapy → obstruction

→ **Reflux esophagitis** back movement of stomach content to esophagus due to defect in ~~lower~~ lower sphincter
↳ inflammatory process → fibrosis → stenosis → Obstruction

Webs (Esophageal web)

- "shelf like inside esophagus"
- lined by mucosa with connective tissue inside
- Seen with Reflux (see paragraph above)

Graft versus host disease (GVHD)

Condition seen after bone marrow transplantation

and **Bullous disease** skin condition that causes swellings filled with fluid (blisters)

Plummer-Vinson syndrome ثلاثي كوفن

iron deficiency anemia + **glossitis** (inflamm. of tongue)
+ **esophageal webs**

morphologically: C.T "as shelf" lined by squamous epithelium

Ring (Ring esophagus). → Schatzki Ring

Difference between ring and web?

- Web → partial extension from wall
- Ring → circumferential, shelf like structure

- part C.T and epithelium in web, but part of superficial mucosal lamina (muscularis propria) in ring

Rings

B. Ring found in gastroesophageal junction

- Lower surface lined by ^(glandular) stomach-like mucosa
- Upper surface → squamous epithelium

A. Ring - all the rest

- Lined from upper and lower surface by squamous epithelium