



Esophagitis

inflammation of esophages

Triggering factors:

- 1- Prolonged gastric intubation (البسمة بجبر الأنبط)
- 2- uremia + Bullous Diseases + inflammation (بفول، بريغ)
+ Crohn + renal failure
- 3- Ingestions of corrosives (خاصة في الأطفال أو محارون)
- 4- Iatrogenic: Radiation + chemotherapy, GVHD (الاشعاع)

↓
Stop cell division " in G.T.T.
the cells are continuously
proliferating (each 3 day the
surface epithelium replaced) "

- * In healthy person (with no triggering factors)
HSV is the cause

- * In immunosuppressed patient The causes → (CMV and fungi)

• Morphology: inflammatory cells in the esophageal mucosa.

if caused by
corrosive →
fibrosis

if the cause is virus → inclusion
body of
virus.

if Candida → microorganism and
its extensions



if we take an esophagitis
biopsy $\rightarrow$ ulcerated areas

(yellow spots) $\leftarrow$ exudate above
the ulcer

In stomach / Severe ulceration of
inflammatory process

Microscopically: neutrophil leukocyte
infiltration in squamous
epithelium

Mixture of inflammatory
cells in the lamina
propria

Reflux esophagitis

GERD

The most common cause of
esophagitis

normally food pass proximally / from
esophagus to stomach but
some defects in (the sphincter
+ peristalsis + control mechanism)
no enough tone or contraction
at the sphincter

that lead stomach contents

come back to
esophagus distal
part





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one of GERD discomfort symptoms:
Retrustersternal pain sensation

x GERD Triggering factors:

① Deficient esophageal antireflux mechanism

- ① Close of gastroesophageal sphincter
- ② normal peristalsis

② Inadequate esophageal clearance of refluxed material

③ Sliding hiatal hernia
(gastro-esophageal junction at abnormal anatomical position)

④ increased gastric volume

⑤ impaired reparative capacity

[in stomach : the rapid proliferated rate is one of the defense mechanism
So if it impaired the defense Mechanism]



Morphology

basement membrane
أول سطح من

- 1) epithelial esinophils + in epithelium
- 2) Basal Zone hyperplasia
(becomes darker) + ↑ Thickening of Basal Zone
Proliferation or inflammatory process

Scattered

esinophilic lymphocyte invade squamous epithelial

neutrophils

3) elongation of Lamina propria

Squamous epithelium (others) esophagus or skin or
Straight line basement membrane
adhesion between endothelium and subendothelium
Subendothelial + endothelial
External environment (C-T)
Papilla (C-T) extensions of lamina propria
epithelium

ridge
Ready ridge

elongation of ridges
hyperplasia of basal layer

Complications:-

- 1- Bleeding
- 2- inflammation → fibrosis
Mechanical obstruction

Stricture

3- Barrett esophagus

intestinal metaplasia

intestinal mucosa
Contain goblet cells



may become carcinoma after changes

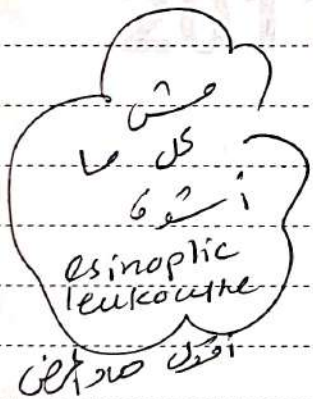


esophagus stratified squamous epithelium → metaplasia to protect against acid →



Eosinophilic esophagitis

- In atopic persons there's an increase sensitivity to the different environmental elements. Some agents affect them lead the immune system to make an exaggerated response.
- These persons have allergic reaction for one or more agents.
- ② Family clustering
- ③ eosinophilia (↑ number of eosinophilic leukocytes)
- ④ ↑ Serum level of IgE



Microscope :-

- bright eosinophilic cytoplasm
- eosinophilic leukocyte packed closely together

Scattered
نثر

Reflux
esophagitis

* esophagus by endoscope is a circular rings "feline"



Varices

• Systemic Circulation → tissue
Venous drainage to Rt side of the
heart by SVC + IVC

• Portal Circulation → from G.I.T.
Capillaries converge together to
form portal vein → enters the porta hepatis

form sinusoids
↓
drain into hepatic veins.

Portal System: Capillary System between
2 veins

في الوضع الطبيعي بين ال Portal و ال Systemic ما في اتصال بين

• لكنه في بعض النقاط زي
proximal + distal Esophagus
Stomach

intraabdominal
organ ما بين Esophagus Capillary و anastomosis في
Capillary System

↓
M. portal circulation
Systemic circulation

عادي هو
يقول ما كان
لكل له

dilation + and tortuosity of the
↑ Portal system pressure
[1 - Cirrhosis
2 - Portal vein thrombus
3 - Budd-Chiari syndrome = hepatic vein thrombosis

Capillary

Structure under the mucosa.





مؤثره ال dilated Capillary من زيادة

أننا تقل كمية كبيرة من الدم

That lead to life threatening bleeding

* Bad prognosis.

(البهايميا) Schistosomiasis → Cirrhosis → Varices
(may be esophagus)

Morphology :

لورظنا بالنظر

- dilated, tortuous Capillary structures
- exot of bleeding (blood clotting or dark-brown color a result of hemosiderin)

من أهم أسباب الفرفرية في هذا المرض rupture + massive hemorrhage

- erosion → because there's a thin wall blood vessel lined by thin layer of mucosa so if a hard food pass may tear or rupture the blood vessel or increased intravascular pressure that lead to bleeding



- Varices bleeding have the half of deaths in advanced Cirrhosis

تنزل

bleeding episode
بمعنى كل
أحداثية الوشاح منه
في سنة 15-20

- Subsides Spontaneously in 50% of cases
- 15-20% death rate for each bleeding episode

- 70% of patients who have a single bleeding episode may re-bleeding within 1 year

Barrett esophagus.

- replacement of normal epithelium by another epithelium
normal
- Studies showed that mutations of Adeno. Carcinoma "that occur after Barrett esophagus" was in the Barrett esophagus ~~metaplasia~~ mucosa before the dysplasia or the cancer formation.
- So this metaplasia is a direct precursor of cancer
- occur in 10% of patients with persistent reflux
- Male / female : 4 / 1
- Common in whites



شركة

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Similar mutations to those in esophageal adenocarcinoma particularly when dysplastic

• Vast majority of adenocarcinoma are associated with Barrett in contrast most cases don't develop tumor

esophageal mucosa is white

Morphology:-

• tongue mucosa extending upward from the gastric mucosa

• May be short or long segments
mm cm

Microscopically:

intestinal type mucosa with columnar absorptive cells and goblet cells
↓
mainly

ولذلك نشبه أنه ما في أي Dysplastic changes

Stomach acid irritation

(dense inflammatory process)

adenocarcinoma



normal esophagus mucosa

حالياً لو بنينا الصور خلاصة

أمر و أبيض
↓
و من ي

وجود Barrett



Esophageal Cancer :-

- Common in some areas
- Some Cancer as Barrett related adenocarcinoma has an exponential increase in number of affected patients.

مرتبط بـ

عدد الناس كل سنة يزيد

1. GERD
2. food types
3. stomach overdistention
4. Smoking
5. obesity

* Most Common Esophageal Cancer is SCC + others from mesenchymal origin
most common is leiomyoma

* Adenocarcinoma :-

- Typically on Barrett and long standing GERD + obesity + tobacco + radiation
- 7 fold in men
- Risk reducing factors: diet + some serotypes of H. pylori

They tend to cause atrophy which ↓ HCL secretion → So if there's Reflux but



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None

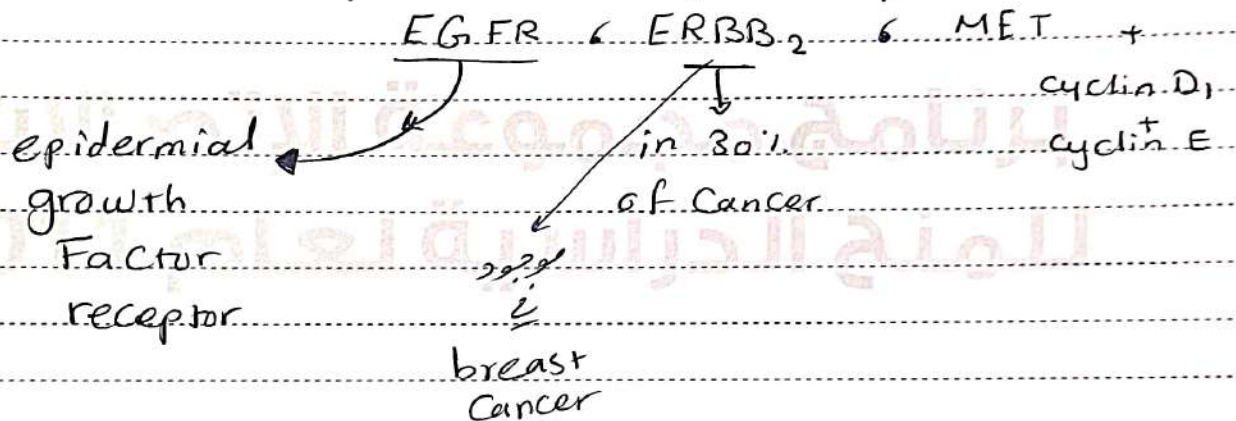
mutations
not for
certentype
of cancer

AdenoCarcinoma Pathogenesis :-

- Early stages → mutation of tumor suppressor genes as
Tp53
CDKN2A

With progression

Amplification of oncogenes as



bad prognosis

Morphology : mass located at the Barrate esophagus area

rich in blood supply + lymphatic

The mass may be exophytic or infiltrative or ulcerated "depressed"

bulging toward the lumen

Exophytic pattern lead to obstruction
infiltrative pattern lead to obstruction

زي نوردان



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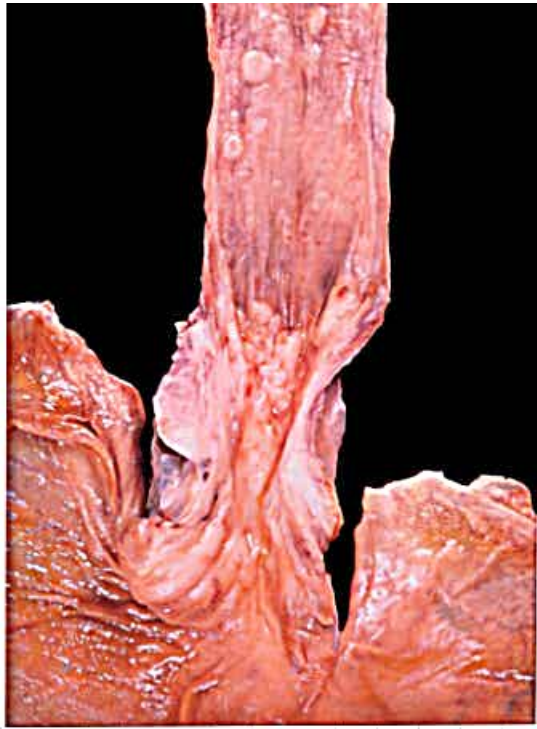
@PGFoundation

Cancer associated fibrosis

Strong desmoplastic reaction

CT formation → rigidity

Cancer جي بڻجڻ



an esophageal autopsy

① it's a full
Circumferential

② Occupied by
ulcerated or fungated
vegetative invasion pattern
that invade
the entire wall

⑤ esophagus width
decreased → Stenosis

the patient will
have dysphagia



SCC :

- in elderly (> 45 year)
- Male / Female 4 / 1

Risk factor :
 in western world
 alcohol
 Smoking
 and some poorly understood factors

- poverty
 نسبيته في مناطق البنية كالمصر
- Caustic injury
- Skin burnes → Cancer)
 مقلوبة
- Achalasia
- plummer - Vinson Syndrome
- Frutis - vegetables poor diet
- hot beverages
 الشربيات الساخنة جداً
- Radiation
 regular ingestion

* Pronounced variability in international incidence
 (endemic في بعض المناطق)
 في مناطق معينة

In Kenya the SCC rates are high because of the consumption of a traditional fermented milk "mursik" contains the Carcinogenic (Acetaldehyde)

* area where there's a high SCC there's a Carcinogenic or fungal agent contamination.



SCC pathogenesis :-

① Recurrent genetic abnormalities

↓ as

- A) Amplification of SOX2
- B) overexpression of cyclin D1

- * $\frac{1}{2}$ Cases are at the Middle third of the esophagus
- * may preceded by dysplastic changes

- * Macroscopy : exophytic growth pattern
or
infiltrative "
+
ulceration

- * Rich lymphatics , circumferential and longitudinal spread

- * Metastasis according to tumor location
if in proximal $\xrightarrow{\text{فوق}}$ supraclavicular + neck lymph nodes
if in middle $\xrightarrow{\text{وسط}}$ mediastinum
trachea + major blood vessel
if in distal $\xrightarrow{\text{تحت}}$ in celiac lymph nodes.



is an ulcerative tumor
and infiltrative rather than
exophytic mass with extensions



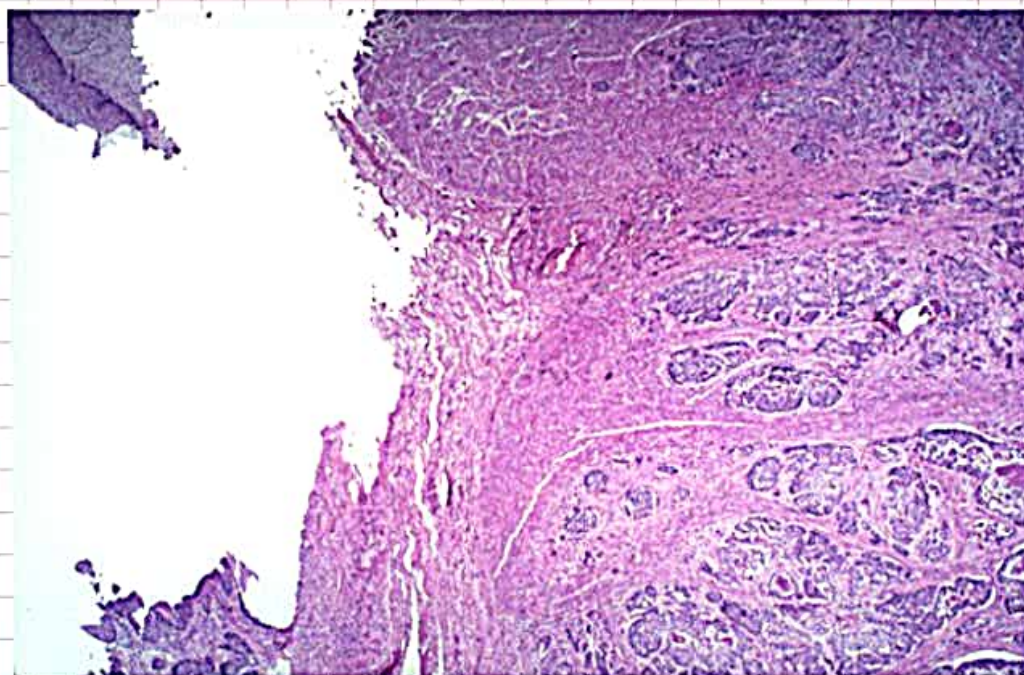
is an exophytic
mass in the
middle part



under the
microscope
there're groups
of malignant
cells.

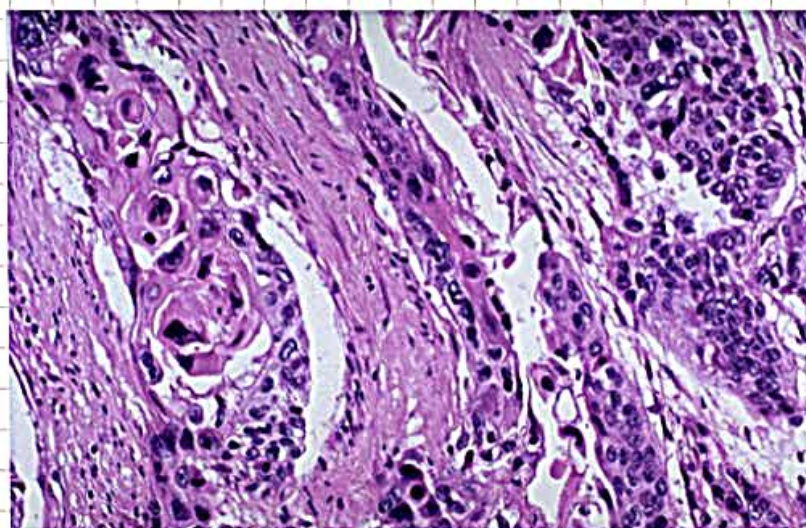
1. hyperchromatic nuclei
2. polymorphism
3. abundt eosinophilic cytoplasm

keratin
+ formation
(bright eosinophilic areas)



infiltrative growth pattern :

there's an ulceration above the normal mucosa + There are group of cell invading through the desmoplastic C.T



Nuclear changes

لون غامق

- hyperchromsine
- polymorphism (تفاوت في حجم النواة)
- nuclear irregularity