Lec 1

GIT PATHOLOGY

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Oral pathology

1- Aphthous ulcer

-It is a common, self limiting condition.

-It is small(less than 5mm), painful, shallow, ulcer.

-It affects the oral mucosa, soft palate, buccal and labial mucosa, floor of the mouth.

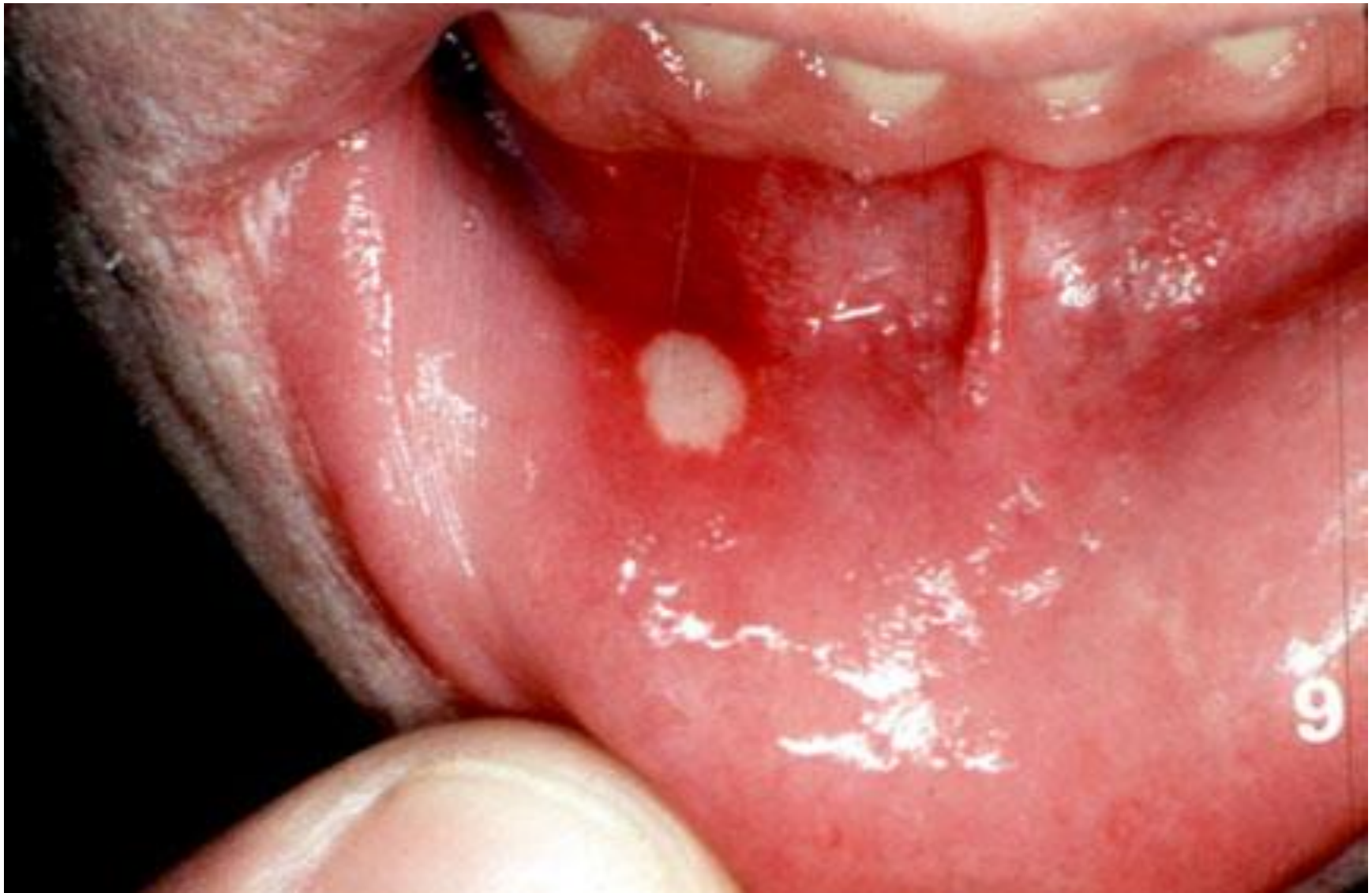
-It is round, covered by gray- white exudate with erythematous rim.

- Etiology:

Unknown, but it is triggered by stress, fever, ingestion of certain food….etc.

Autoimmune base is suspected.

Aphthous ulcer



2- Herpes simplex virus infection

-Is caused by HSV type I

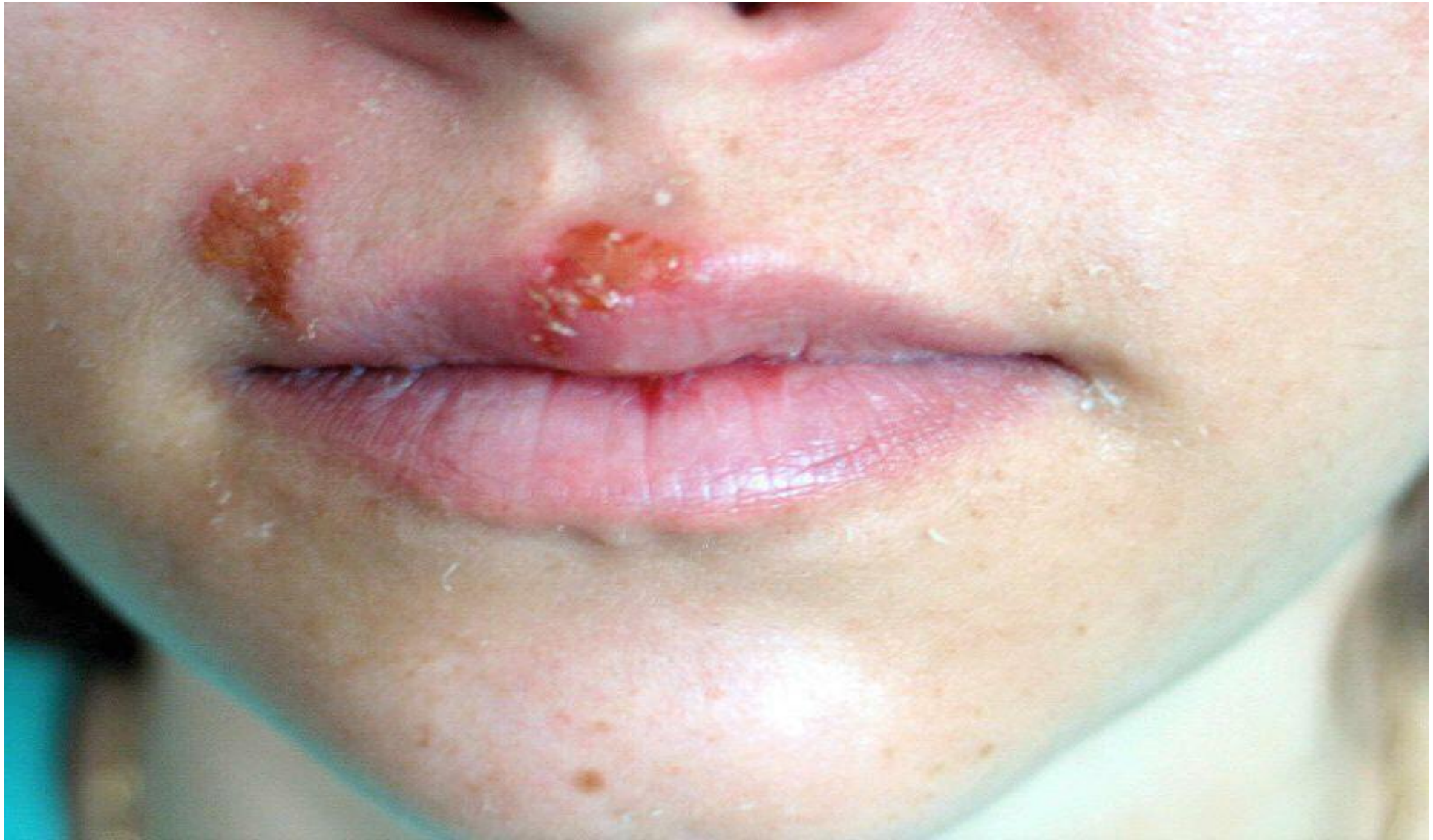
-Called fever blister or cold sore

-the primary infection is usually asymptomatic, as a result of it the virus will persist in a dormant state within the ganglia about the mouth(e.g. trigeminal)and get reactivation by:

* Upper respiratory tract infection.
* Excessive cold
* fever
* Sunlight
* trauma

This will lead to the formation of small vesicles which rupture and leave a shallow painful ulcer located around the mouth, lips &nasal orifices.

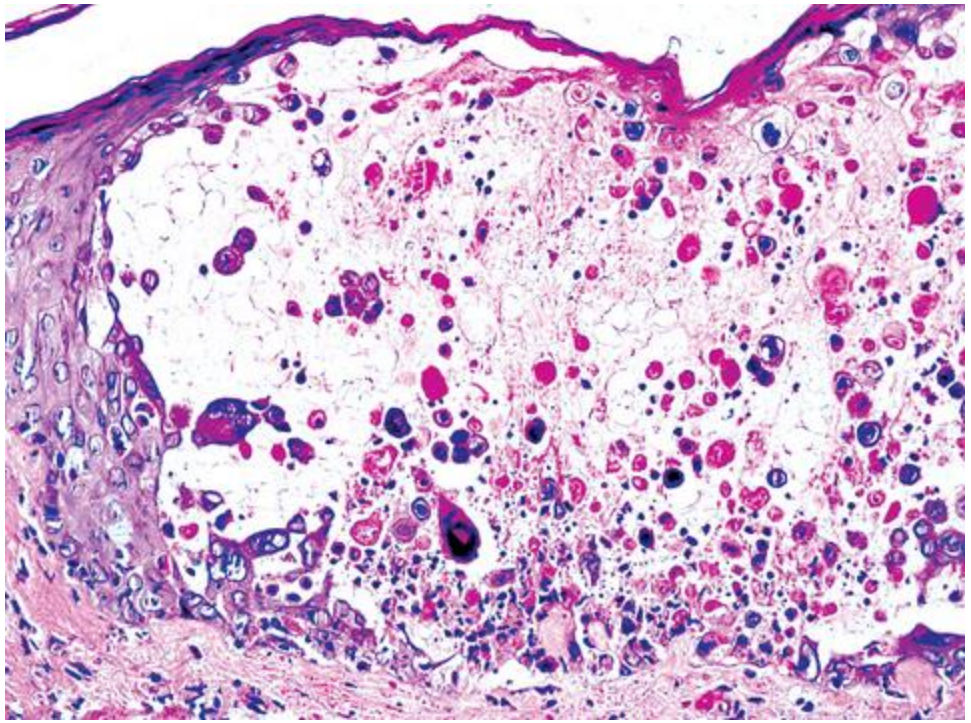
Herpes labialis

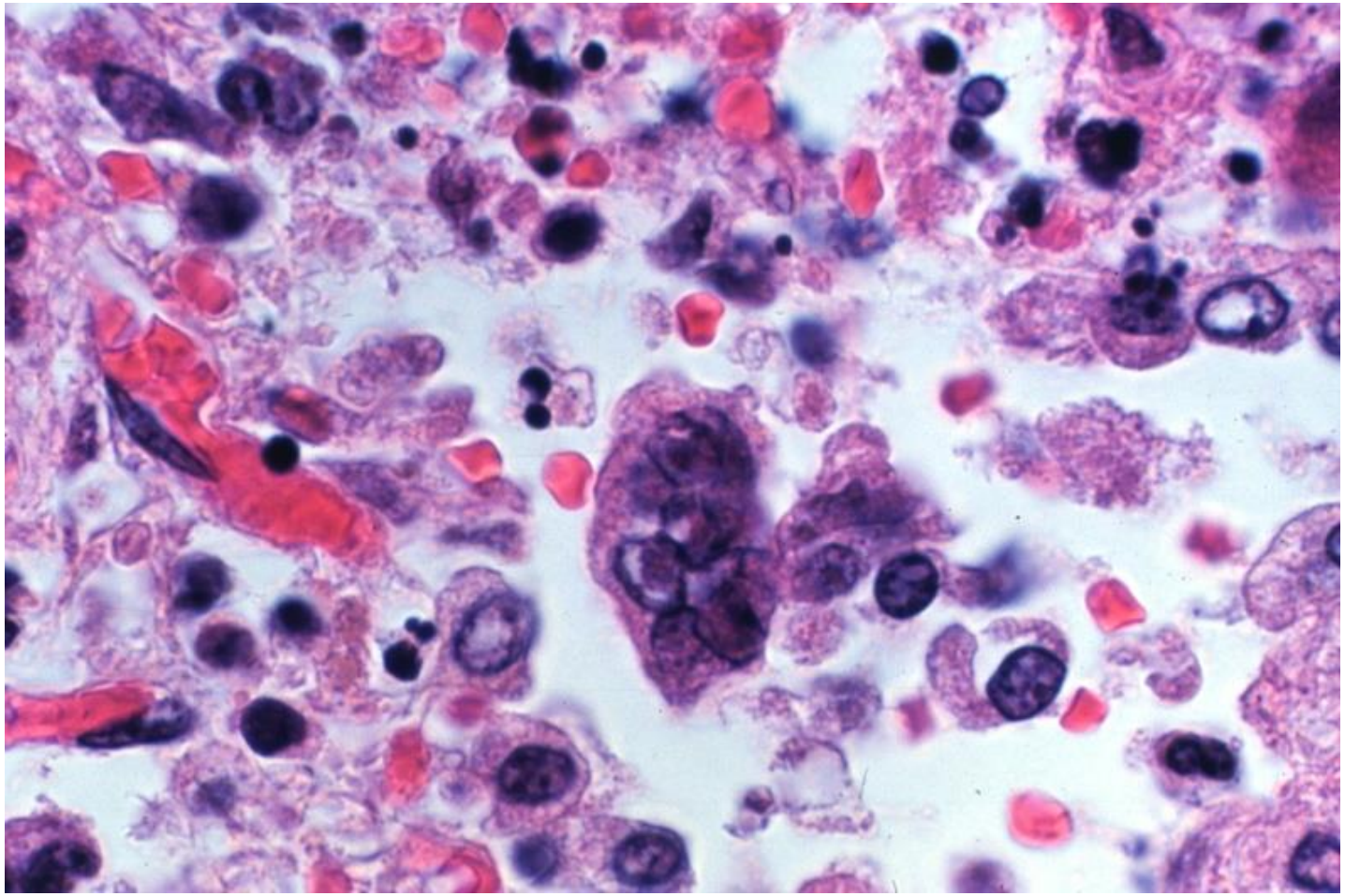


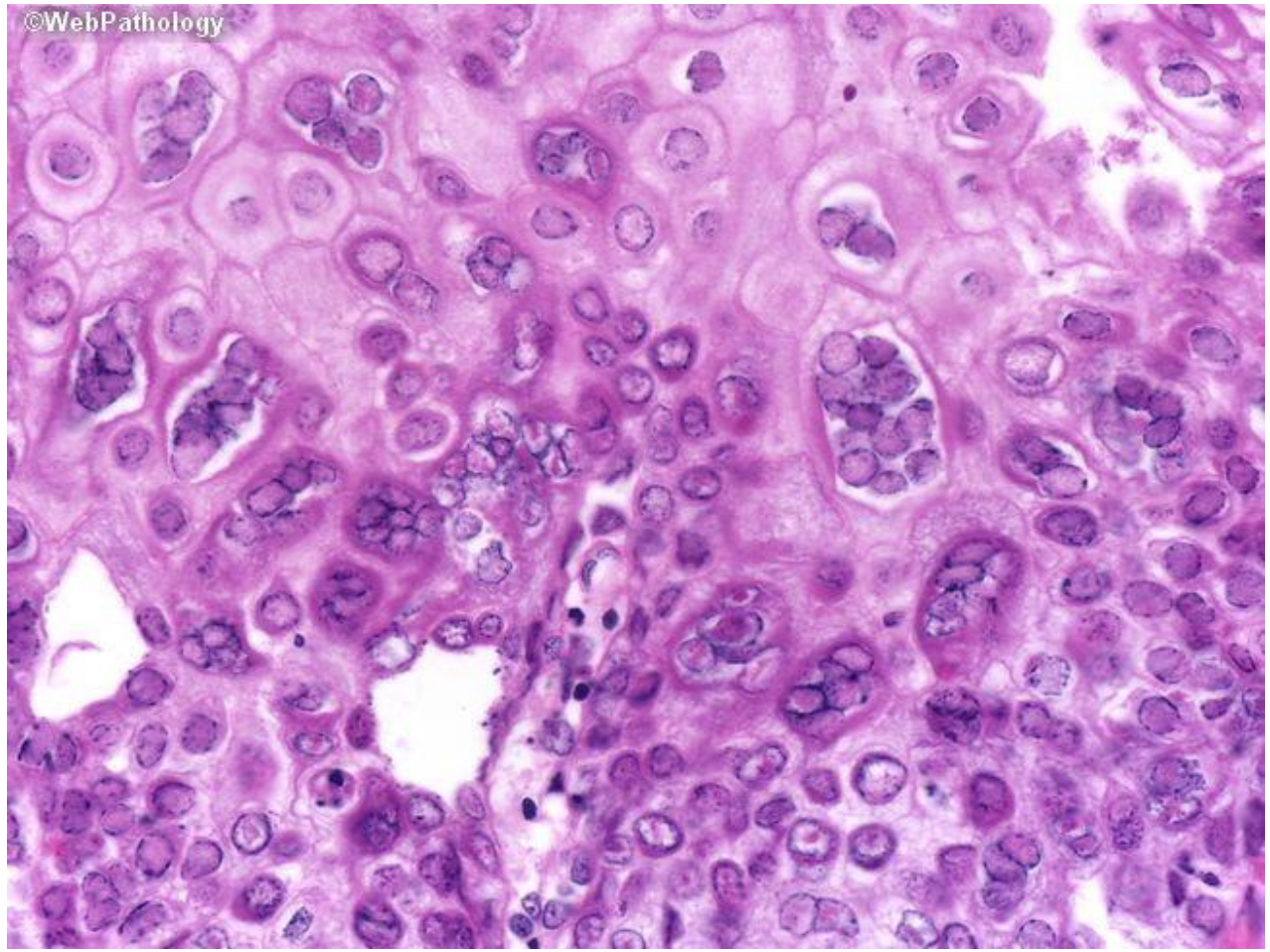
Microscopically:

The infected cells become ballooned and have large eosinophilic intranuclear inclusions. Adjacent cells commonly fuse to form large multinucleated cell.

Keratinocytes are multinucleated, acantholytic with distinct nuclear inclusions,



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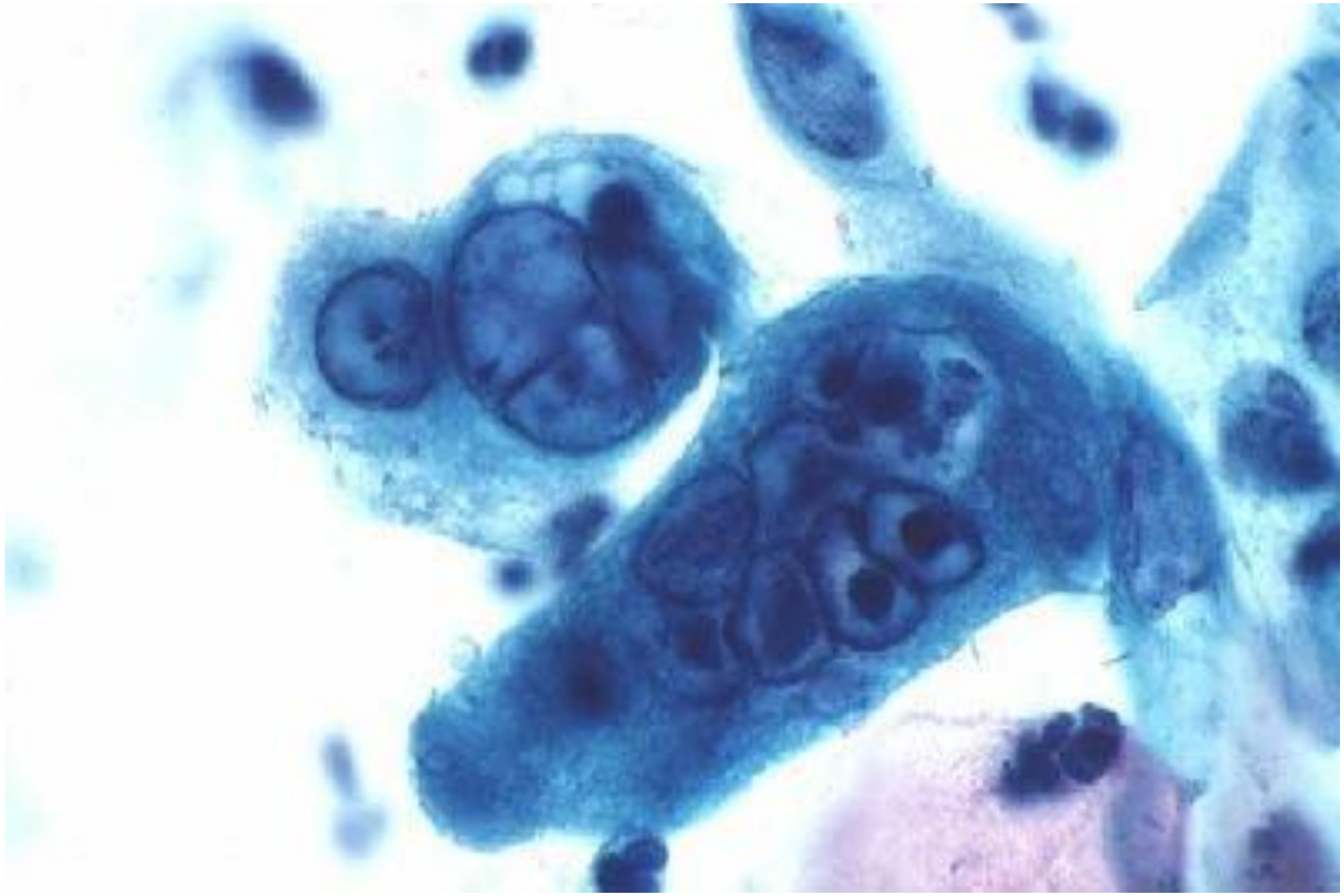
<u>3Ms of Herpes</u>: Molding, Margination of chromatin and Multinucleation



3- Oral candidiasis

Causative agent: Candida albicans

Gross: Adherent white curd like plaque (fibrino-suppurative exudate with matted micro-organisms) which is after scrapping will reveal an erythematous inflammatiry base base.

- **Etiological factors:**
- It is opportunistic infection, so it will appear if there is any decrease in immunity

e.g Diabetes mellitus,

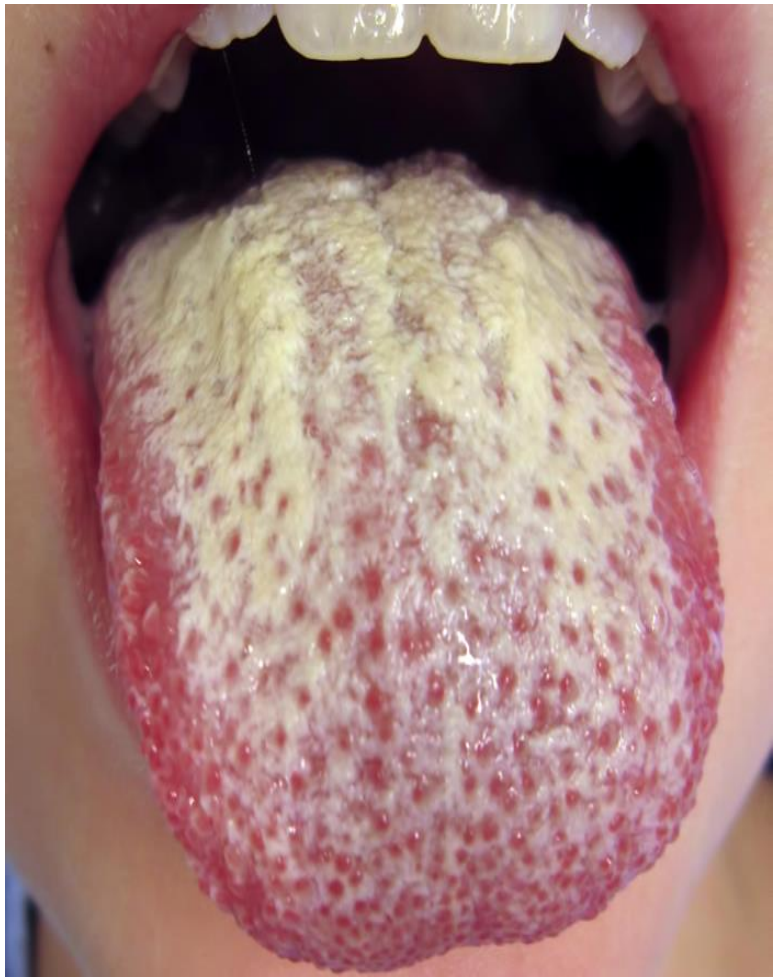
Immunodeficiency(congenital or acquired),

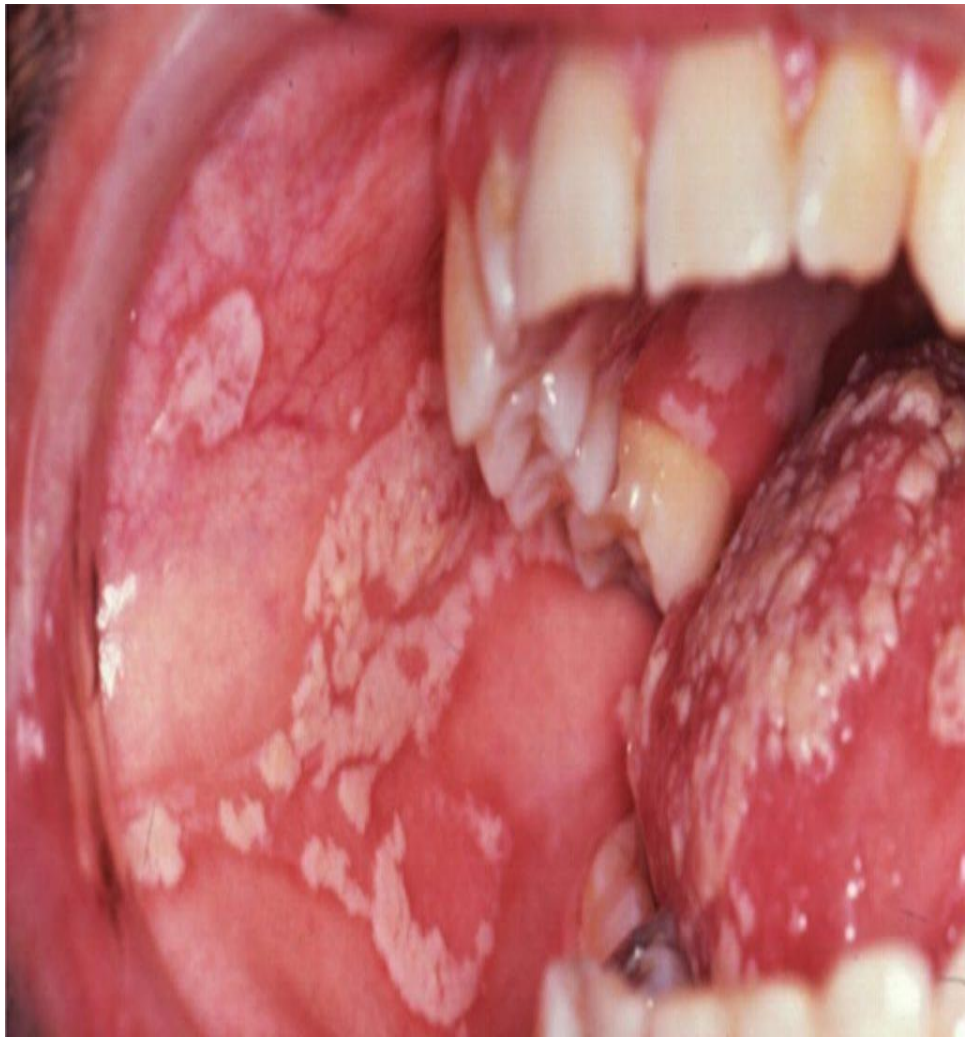
anemia,

antibiotic or glucocorticoid therapy,

debilitating dis e.g CA &AIDS.

oral candidiasis





4- Leukoplakia

Is a white, well defined, oral mucosal patch which can't be removed by scraping.

It is a clinical term and not a disease entity.

Microscopically:

*Hyperkeratosis or

* dysplasia (mild-sever) or

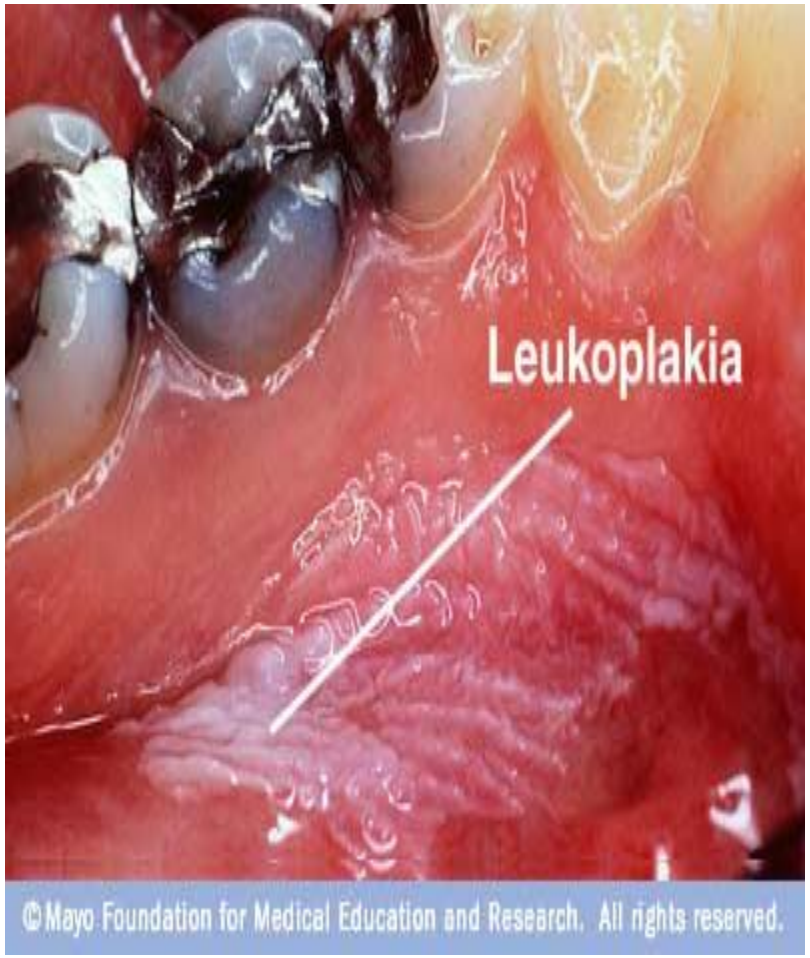
*5-6% carcinoma in situ or invasive carcinoma.

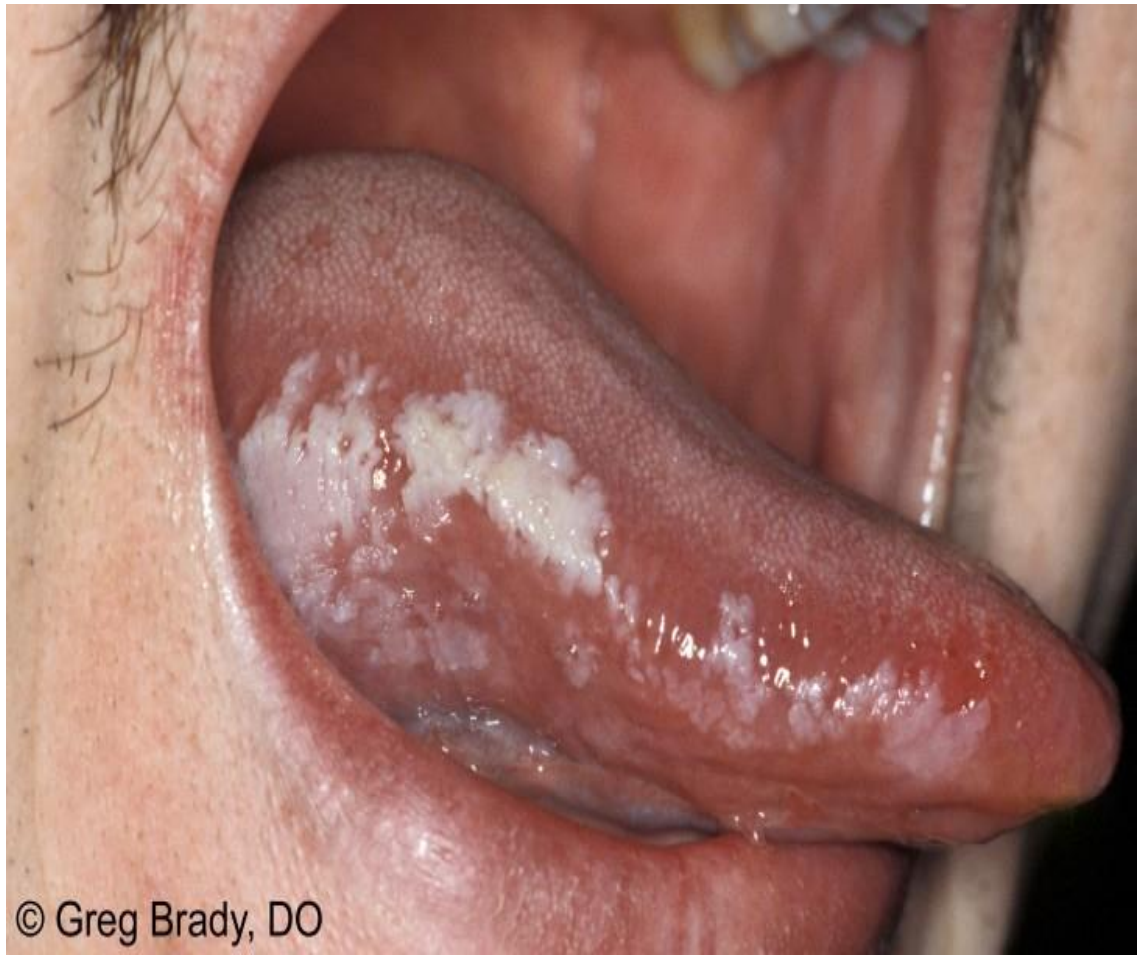
Etiological factors:

The most important: Tobacco, HPV

Others: alcohol, ill fitting dentures, irritant food.

Oral leukoplakia





Approximately 3% of the world’s population has leukoplakic lesions,

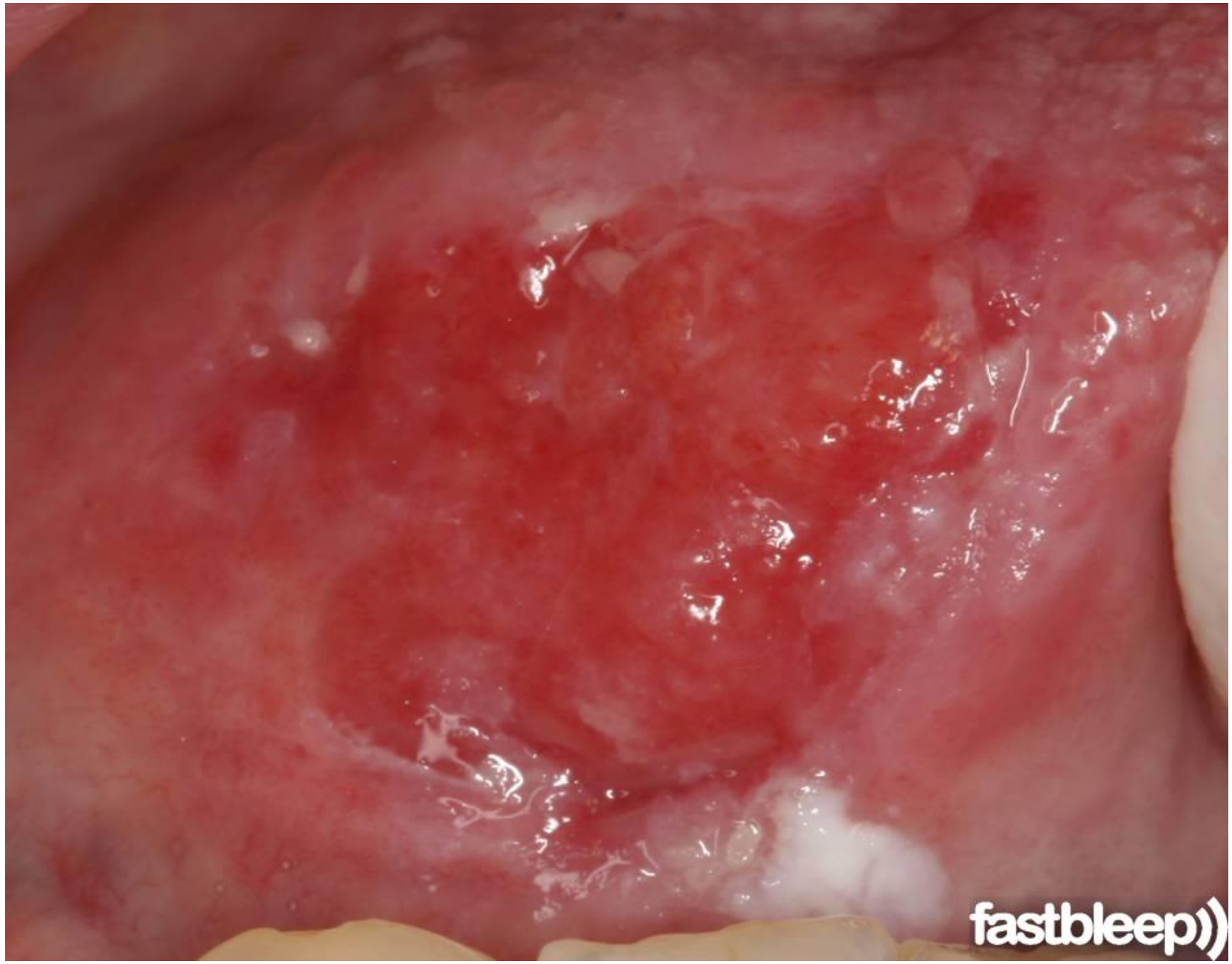
of which 5% to 25% are premalignant and may progress to squamous cell carcinoma. Thus, all leukoplakias must be considered precancerous, until proved otherwise by means of histologic evaluation.

Erythroplakia

is a red, velvety, possibly eroded area that is flat or slightly depressed relative to the surrounding mucosa.

Histologically: marked dysplasia

Erythroplakia is associated with a much greater risk of malignant transformation than leukoplakia(around 50%).

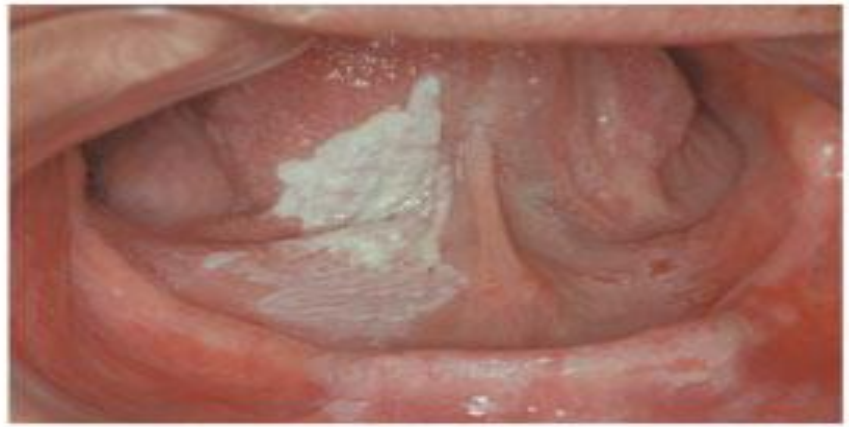

fastbleep

Age : typically affect persons between the ages of 40 and 70 years (it may be seen in adults at any age)

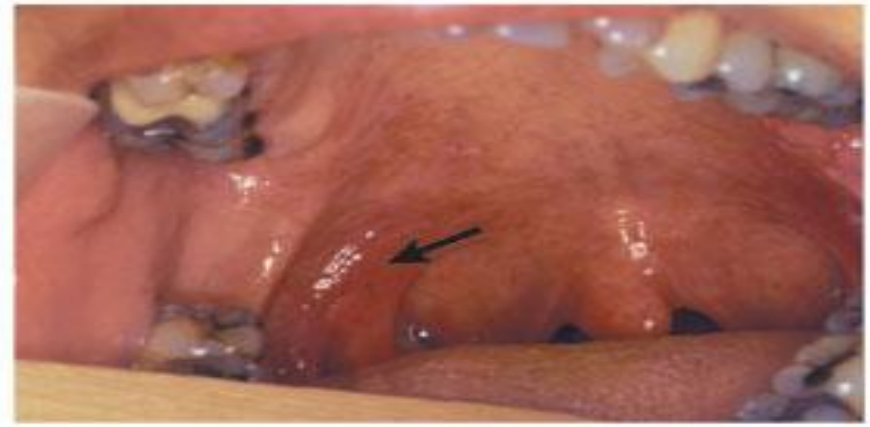
male :female ratio is 2 : 1

Although the etiology is multifactorial, tobacco use (cigarettes and chewing tobacco) is the most common risk factor for leukoplakia and erythroplakia.

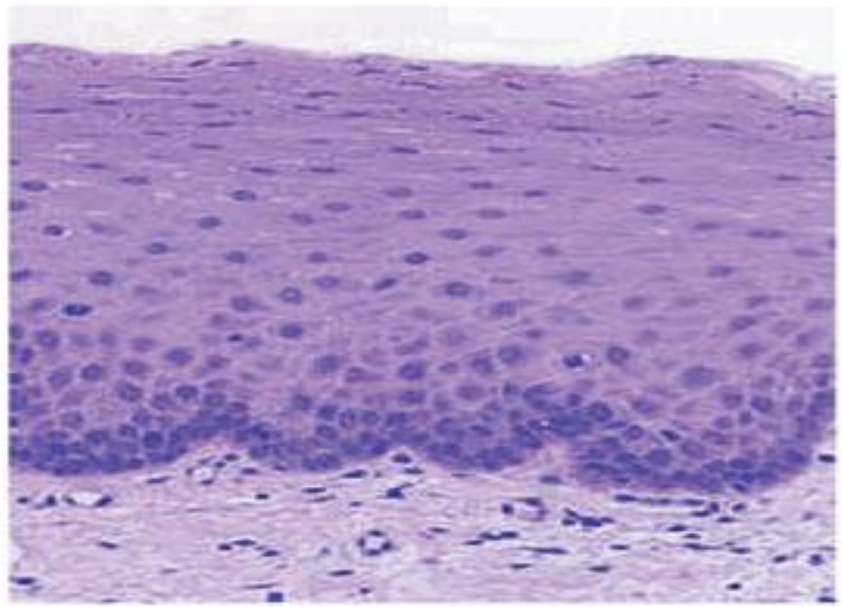
a Leukoplakia



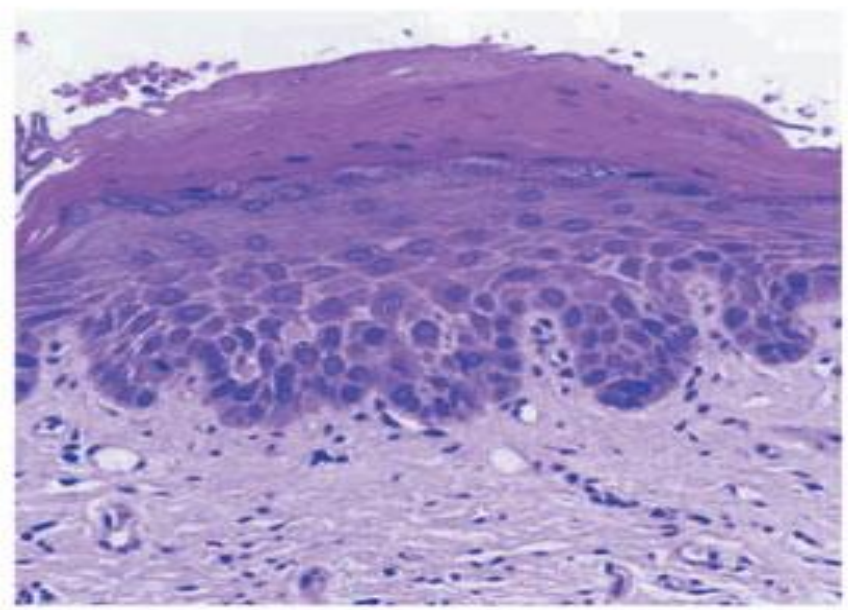
b Erythroplakia



c Normal oral mucosa



d Moderate dysplasia



5- Carcinoma of the oral cavity

95% are squamous cell carcinoma.

Predisposing factors:

- leukoplakia (the risk of transformation is 3-25%),
- Erythroplakia (the risk of transformation is 50%)
- tobacco
- human papilloma virus 16 &18
- alcohol

Pathogenesis:

1-Mutations frequently involve P53 or p63

2-Infection with oncogenic variants of human papillomavirus (HPV), particularly HPV-16

Oral squamous cell carcinomas are classically linked to tobacco and alcohol use, but the incidence of HPV associated lesions is rising.

Grossly:

In early stages, these cancers can appear as raised, firm, pearly plaques or

as irregular, roughened mucosal thickenings. Either pattern may be superimposed on a background of a leukoplakia or erythroplakia.

As these lesions enlarge, they typically form ulcerated and protruding masses that have irregular and indurated or rolled borders

Microscopically

A majority of oral cavity cancers are squamous cell carcinomas with different grades(well, moderate or poorly differentiated).

- **Site:**

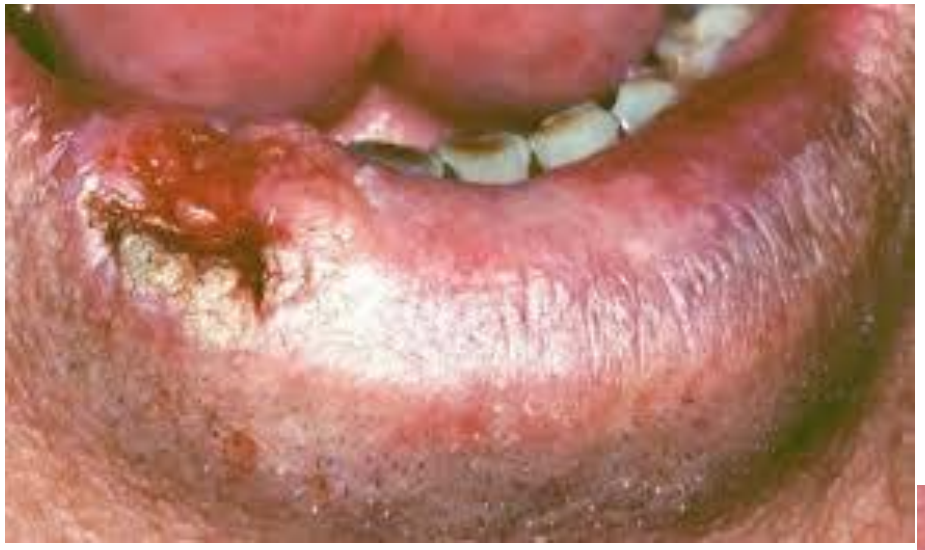
Lower lip, floor of the mouth, tongue, hard palate, base of the tongue .

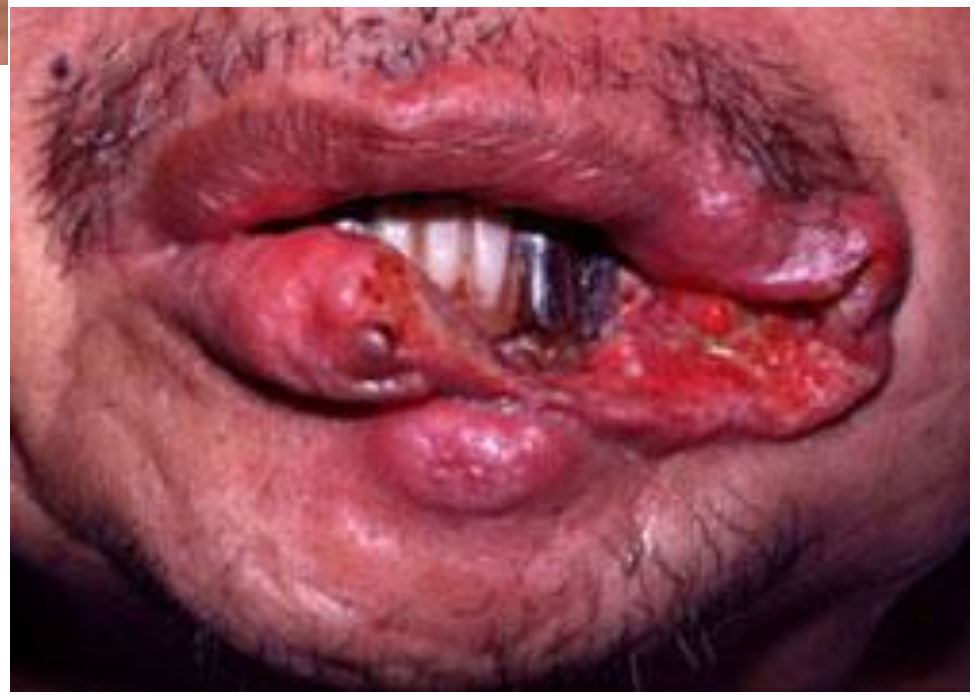
- **Prognosis:**

Best, in lips lesion

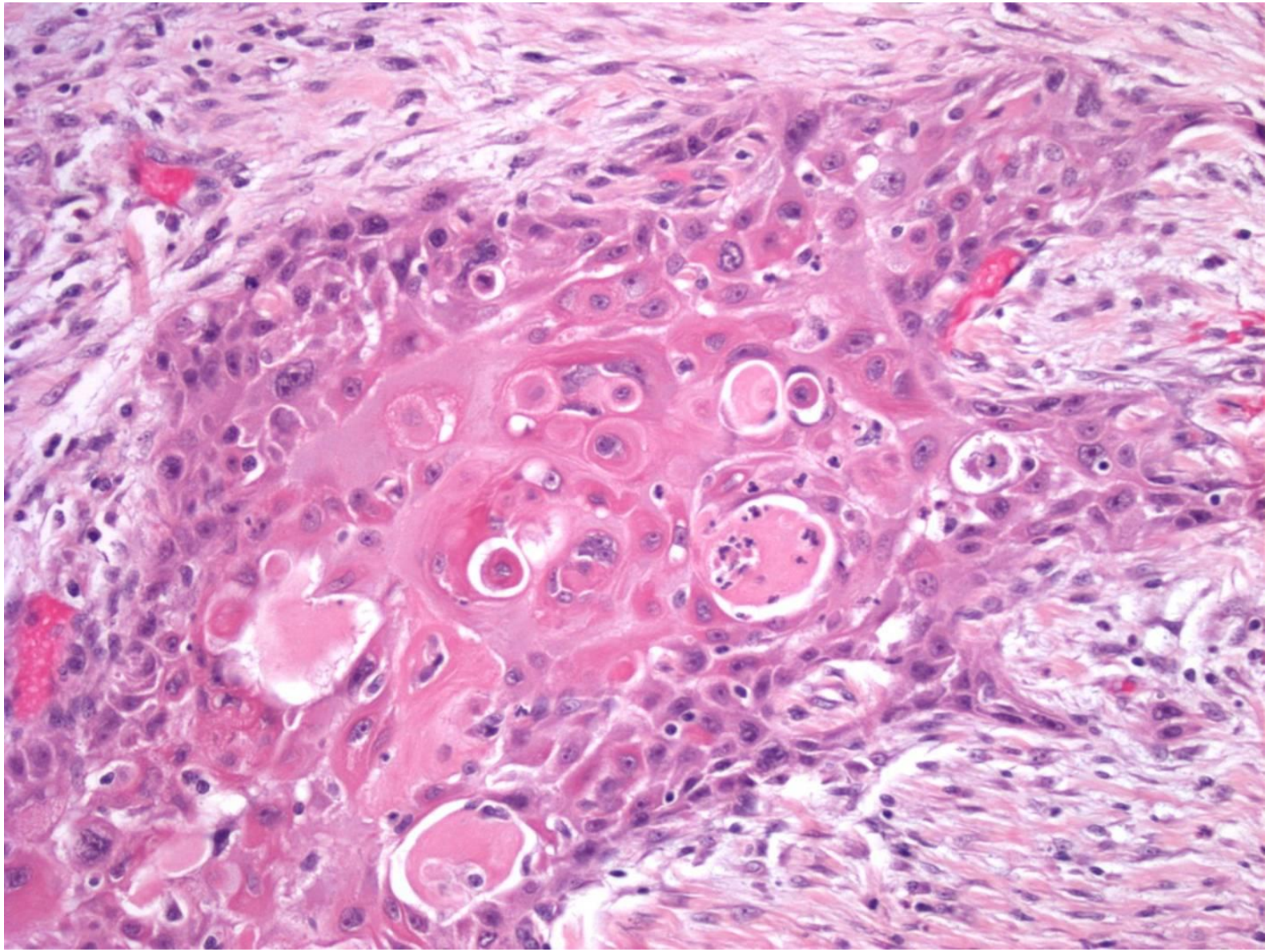
Poor, in floor of the mouth & tongue

Squmous cell carcinoma (lip)





Mic. :invasion of nests of keratinocyte (desmosome and keratin



The Salivary gland

Sialadenitis (inflammation)

Causes: Either: Viral, bacterial, or autoimmune.

1- Viral: the most common virus is mumps virus. which affect mainly the parotid gland.

In children it is self limiting disease.

In adult it may be accompanied by pancreatitis or orchitis causing sterility.

2- Bacterial: Which is mainly unilateral involvement, painful enlargement.

Occur following an obstruction of major excretory duct by e.g stone (sialolithiasis).

Following major surgical procedure in old dehydrated patients.

Most common causative bacteria are staph. aureus &strep. viridans

3- Autoimmune

usually bilateral

seen in Sjogren syndrome : in which there is wide spread involvement of salivary glands, mucous secreting and lacrimal glands causing :

a- Dry mouth (xerostomia)

b- Dry eye (xeroconjunctivitis sicca)

c- Small % may evolve to malignant lymphoma

- **Tumors**

About 80% of the salivary gland tumors occur in the parotid gland.

The **larger** the gland, the **less** likelihood to be malignant .

Sublingual : 50% of the tumors are malignant

Submandibular gland: 40% are malignant

Parotid :15-30% of the tumors are malignant

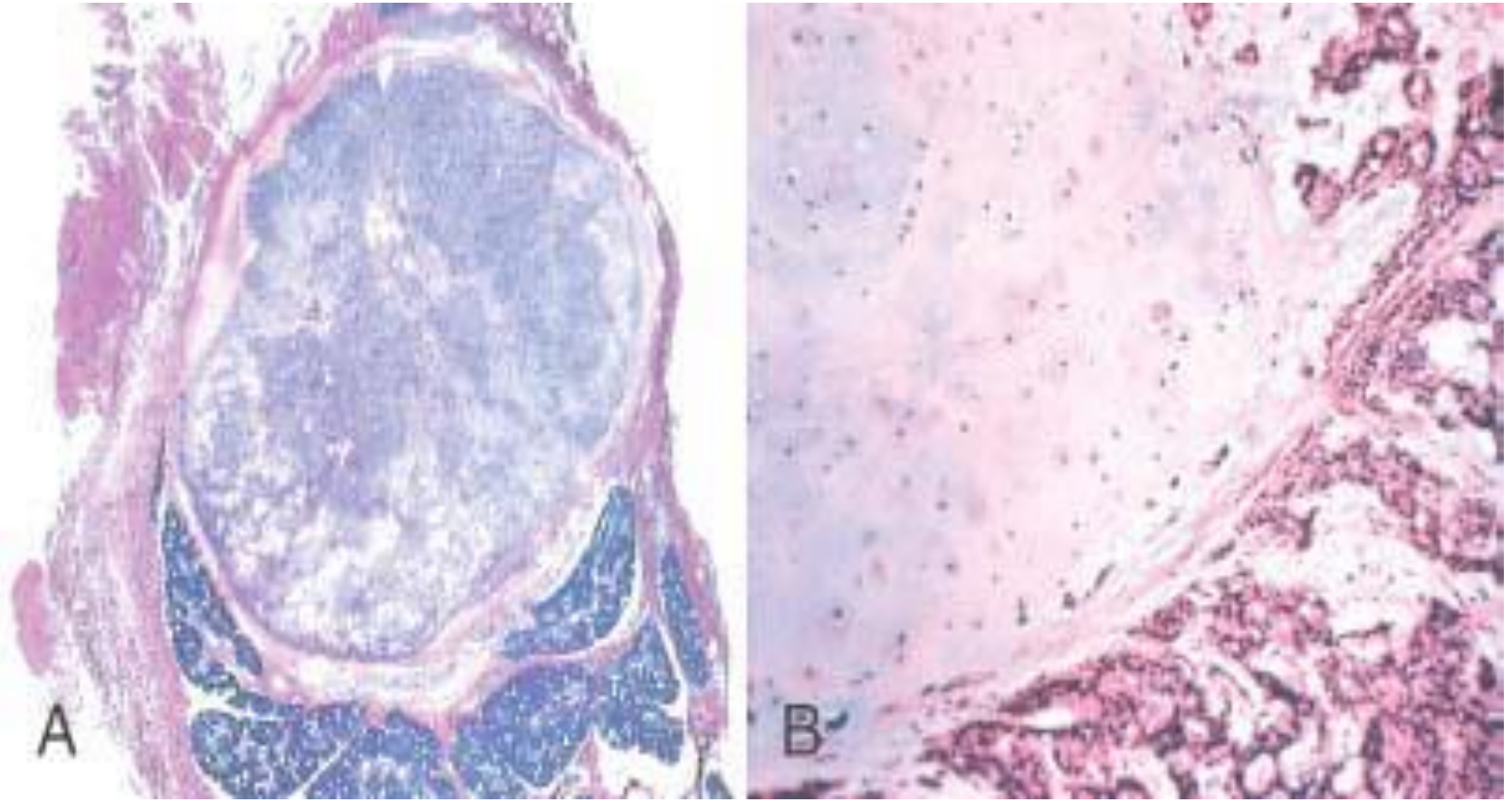
- **Benign tumors**: e.g pleomorphic adenoma (mixed tumor)&Warthin tumor
- **Malignant tumors**: e.g mucoepidermoid carcinoma, adenoid cystic carcinoma

- *Pleomorphic adenoma (benign mixed tumor of salivary gland):*
- Account for 90% of benign tumors of salivary glands
- Causes painless swelling at the angle of the jaw (palpable discrete mass)
- Grossly encapsulated but histological examination reveals capsular penetration which necessities adequate resection margins to prevent recurrence which occur in 10%.

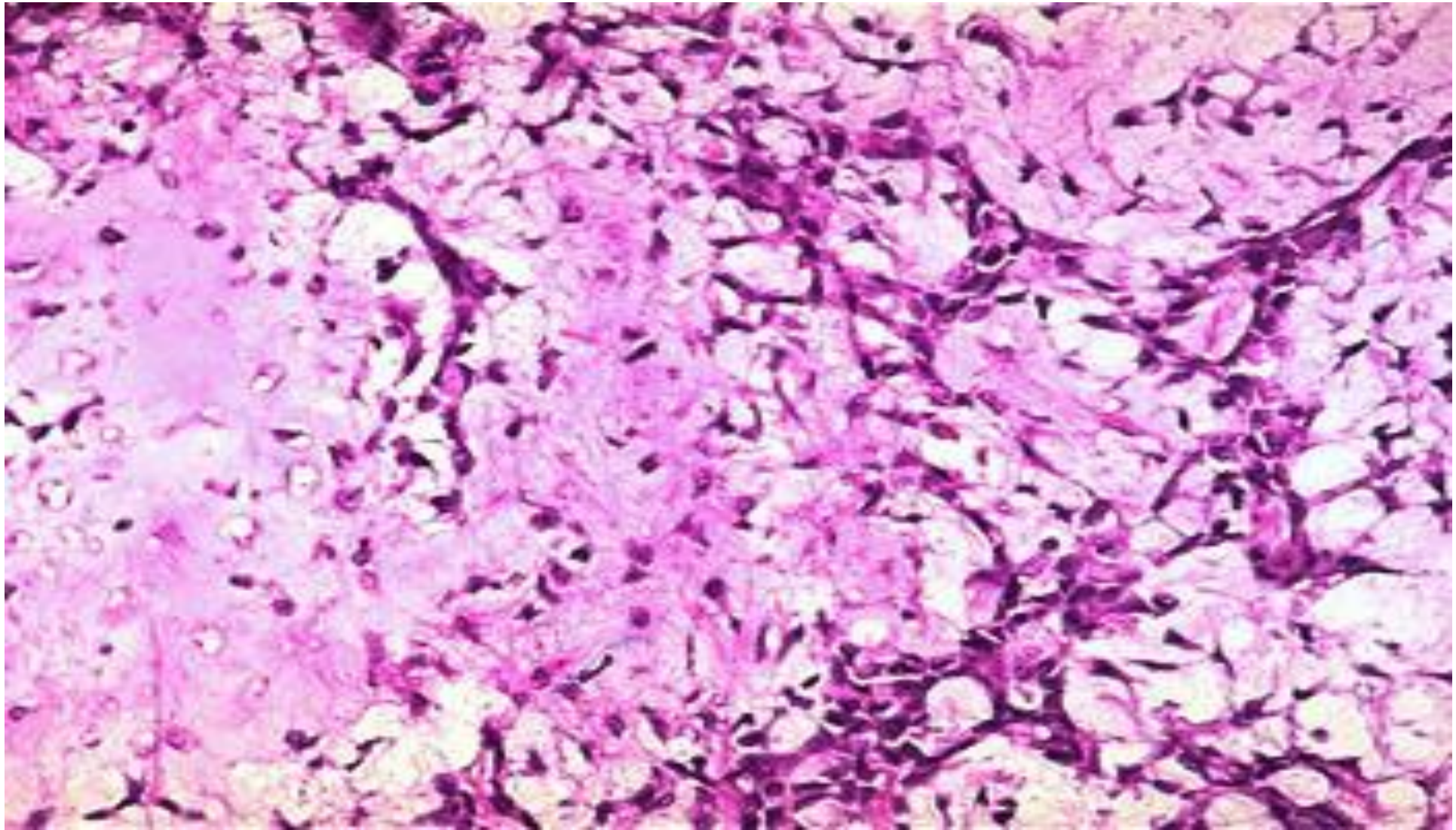
- Microscopically: The characteristic feature is heterogeneity, tumor cells are small &dark &form ducts, acini, tubules, strands or sheets &these cells intermingled with loose myxoid connective tissue stoma containing cartilage &rarely bone.

Pleomorphic adenoma (parotid gland)

Pleomorphic adenoma. *A,* Low-power view showing a well-demarcated tumor with adjacent normal salivary gland parenchyma. *B,* High-power view showing epithelial cells as well as myoepithelial cells found within a chondroid matrix material.



Pleomorphic adenoma (parotid gland)



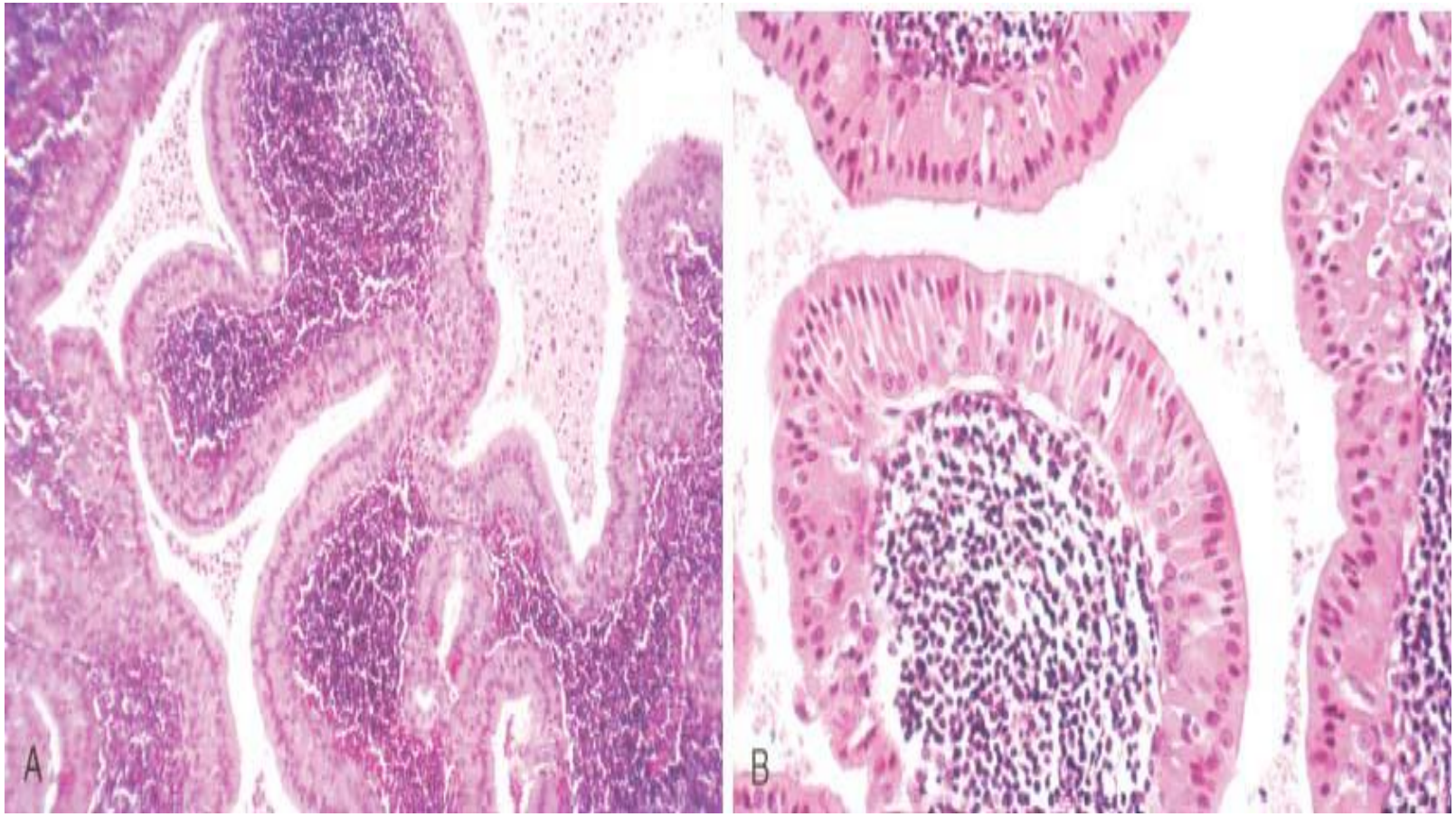
- ***Warthin Tumor***
- Infrequent benign tumor arises from heterotopic salivary tissue trapped within a regional lymph node during embryogenesis.
- It is small, well encapsulated, round to ovoid mass that on section reveal mucin containing cystic spaces within soft gray background.

- **Microscopically:**

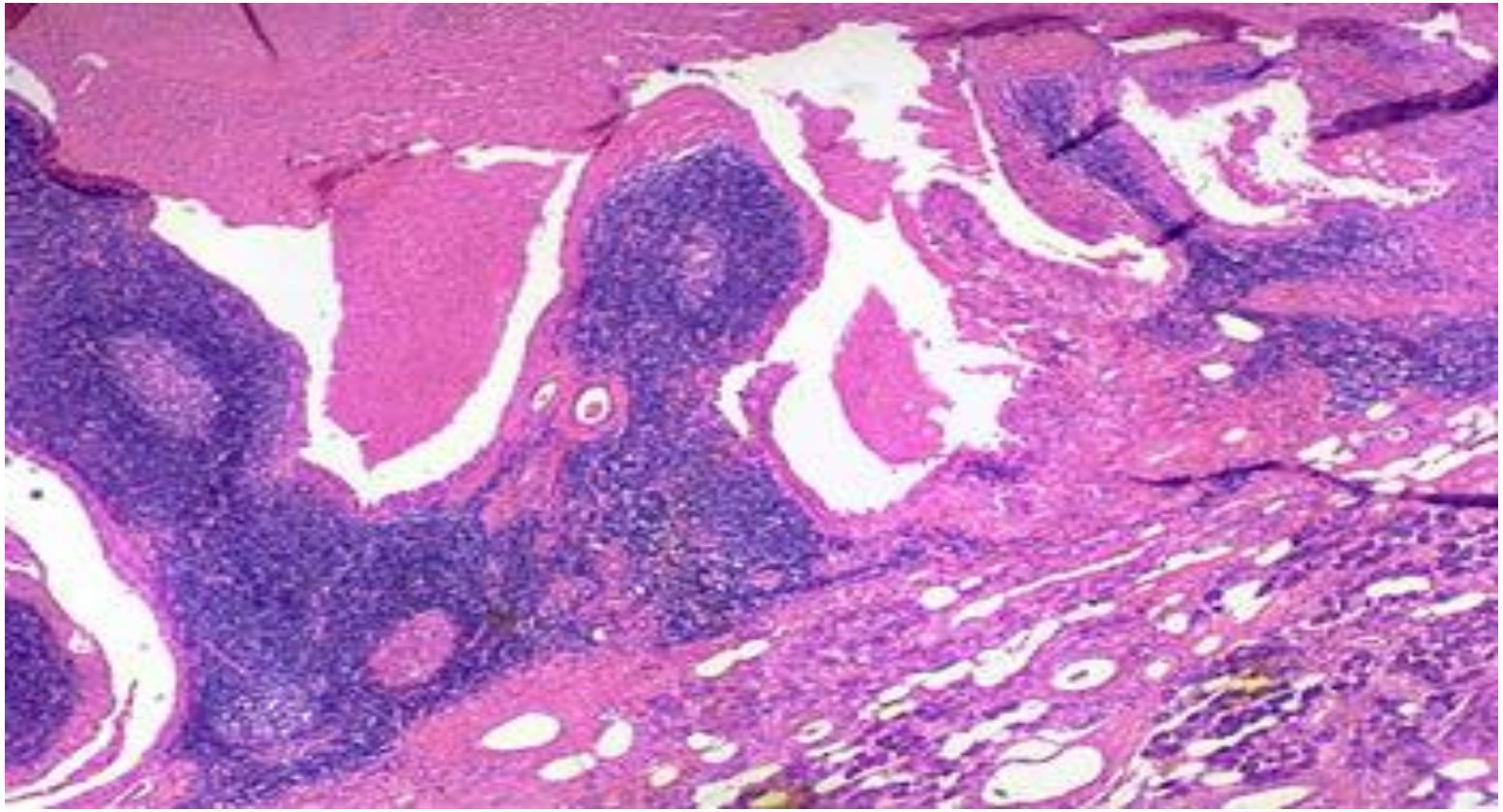
1- Two tired epithelial layer lining the branching cystic or cleft like spaces.

2- An immediately subjacent well developed lymphoid tissue sometimes with germinal centers

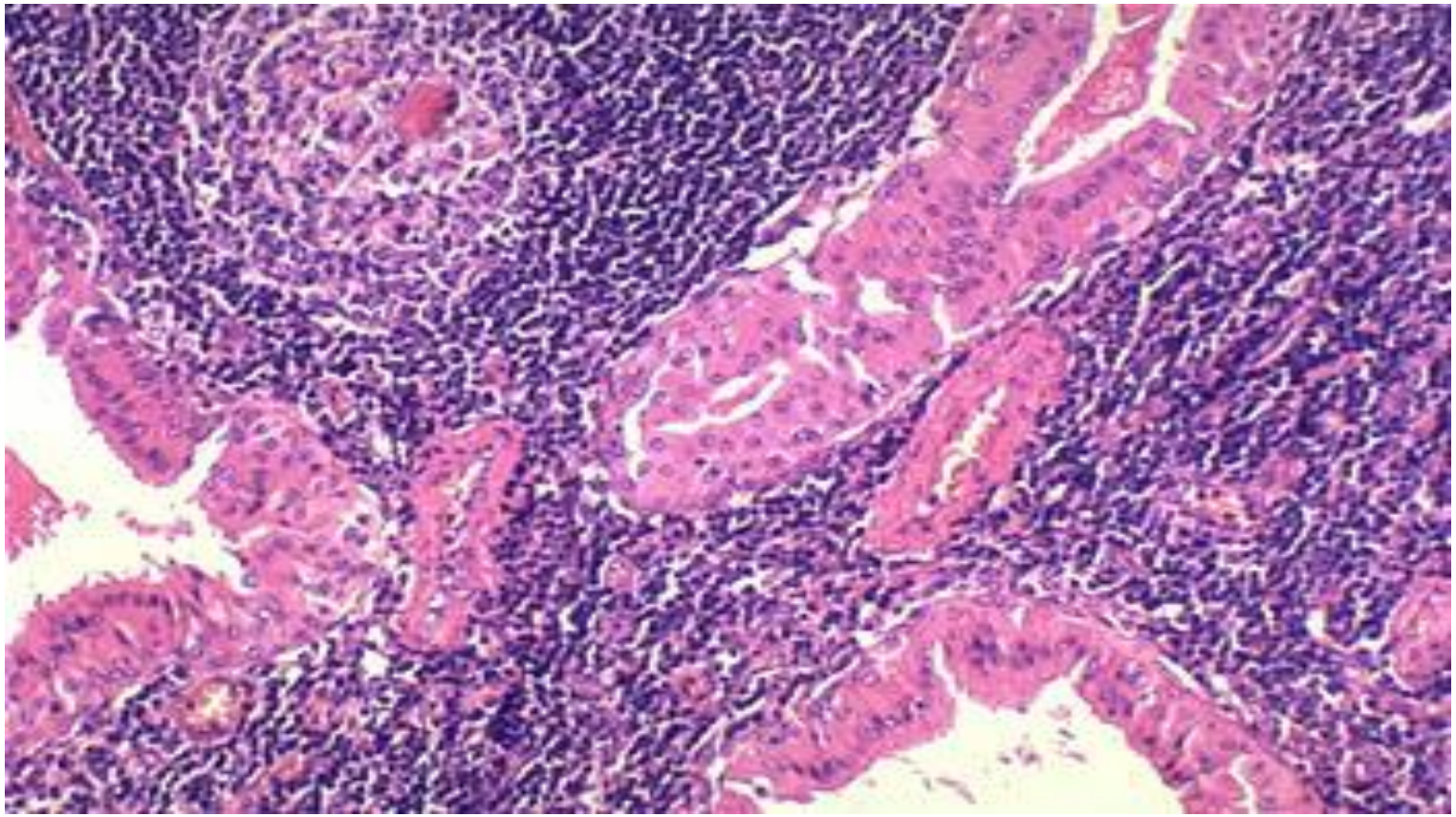
Warthin tumor. A, Low-power view showing epithelial and lymphoid elements. Note the follicular germinal center beneath the epithelium. B, Cystic spaces separate lobules of neoplastic epithelium consisting of a double layer of eosinophilic epithelial cells based on a reactive lymphoid stroma.



Warthin tumor



Warthin tumor



Esophagus

The Esophagus

It is a muscular tube of 23-25 cm, **its function** is to

1-conduct food and fluid from the pharynx to the stomach

2- Prevent reflux of gastric content by the presence of two physiological sphincters:

* Upper esophageal sphincter (UES) at the cricopharyngeal muscle
* Lower esophageal sphincter (LES) proximal to the esophageo-gastric junction.

- **Microscopically:**

1- Mucosa: lined by non keratinizing squamous epithelium.

2- Lamina propria

3- Submucosa: containing glands

4- Muscularis propria:

5- No serosal coat

Lined by stratified squamous epithelium

Muscle coat:

- Inner circular (Thinner)
- Outer longitudinal (Thicker)

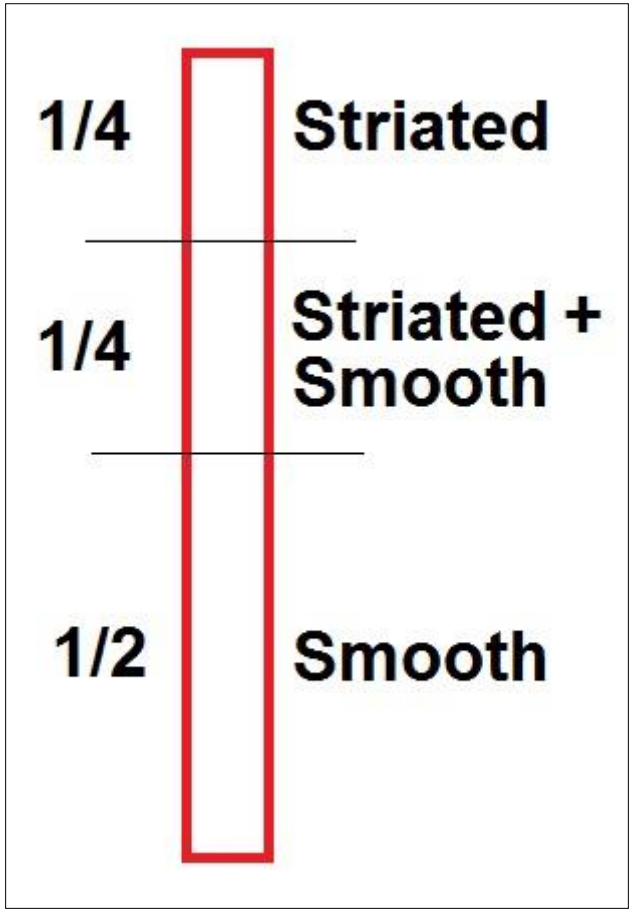
Upper portion – voluntary

Lower portion – involuntary

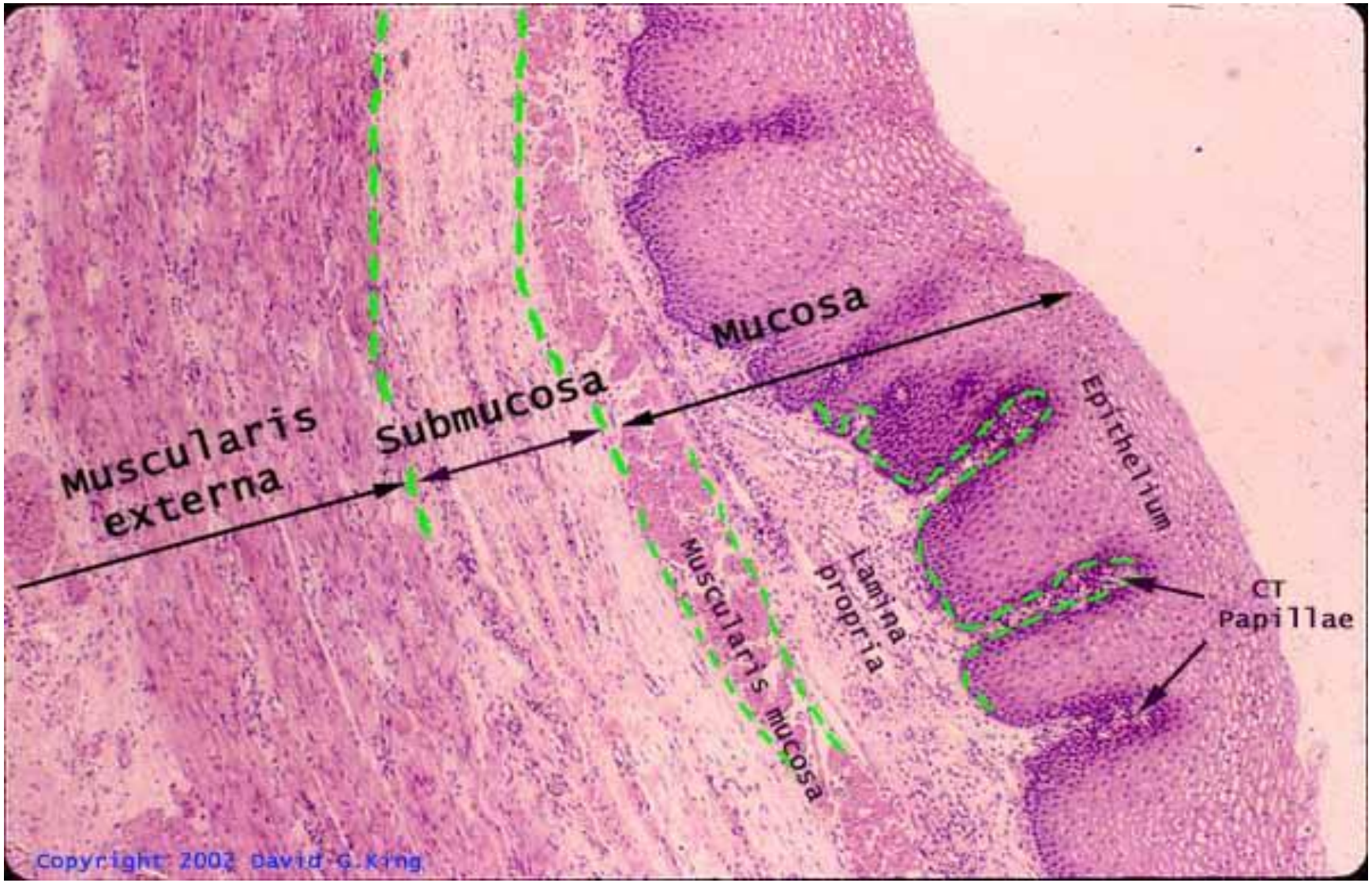
No serosa

Spincters:

- UES (at the level of the sixth cervical vertebra)
- LES (1 to 2 cm above the hiatus)



Esophageal histology



- **Signs &symptoms of esophageal disorder:**

1-dysphagia: is difficulty in swallowing is encountered both with deranged esophageal motor function and with diseases that narrow or obstruct the lumen.

2-Heart burn: is a retrosternal burning pain due to regurgitation of gastric contents into the lower esophagus

3-Pain and hematemesis are sometimes evoked by esophageal disease, particularly by those lesions associated with inflammation or ulceration of the esophageal mucosa, esophageal varices.

- **Congenital anomalies:**
- The most important are esophageal atresia and tracheoesophageal fistula.
- ***Atresia:***

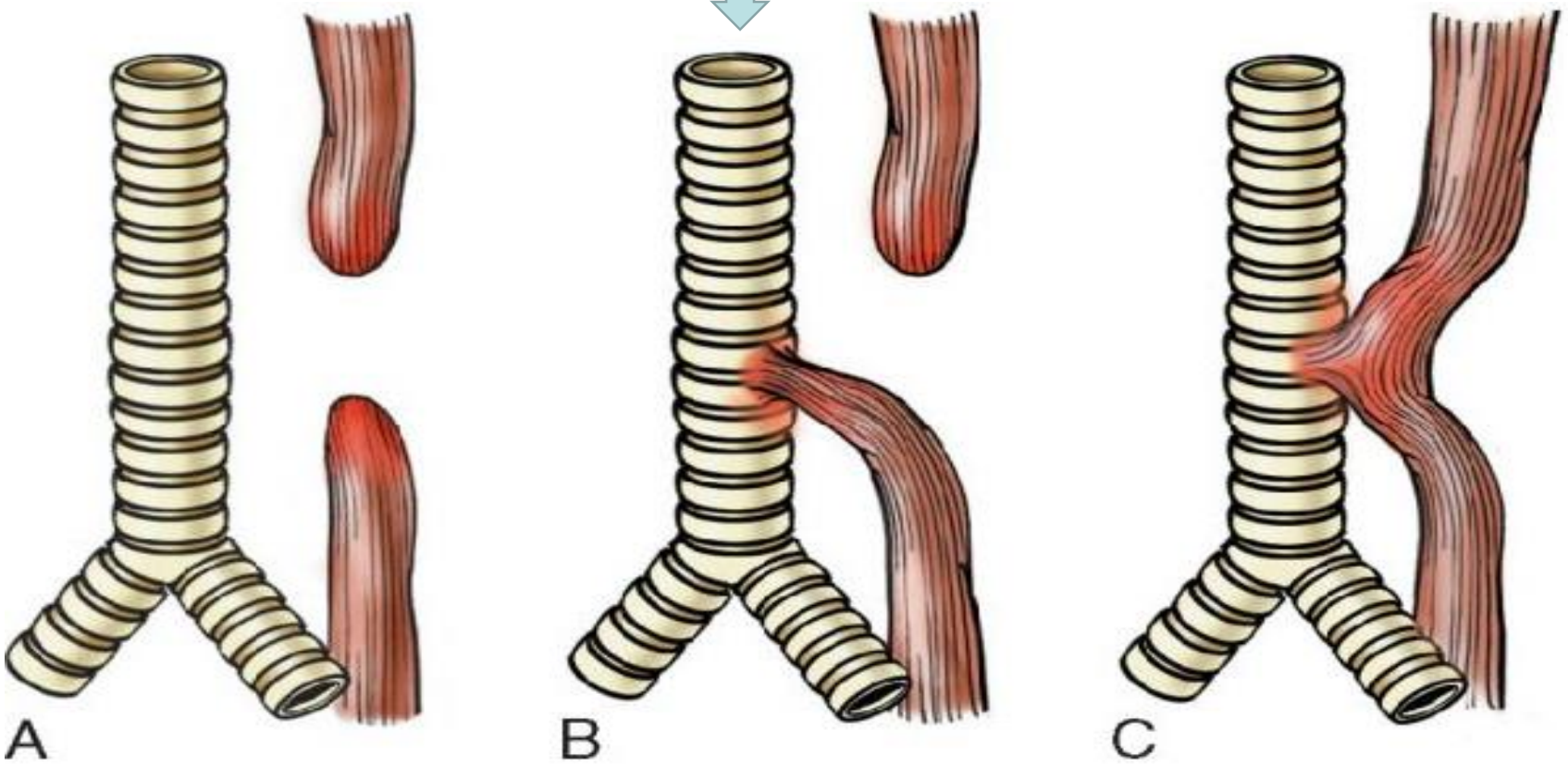
Is congenital absence of the normal canal (blind end of the canal).

Usually atresia is associated with fistula (tracheoesophageal)

- ***Tracheo-esophageal fistula:***

It will lead to the passage of food from the esophagus to the trachea leading to aspiration pneumonia which leads to suffocation from food.

Most common 82%



Esophageal atresia and Tracheoesophageal fistula.

A, Blind upper and lower esophageal segments.

B, Blind upper segment with fistula between lower segment and trachea.

C, Fistula between patent esophagus and trachea. Type B is the most common.

Acquired lesions:

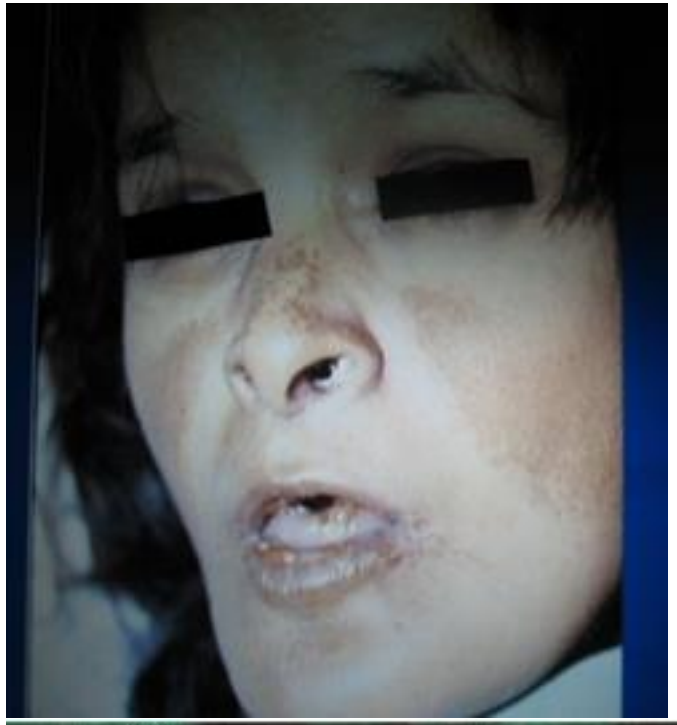
Webs, rings, and stenosis

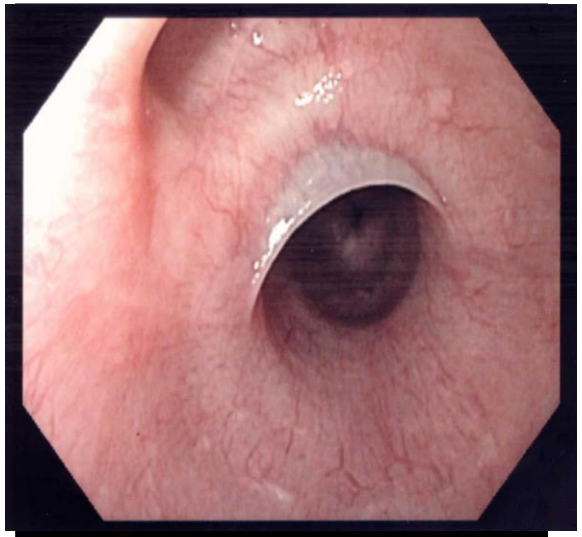
Mucosal webs are thin, eccentric mucosal membrane projects into the esophageal lumen. These are most common in the upper esophagus.

Upper esophageal web + iron deficiency anemia + atrophic glossitis(beef like tongue) + angular stomatitis are called **plummer Vinson syndrome.** This carry a risk of esophageal carcinoma .

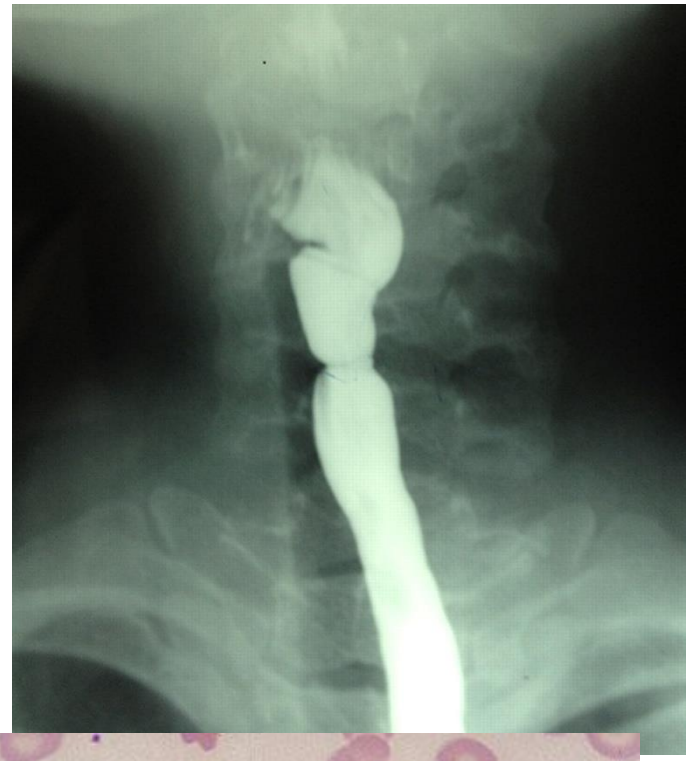
Case - 45yo female with weakness and difficulty in swallowing

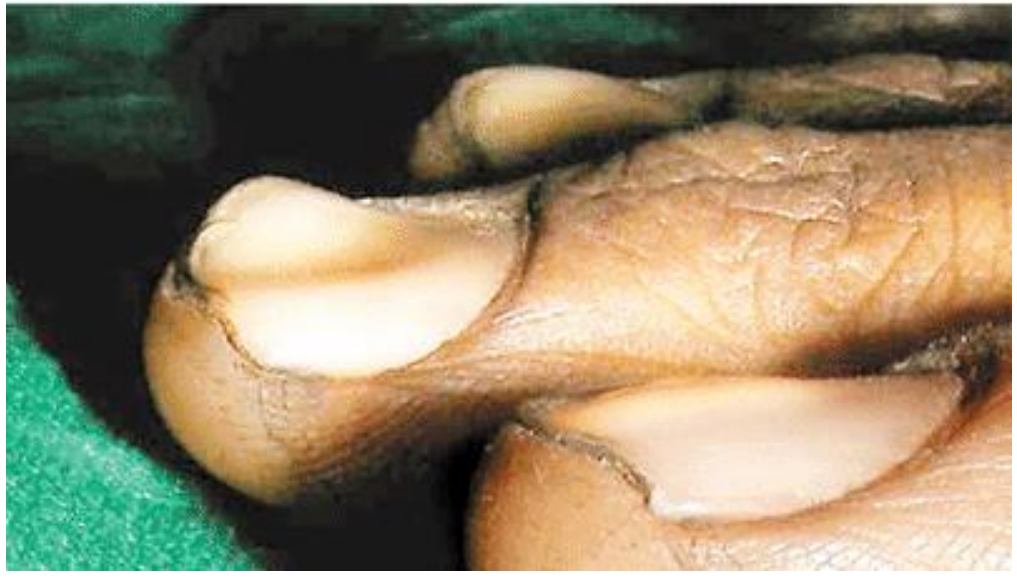
What are your findings? Diagnosis?

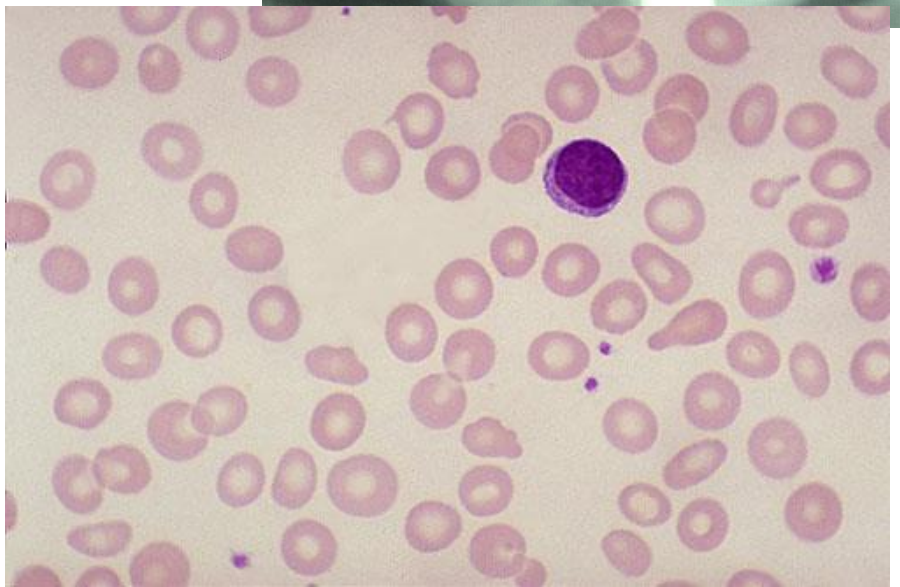




Endoscopic view of esophagus







Findings

Angular stomatitis

Atrophic glositis

Esophageal web (Endoscopy, Barium swallow)

Koilonychia

Microcytic hypochromic anemia

What is your diagnosis?

Synonyms:

Plummer-Vinson Syndrome(USA)

Patterson-Kelly Syndrome(UK)

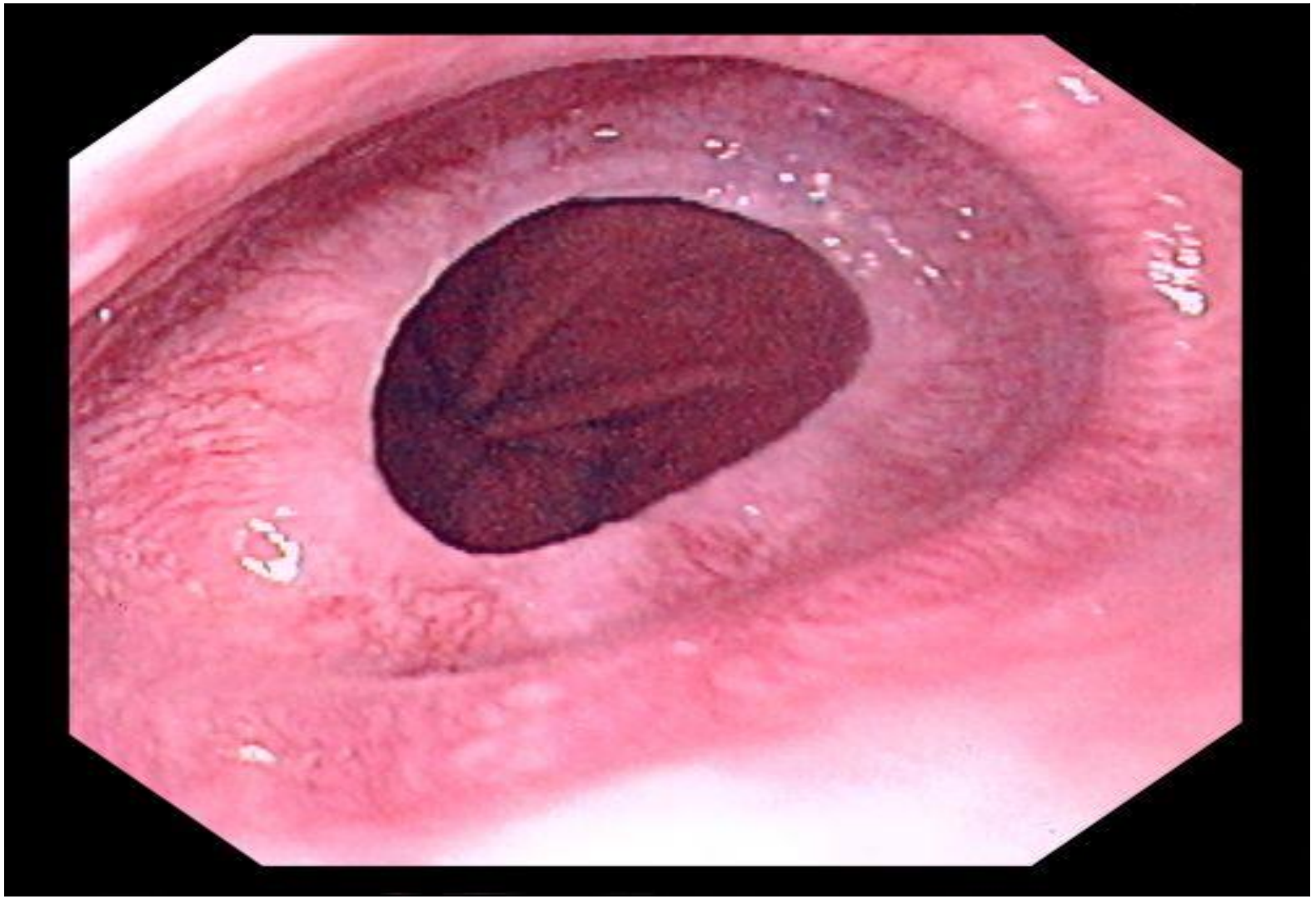
Sideropenic dysphagia

Esophageal rings unlike webs are concentric plates of tissue protruding into the lumen of the distal esophagus.

Esophageal webs and rings are encountered most frequently in women over age 40.

Episodic dysphagia is the main symptom.

Esophageal ring



Rings or Webs

- Ring
  - Circumferential, muscle or mucosa, at distal esophagus
  - Schatzki's ring
  - Eosinophilic Esophagitis (>15 eosinophils/hpf in mucosa)
- Web
  - Part of lumen, mucosal, proximal esophagus
  - Plummer Vinson

Stenosis consists of fibrous thickening of the esophageal wall.

Although it may be congenital, it is more frequently the result of severe esophageal injury with inflammatory scarring, as from gastroesophageal reflux disease (GERD), radiation, scleroderma and caustic injury.

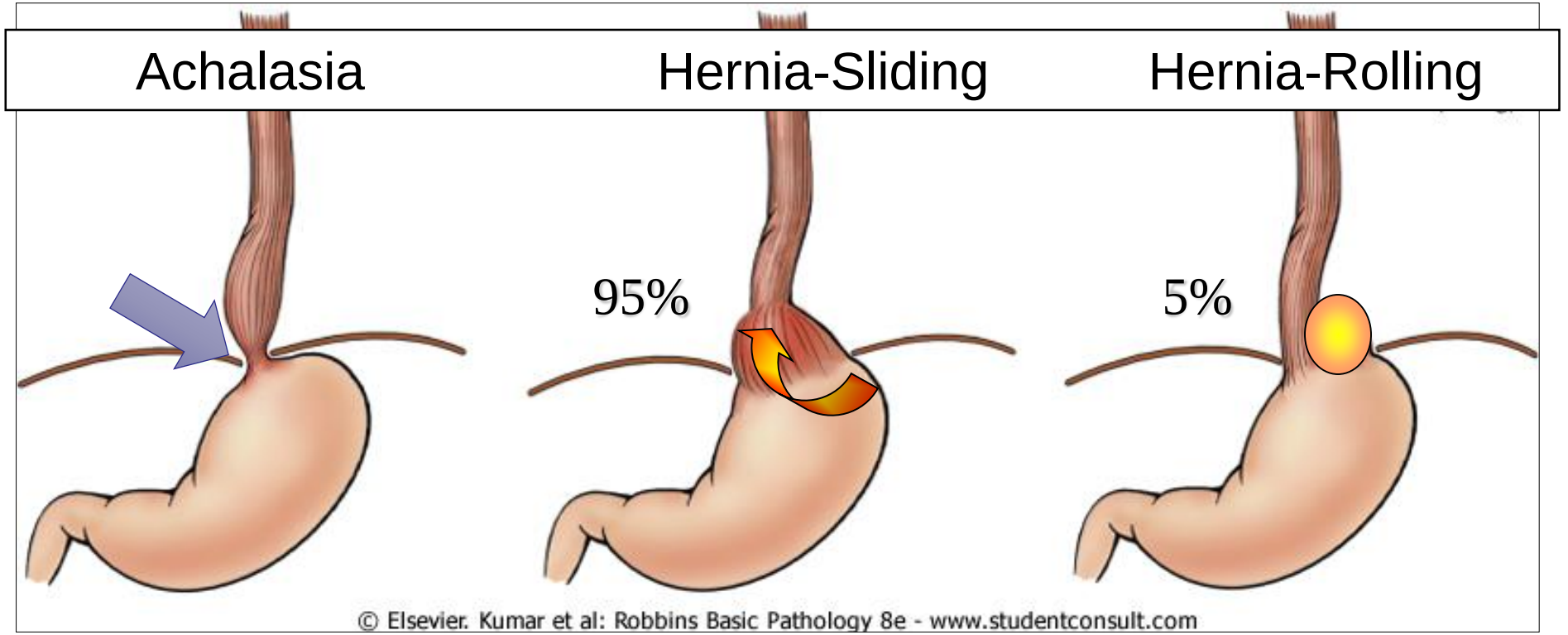
Stenosis usually manifests as progressive dysphagia,at first to solid foods but eventually to fluids as well.

Lesions associated with motor dysfunction:

Coordinated motor activity is important for proper function of the esophagus. The major entities that are caused by motor dysfunction of the esophagus are

1. *Achalasia*
2. *Hiatal hernia*
3. *Diverticula*
4. *Mallory-Weiss tear*

Oesophagus motility Disorders:



Hernia: 30% incidence over 50years. (mostly asymptomatic)
Achalasia: Lack of relaxation of lower sphincter.

Lesions with motor dysfunction:

- ***1-Achalasia(Greek:does not relax):*** Failure of the lower esophageal sphincter to relax in response to swallowing leading to accumulation of food in the more proximal esophagus causing its dilatation & inflammation (esophgitis)

It is characterized by three major abnormalities:

1. Aperistalsis (failure of peristalsis)
2. Increased resting tone of the LES
3. Icomplete relaxation of the LES in response to swallowing

The wall of the esophagus may be of normal thickness, thicker than normal owing to hypertrophy of the muscular wall, or markedly thinned by dilation (when dilatation overruns hypertrophy).

The mucosa just above the LES may show inflammation and ulceration.

Young adults are usually affected and present with progressive dysphagia

- **Signs and symptoms:**

The symptoms become manifested in young adulthood

a- Progressive dyphagia.

b- Regurgitation of undigested food.

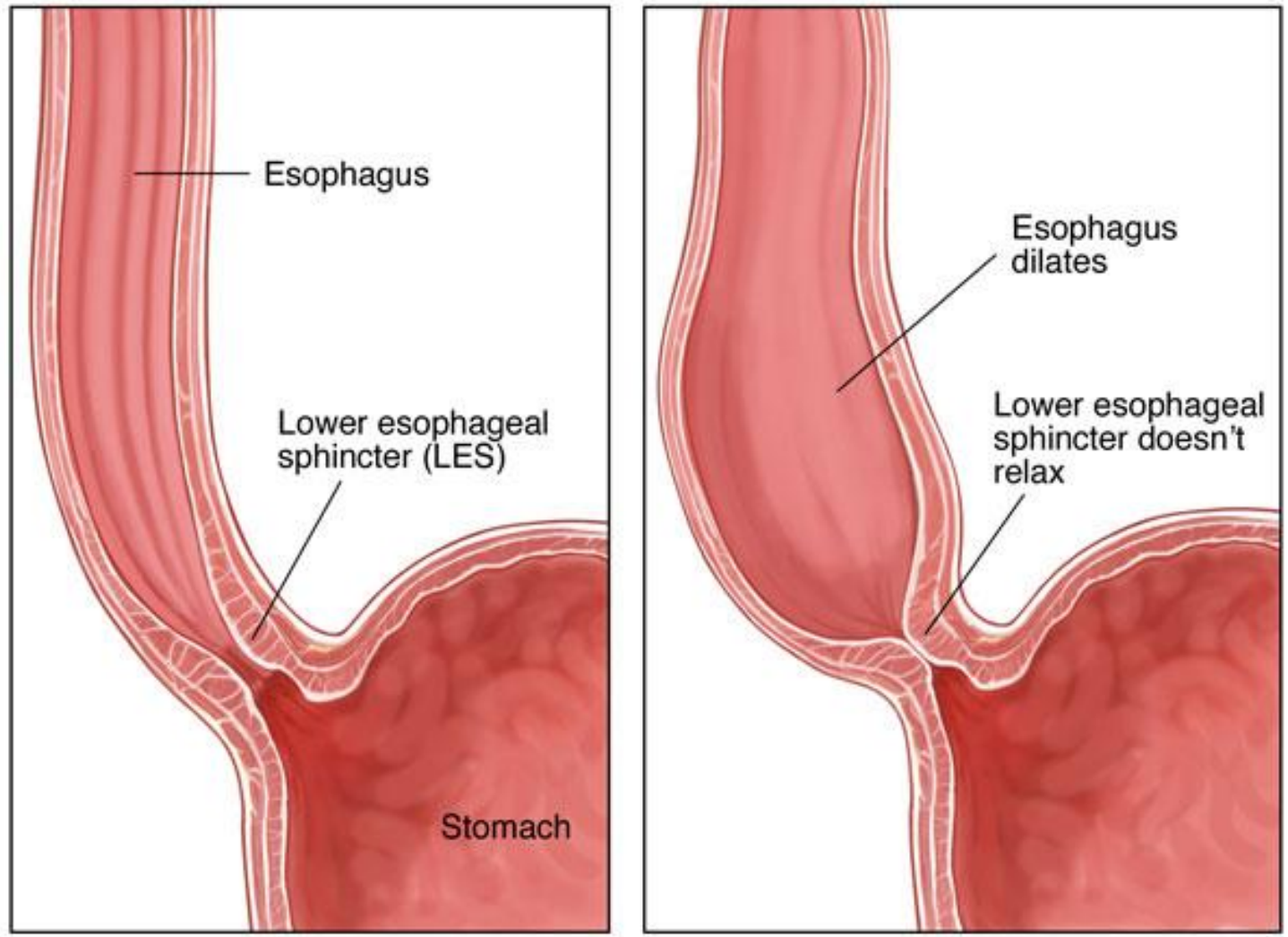
Complications:

* Squamous cell carcinoma develops in 5% of cases.
* Aspiration of undigested food which may cause pneumonia
* Candida esophagitis
* Lower esophageal diverticuli

- **Etiology:**

1- Primary: Uncertain causes (degenerative process involving the neural innervations of the esophagus).

2- Secondary: Chagas disease (in which there is destruction of the myenteric plexus of the esophagus)



Esophageal Achalasia