2ND YEAR / GIT BLOCK

MED TEAMS 43

2012

# PATHOLOGY TEANS

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## **1 PLEOMORPHIC ADENOMA**



## i.e., MIXED TUMOR

The classic place for ANY visible parotid swelling or tumor to present, <u>between the tip of the ear and the tip (angle) of</u> <u>the mandible.</u>



Mixed tumors are generally <u>benign</u>, have BOTH connective tissue (i.e., usually cartilagenous) components as well as glandular components, hence the name pleomorphic or mixed, they generally look and feel like little round soft cartilage balls.



Mixed tumor of the parotid gland contains

- epithelial cells forming ducts (\*)
- myoepithelial cells ( #)
- chondromyxoid stroma (@).

Pleomorphic adenoma of the salivary gland: Section shows an incomplete fibrous capsule separating the tumour from normal salivary gland:

- Tumour shows mixed cellular components like epithelial, myoepithelial, chondriod and myxoid elements.
- Epithelial areas shows small ducts, acini and strands or sheets of cells.
- Myxoid areas are formed of loose myxomatous tissue
- chondriod areas consists of pale blue matrix.

\*treatment : total execution

\* what is the biological behavior of the tumor ? benign

## **2 - BARRETT'S ESOPHAGUS**





Inflammatory Cells

Eosinophils ,Neutrophils & Lymphocytes

- Basal zone hyperplasia
- Lamina Propria papillae elongated and congested

### BARRETT'S ESOPHAGUS

## "Barrett's esophagus"

- gastric-type mucosa above the gastroesophageal junction.
- Note the columnar epithelium to the left and the squamous epithelium at the right.
- Typical Barrett's mucosa shows intestinal metaplasia with chronic inflammation in lamina propria (esophagitis)
- (note the goblet cells in the columnar mucosa).



- INTESTINALIZED metaplastic mucosa is <u>AT RISK</u> for glandular dysplasia.
- Searching for dysplasia when BARRETT's is present is of utmost importance
- MOST/ALL <u>adenocarcinomas arising</u> ( as a complication ) in the esophagus arise from previously existing BARRETT's

## **3- Carcinoma of the esophagus**







Squamous cell carcinoma is the most common type of esophageal cancers

• dysplasia→in-situ→infiltration (cancer)

Would you call this squamous "dysplasia"? Answer: YES

Would your fear it would develop into squamous cell carcinoma? Answer: YES

Does it always? Answer: NO

Does it usually? Answer: With time, YES

Squamous cell carcinoma of the esophagus in a patient who presented with progressive dysphasia. It is easy to see how this mass produced this symptom. The oval structure adjacent to the esophagus represents metatastic squamous cell carcinoma within a lymph node.





# **4-"PEPTIC" ULCERS**





A 1 cm acute gastric ulcer is shown here in the upper fundus. The ulcer is shallow and sharply demarcated, with surrounding hyperemia. It is probably benign. However, all gastric ulcers should be biopsied to rule out a malignancy.

The Base of a Nonperforated Chronic Peptic Ulcer

Necrosis (N) Inflammation (I) Granulation tissue (G) Scar (S) (Top - luminal surface, Bottom - muscular wall)









Helicobacter pylori, gastric biopsy, silver stain on left, giemsa stain on right.

# "PEPTIC" ULCERS Ulcer: discontinuation of epithelium

- *"PEPTIC" implies acid cause/aggravation*
- MUC→SUBMUC→MUSCULARIS→SEROSA
- Chronic, solitary (usually), adults
- 80% caused by H. pylori
- 100% caused by H. pylori in duodenum
  NSAIDS

#### Gastritis

- IN the lamina propria we see neutrophils, Lymphocytes, lymphoid follicles
- Some REGENERATIVE CHANGES can also be noted like
- METAPLASIA, intestinal
- ATROPHY, mucosal hypoplasia, "thinning" or
- DYS-PLASIA
  - Gastritis is often accompanied by infection with Helicobacter pylori.
  - This small curved to spiral rod-shaped bacterium is found in the surface epithelial mucus of most patients with active gastritis.
  - The rods are seen here with a silver stain as well as methylene blue stain

# 5-Gastric adenocarcinoma





- The LINITIS PLASTICA is the most SPECTACULAR, and most FEARED, of all gastric adenocarcinomas. It grows DIFFUSELY through all layers of the stomach, greatly thickening its wall, and giving the stomach a classic LEATHER BOTTLE appearance.
- Diffuse thickening of the gastric wall which is infiltrated by a white firm tumor . It has a horrible **prognosis with early metastases to lymph nodes** .



- Signet ring malignant glandular cells with evidence of intracellular mucin secretion and mitoses
- Signet ring cells are POORLY differentiated adenocarcinoma cells, and are OFTEN seen with linitis plastica. Those large "holes" in the cytoplasm represents intracellular mucin which push the nucleus to the periphery giving the cell signet <u>ring appearance</u>.



Gastric adenocarcinoma, intestinal pattern



Photomicrograph of a poorly differential intestinal type adenocarcinoma of the stomach.

# 6- celiac disease



Normal villous length to crypt length is 3/1



Celiac disease: complete lack of villi.



a- Low-power view of fully developed (celiac disease )sprue-type changes. Note the elongated crypts with complete lack of villi.

b- High-power view showing damaged surface epithelium with large numbers of intraepithelial lymphocytes.

- Increase number of Intraepithelial lymphocytes
- infiltration of lymphocytes & plasma cells
- villous atrophy and crypt hyperplasia .

Serology test show: Antigliadin antibodies in the sera of the patient.

Later the patient can develop T cell lymphoma as a complication

Yong Patient come with history of : failure to thrive

## **7-CARCINOID OF SMALL INTESTINE**



Section of small intestine shows surface ulceration and an infiltrating tumour mass in mucosa and submucosa



Tumour consists of alveolar groups and clumps of small uniform polygonal cells having centrally placed round nuclei and abundant granular cytoplasm.

## 8-Crohn disease





- Here the inflammation has produced large, irregularly shaped to rake-like ulcers that are separated from each other by mucosa that appears close to normal.
- Though any portion of the gastrointestinal tract may be involved with Crohn's disease, the small intestine and the terminal ileum in particular is most likely to be involved



Microscopically, Crohn's disease is characterized by transmural inflammation. Here, inflammatory cells (the bluish infiltrates) extend from mucosa through submucosa and muscularis and appear as nodular infiltrates on the serosal surface with pale granulomatous centers.

At high magnification the granulomatous nature of the inflammation of Crohn's disease is demonstrated here with **epithelioid cells, giant cells, and many lymphocytes.** 

Special stains for organisms are negative

Crohn's disease of the intestine: Section of large bowel shows alternating normal and ulcerating mucosa:

- All layers of intestinal wall show transmural chronic inflammatory cell infiltrate, lymphoid aggregates and mild fibrosis.
- Subserosa contains few epithelioid granulomas.

\*to rule out TB the pathologist stain the sample with AFB

## **9- ULCERATIVE COLITIS**









Cryptitis, crypt abscess and depletion of goblet cells

The entire colon is abnormal, and the usual transverse rugal folds have been almost completely effaced. We see Dilated colon, ulceration, mucosal congestion and hemorrhage.

At higher magnification, the pseudopolyps can be seen clearly as raised red islands of inflamed mucosa.

Between the pseudopolyps is only remaining muscularis

Microscopically, the inflammation of ulcerative colitis is confined primarily to the mucosa.

## Ulcerative colitis:

Section of large bowel wall show a few relatively superficial ulcers lined by acute inflammatory exudate.

- The mucosa adjacent to the ulcers contains several crypt abscesses and there is evidence of goblet cells depletion in many glands.
- No granulomas or glandular dysplasia are noted.

Ulcerative colitis can give rise to

toxic megacolon, glandular dysplasia and adenocarcinoma.

# 10- adenoma of the colon





Organ: Colon

Dx: adenoma



Colonic polyp.



# 11 -Familial polyposis coli

\*

The mucosal surface of the colon is essentially a carpet of small adenomatous polyps. Even though they are small now, there is a 100% risk over time for development of adenocarcinoma, so a total colectomy is done, generally before age 20



## Multiple colonic polyps.

It is caused by **mutations** of the adenomatous polyposis coli , or APC gene .



## ADENOMATOUS POLYP (TUBULAR)

- Crowded dysplastic glands with chronic inflammation
- TUBULAR adenoma, note how all the epithelial (glandular) cells look the same.



# ADENOMATOUS POLYP (VILLOUS)

- Villous adenomas behave more aggressively than tubular adenomas.
- They have a HIGHER rate of **developing into frank** adenocarcinomas than the "tubular" patterns

# 12- adenocarcinoma



This cancer is more exophytic in its growth pattern. Thus, one of the complications of a carcinoma is obstruction (usually partial).



The encircling mass of firm adenocarcinoma in this colon at the left is typical for adenocarcinomas arising in the descending colon.

A change in stool or bowel habits can be created by the mass effect.



## Adenocarcinoma of the large intestine: Section of large intestine shows:

A tumour mass at one end, and a normal mucosa on the other side:

- **Tumour** consists of crowded irregular malignant acini separated by thin fibrovascular stroma.
- The acini are lined by one or several layers of neoplastic cells with papillary projection showing pleomorphism, hyperchromatism and few mitoses.
- **Muscle coat** is invaded by neoplastc glands.

## 13- Chronic hepatitis













Chronic hepatitis: Section from this liver biopsy show:

- Moderate chronic inflammatory cells infiltration consisting of <u>lymphocytes and histiocytes</u> in both portal tracts and liver parenchyma.
- Piecemeal necrosis (necrosis around the portal tried), hepatocytes swelling and "spotty" hepatocytes necrosis are also noticed.



## 14- macronudular cirrhosis (HBV)



- Multiple nodules of variable sizes with fibrosis.
- The complications of cirrhosis can be :

Portal hypertension, Hepatic failure, hepatocellular carcinoma.



irregular nodules separated by portal-toportal fibrous bands



- **Cirrhosis** >> Very low power microscopic view of the liver.
- The parenchyma shows darker tan nodules of varying sizes.
- These nodules are composed of hepatocytes.
- The paler areas in between are collagen.



Cirrhosis, trichrome stain for fibrosis

Cirrhosis of the liver Section of liver show:

- Loss of lobular architecture and formation of regenerative nodules of variable size and shape, surrounded by fibrous tissue.
- Each **nodules consists** of liver cells without any arrangement and with no central vein.
- Large number of proliferated bile ducts and chronic inflammatory cells are present in fibrous tissue.

## **15** -Hepatocellular carcinoma





This example of well-differentiated HCC shows a trabecular pattern with intervening sinusoids.



Hepatocellular carcinoma

Anaplastic tumor giant cells can be seen in poorly differentiated HCC

The key to the identification of HCC is its resemblance to hepatocytes, the presence of more than 2-3 cell-thick hepatocellular plates/cords, nuclear atypia, and absence of portal tracts. Note the hepatic plates are separated from each other by sinusoids

Hepatocellular carcinoma: Section show tumour consisting of:

- Thick cords, trabeculate and nests of malignant liver cells separated by sinusoidal spaces.
- Malignant liver cells <u>are pleomorphic</u>, <u>binucleated or forming giant cells</u> with <u>hyperchromatic nuclei</u>.
- Mitoses are numerous.
- Areas of haemorrhage and necrosis are present.

## 16- chronic cholecystitis





Chronic cholecystitis: Section of gallbladder wall shows:

- Irregular mucosal folds and foci of ulceration in mucosa.
- Wall is penetrated by mucosal glands which are present in muscle coat (Rokitansky-Aschoff sinuses).
- All layers show chronic inflammatory cells infiltration and fibrosis.



The surface epithelium has lost its normal delicate papillary appearance(green arrow) with an increase in fibrous tissue and mild chronic inflammation in the lamina propria

- Rokitansky-Aschoff sinuses are seen in the muscularis (black arrow)
- The degree of chronic inflammation is quite variable and as in this case **surprisingly mild**

## 17- acute pancreatitis





In severe acute pancreatitis, **black areas** of hemorrhage are present within the pancreas as well as chalky, **yellow-white areas** of fat necrosis. Pancreatic parenchyma is soft and graywhite due to necrosis



Does this look like a partly digested piece of meat? It is.



This image of severe acute pancreatitis shows an area of acute inflammation with necrosis. Within the necrotic area is a **blood vessel** <u>showing fibrinoid</u> <u>necrosis</u> of the vessel wall. Damage such as this leads to severe, hemorrhagic, acute pancreatitis

## **18 - Chronic Pancreatitis**



Hard, shrunken, dilated ducts, visible calcified concretions



Unfortunately dense fibrosis is a feature BOTH of chronic pancreatitis as well as adenocarcinoma.



Loss of acini and ductal tissue with relative sparing of islets, irregularly distributed bland peri-ductal fibrosis.

Chronic inflammation around lobules and ducts.

What is every pathologist's nightmare?

Ans: Getting a small needle biopsy of sclerosing pancreatitis and calling it it cancer, getting the "Whipple" specimen the next day, and realizing you were WRONG! The patient has now undergone an operation which has a 10% mortality rate, for no reason, and the malpractice attorneys at at your door like jackals.

# **19 - Pancreatic Adenocarcinoma**





Ill defined pale and firm pancreatic mass

Malignant glands or acini surrounded by desmoplastic fibrous stroma.

the risk factors are

Smoking ,chronic pancreatitis and diabetes mellitus



Malignant glands surrounded by desmoplasia

