# Small and Large Intestinal pathology, part 3

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# INFLAMMATORY INTESTINAL DISEASE

- Sigmoid Diverticulitis
- Chronic Inflammatory bowel diseases (CIBD)

Crohn disease

Ulcerative colitis

### Sigmoid Diverticulitis

- Acquired.
- Elevated intraluminal pressure in the sigmoid colon
- Exaggerated peristaltic contractions,
- Low fiber diet and constipation.
- Pseudodiverticulae
- Outpouchings of colonic mucosa and submucosa

#### **MORPHOLOGY**

- ► Flasklike outpouchings
- Mostly in sigmoid colon.
- Thin wall (atrophic mucosa, compressed submucosa)
- Attenuated or absent muscularis.
- Obstruction leads to diverticulitis.
- Risk of perforation.
- Recurrent diverticulitis leads to strictures.



## Diverticulosis Diverticulitis Inflammation and complications of Diverticula Mere presence of Diverticula Intestines Intestines Bleeding



#### Clinical Features

- Mostly asymptomatic.
- Intermittent lower abdominal pain
- Constipation or diarrhea.

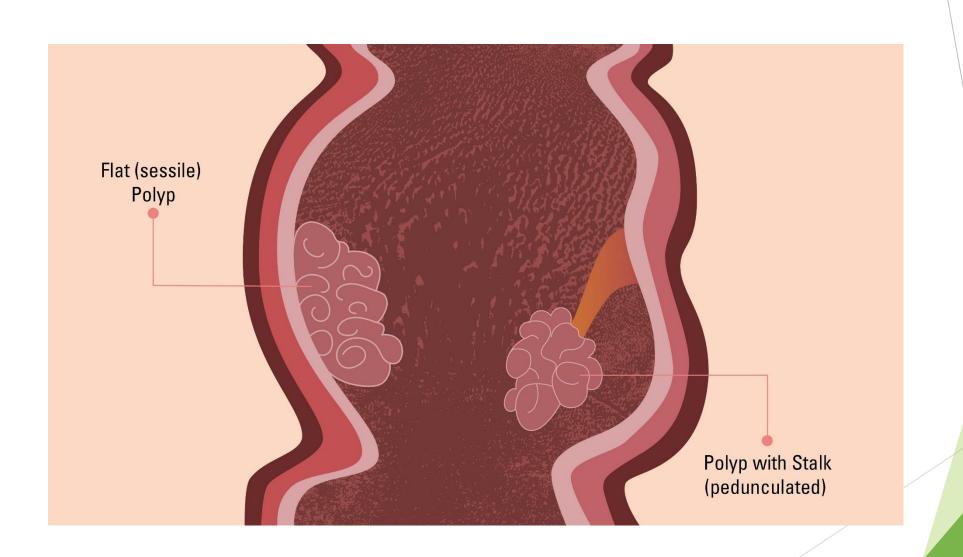
- ► Tx
- High fiber diet.
- Antibiotics in diverticulitis.
- Surgery.

#### Diseases of the intestines

- Intestinal obstruction
- Vascular disorders
- Malabsorptive diseases and infections
- Inflammatory bowel disease.
- ► Polyps and neoplastic diseases

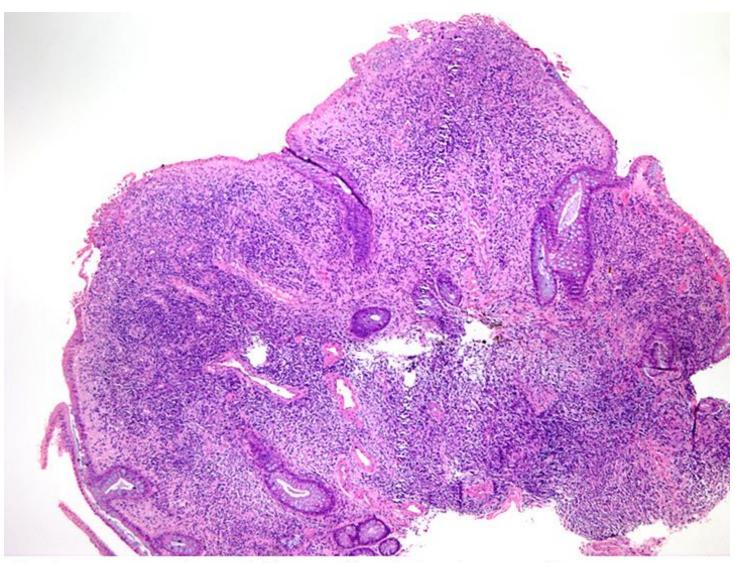
# COLONIC POLYPS AND NEOPLASTIC DISEASE

- Colon is most common site for polyps
- **Sessile polyp:** no stalk
- Pedunculated polyp: stalk.
- Neoplastic polyps: adenoma.
- Non neoplastic polyps: inflammatory, hamartomatous, or hyperplastic



## Inflammatory Polyps

- Solitary rectal ulcer syndrome.
- Recurrent abrasion and ulceration of the overlying rectal mucosa.
- Chronic cycles of injury and healing give a polypoid mass of inflamed and reactive mucosal tissue.



4x: low power, dense inflammation in lamina propria

### Hamartomatous Polyps

- Sporadic or syndromatic.
- Disorganized, tumor-like growth composed of mature cell types normally present at that site.

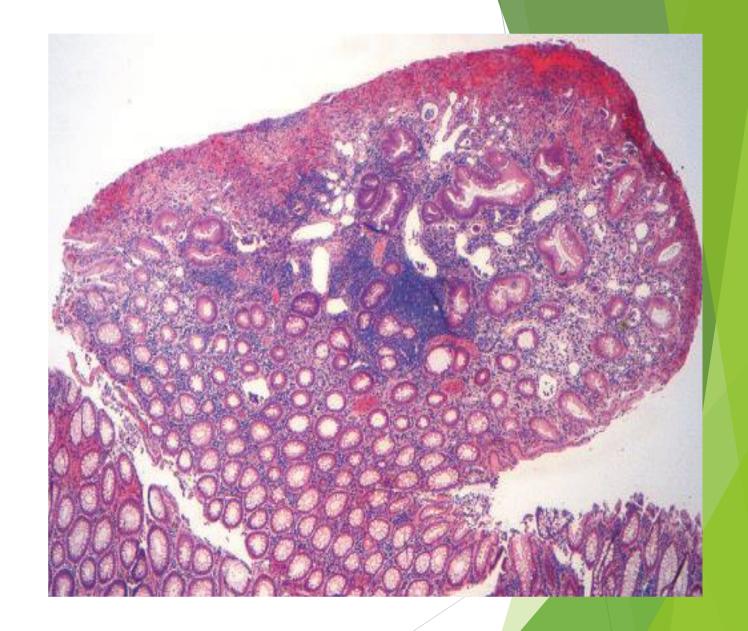
- Juvenile Polyps
- Peutz-Jeghers Syndrome

## Juvenile Polyps

- Most common hamartomatous polyp
- Sporadic are solitary.
- Children younger than 5 years of age
- Rectum.
- Syndromic are multiple.
- ▶ 3 to as many as 100. Mean age 5 years
- Autosomal dominant syndrome of juvenile polyposis
- Transforming growth factor-β (TGF-β) mutation.
- Increased risk for colonic adenocarcinoma.

## Juvenile Polyps

- Pedunculated
- Reddish lesions
- Cystic spaces on cut sections
- Dilated glands filled with mucin and inflammatory debris.
- Granulation tissue on surface.

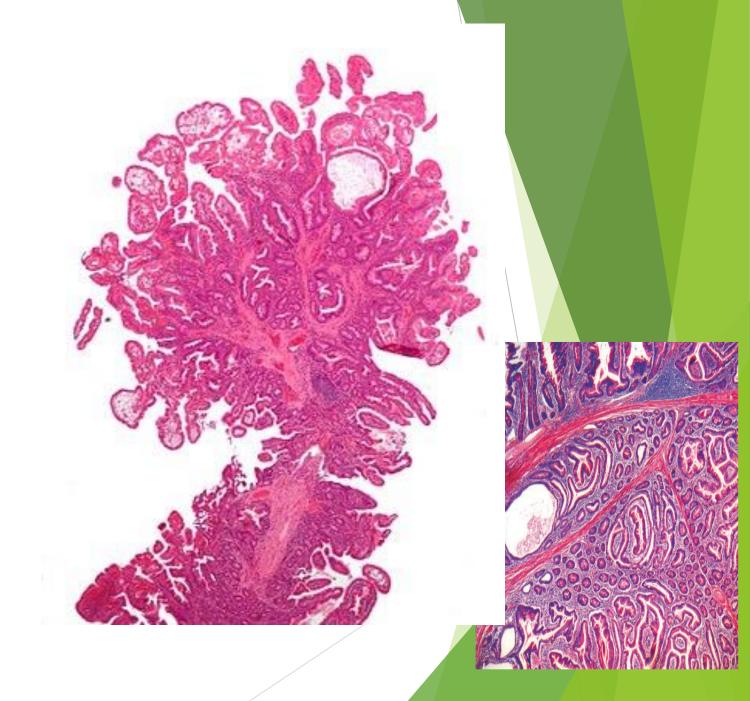


## Peutz-Jeghers Syndrome

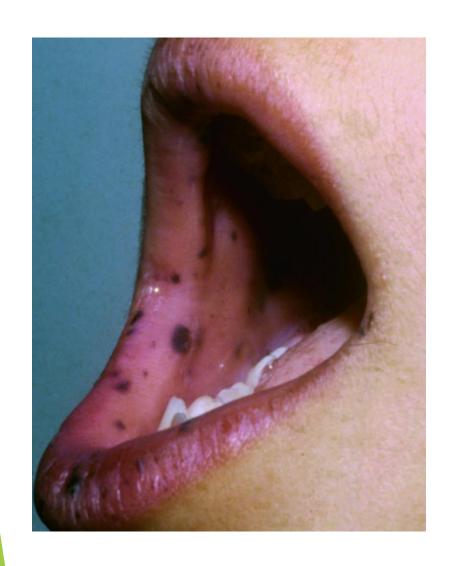
- Autosomal dominant, rare
- ► Mean age: 10-15 years.
- Multiple gastrointestinal hamartomatous polyps
- Most common in the small intestine.
- Mucocutaneous hyperpigmentation
- Increased risk for several malignancies: colon, pancreas, breast, lung, ovaries, uterus, and testes,
- ► *LKB1/STK11* gene mutation.

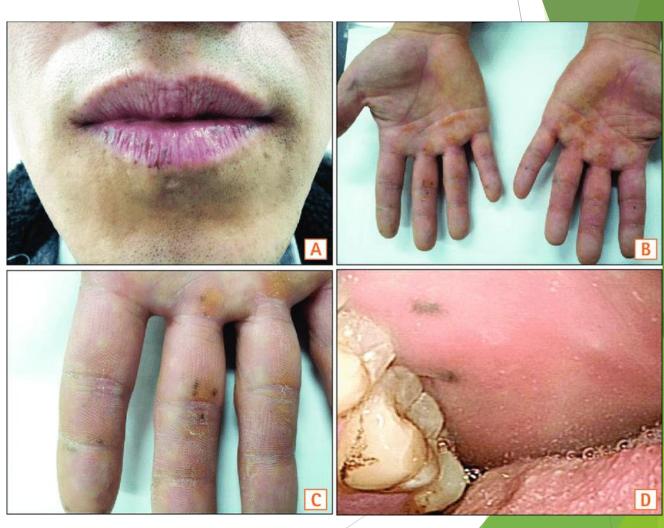
## Peutz-Jeghers polyp

- Large.
- Arborizing network of connective tissue, smooth muscle, lamina propria
- Glands lined by normal-appearing intestinal epithelium
- Christmas tree pattern.



# Mucocutaneous pigmentation



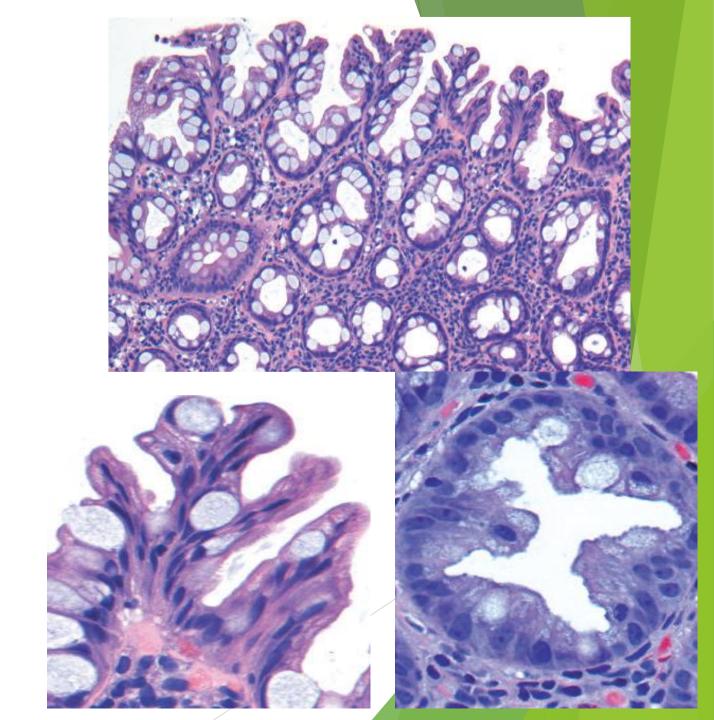


## Hyperplastic Polyps

- Common
- ► 5<sup>th</sup>-6<sup>th</sup> decade.
- Decreased epithelial turnover and delayed shedding of surface epithelium >>> pileup of goblet cells & epithelial overcrowding
- No malignant potential

## Hyperplastic polyp

- Left colon
- Rectosigmoid.
- ► Small < 5 mm
- Multiple
- Crowding of goblet & absorptive cells.



#### **Adenomas**

- Most common and clinically important
- Increase with age.
- Definition: presence of epithelial dysplasia (low or high).
- Precursor for majority of colorectal adenocarcinomas
- Most adenomas DO NOT progress to carcinoma.
- ► USA: screening colonoscopy starts at 50 yrs.
- Earlier screening with family history.
- Western diets and lifestyles increase risk.

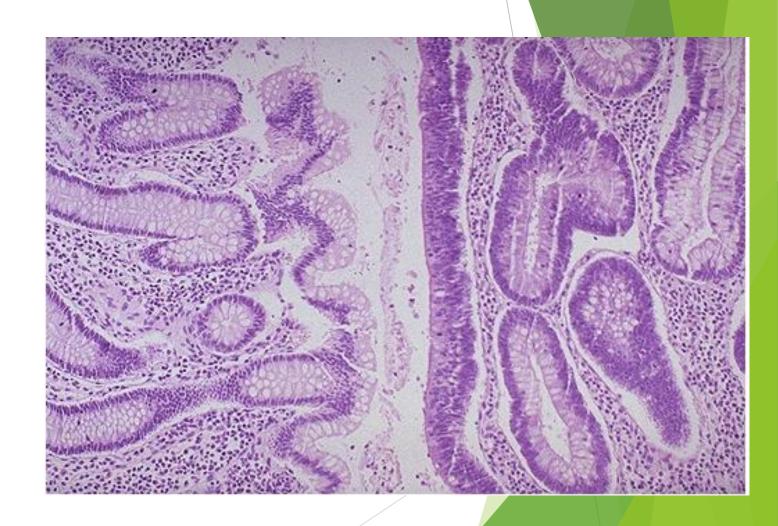
## Pedunculated or sessile



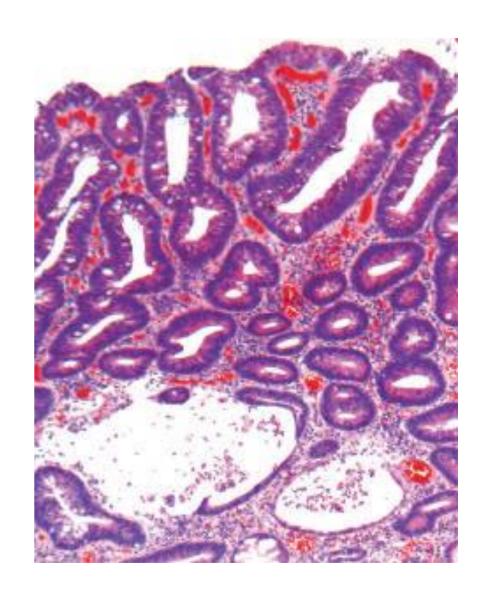


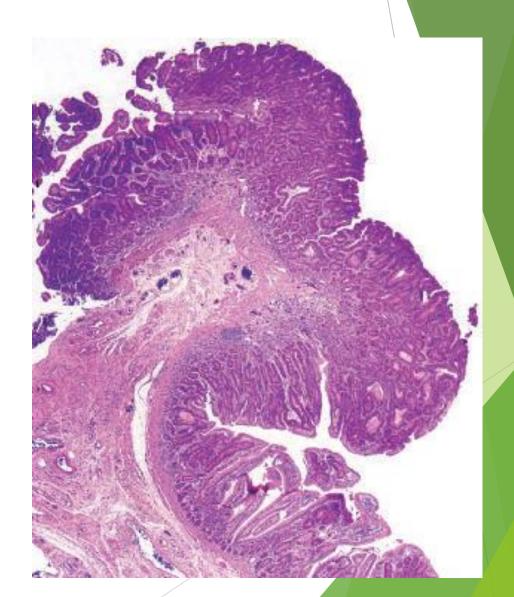
#### Colon adenoma

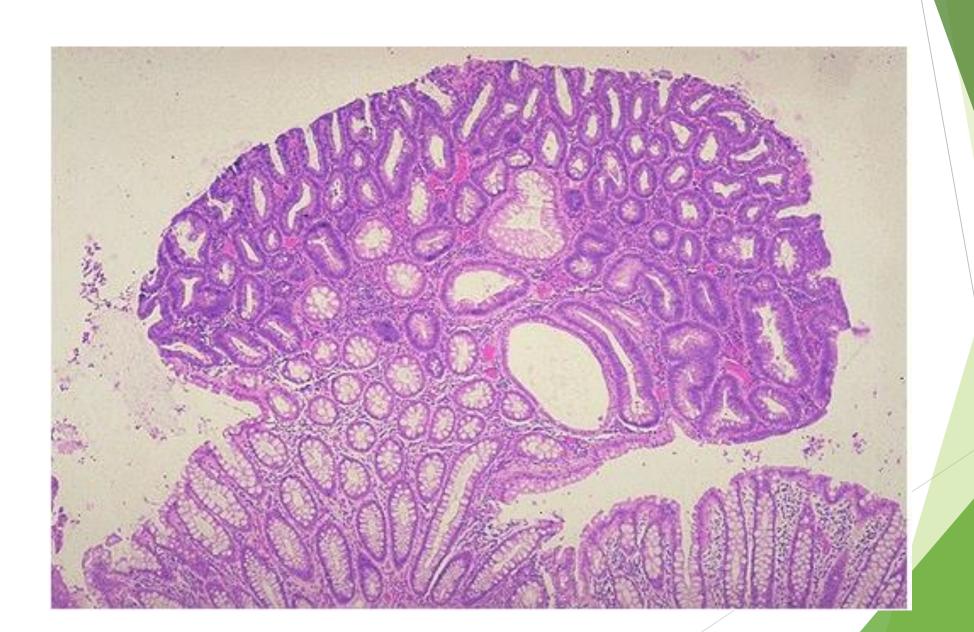
- Hallmark: epithelial dysplasia
- Dysplasia: nuclear hyperchromasia, elongation, stratification, high N/C ratio.
- Size: most important correlate with risk for malignancy
- High-grade dysplasia is the second factor



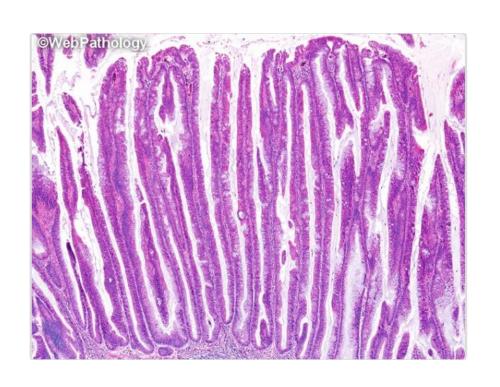
## Tubular adenoma







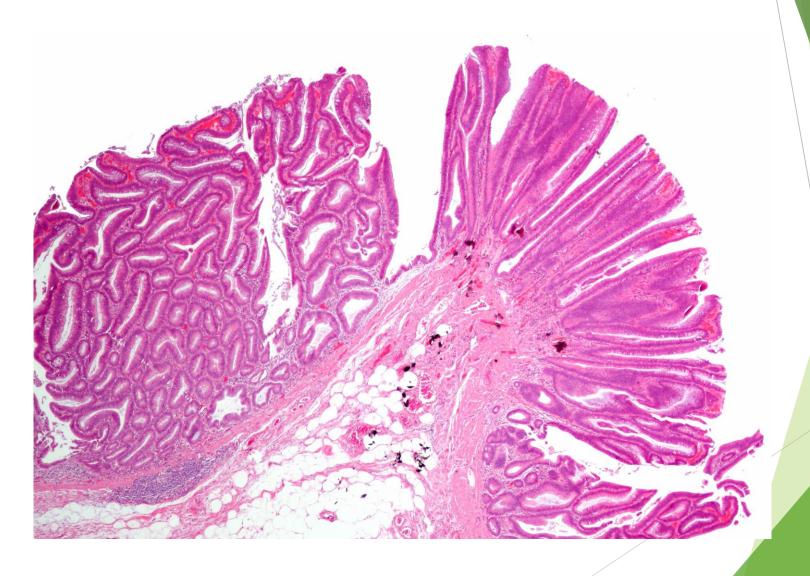
#### Villous adenoma.



- Long slender villi.
- More frequent invasive foci

- Architecture:
- Tubular.
- ► Tubulovillous.
- Villous.

## Tubulovillous adenoma



### Familial Syndromes

- Syndromes associated with colonic polyps and increased rates of colon cancer
- Genetic basis.

- ► Familial Adenomatous Polyposis (FAP)
- Hereditary Nonpolyposis Colorectal Cancer (HNPCC)

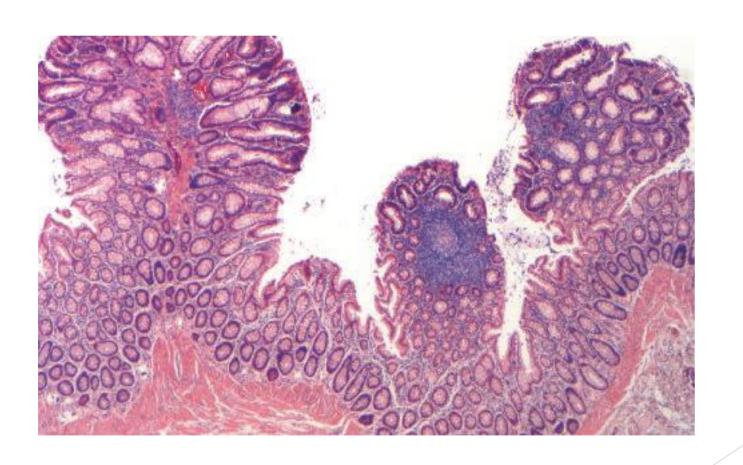
### Familial adenomatous polyposis FAP

- Autosomal dominant.
- Numerous colorectal adenomas: teenage years.
- Mutation in APC gene.
- ▶ At least 100 polyps are necessary for a diagnosis of classic FAP.
- Morphologically similar to sporadic adenomas
- ▶ 100% of patients develop colorectal carcinoma, IF UNTREATED, often before age of 30.
- Standard therapy: prophylactic colectomy before 20 Year of age.
- Risk for extraintestinal manifestations,

► Variants of FAP: Gardner syndrome and Turcot syndrome.

- ► **Gardner syndrome**: intestinal polyps + osteomas (mandible, skull, and long bones); epidermal cysts; desmoid and thyroid tumors; and dental abnormalities.
- Turcot syndrome: intestinal adenomas and CNS tumors (medulloblastomas >> glioblastomas )





# Hereditary Nonpolyposis Colorectal Cancer: HNPCC, Lynch syndrome

- Clustering of tumors: Colorectum, endometrium, stomach, ovary, ureters, brain, small bowel, hepatobiliary tract, and skin
- Colon cancer at younger age than sporadic cancers
- Right colon with excessive mucin production .
- Adenomas are present, BUT POLYPOSIS IS NOT.
- Inherited germ line mutations in DNA mismatch repair genes.
- Accumulation of mutations in microsatellite DNA (short repeating sequences)
- Resulting in microsatellite instability
- Majority of cases involve either MSH2 or MLH1.

# Cecal polyps in HNPCC.

