

Small and Large Intestinal pathology, part 3

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Diseases of the intestines

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

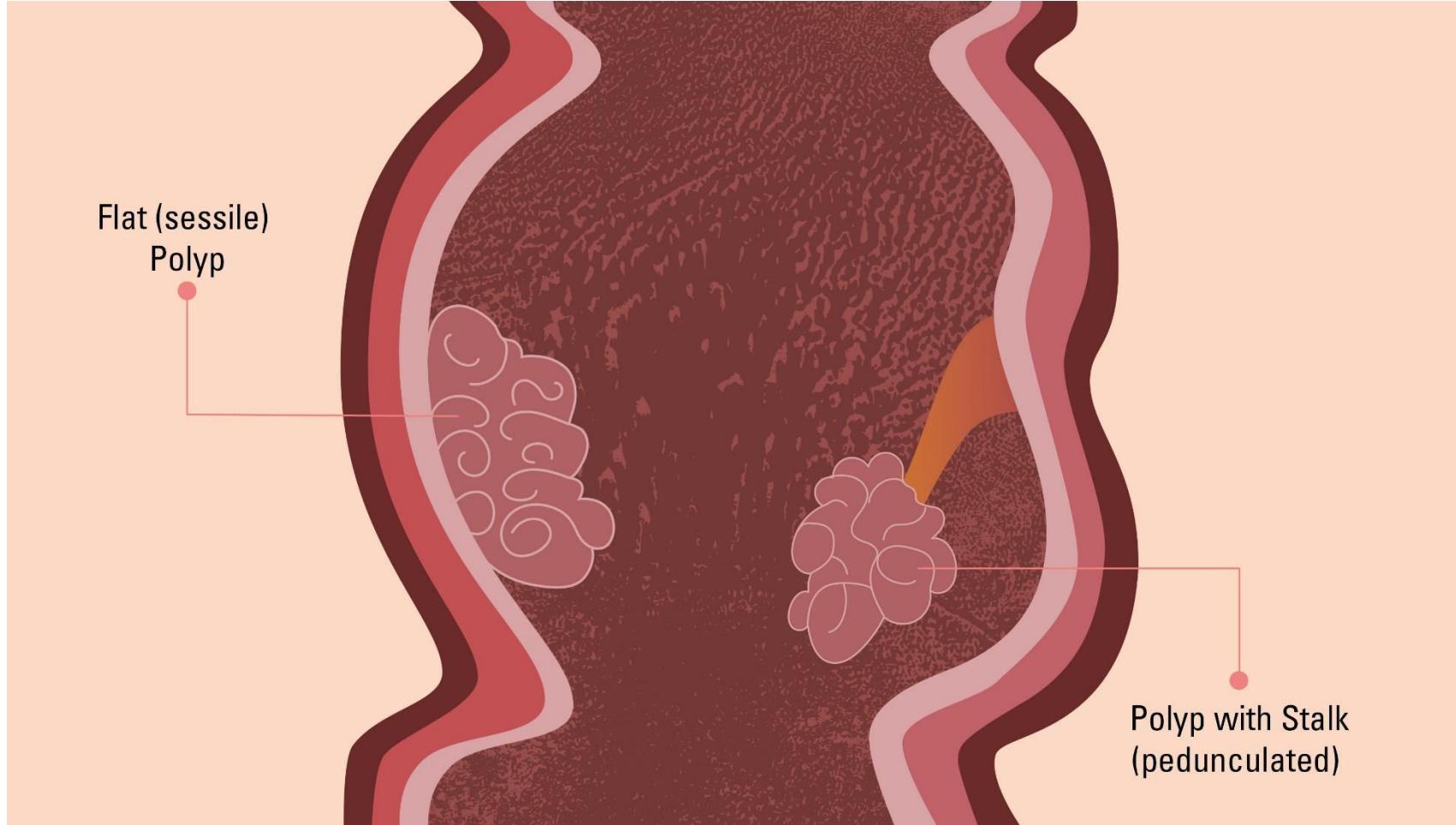


COLONIC POLYPS AND NEOPLASTIC DISEASE

- ▶ Colon is most common site for polyps
- ▶ *Sessile polyp*: no stalk
- ▶ *Pedunculated polyp*: stalk.

- ▶ *Neoplastic polyps*: adenoma.
- ▶ *Non neoplastic polyps*: inflammatory, hamartomatous, or hyperplastic

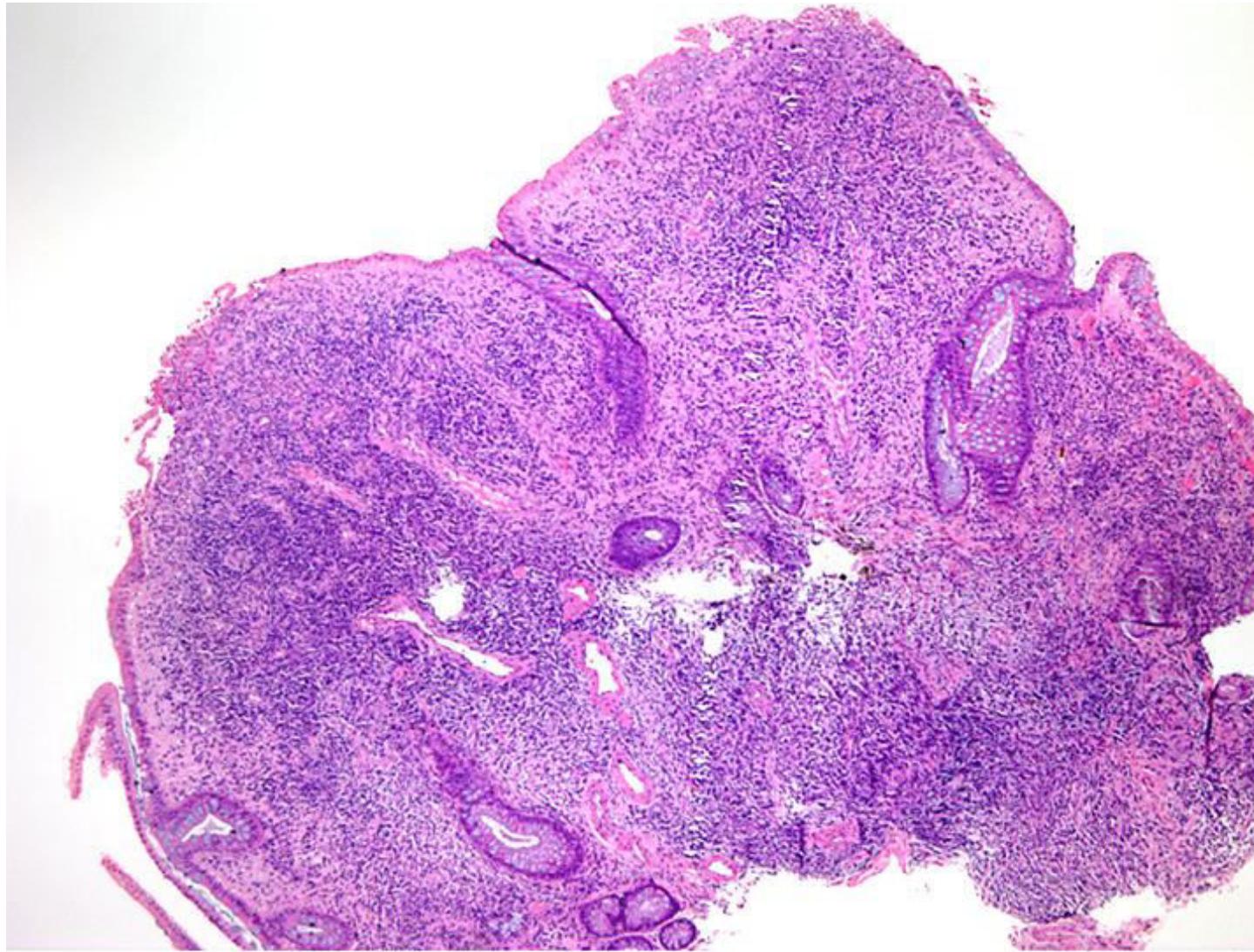




Inflammatory Polyps

- ▶ *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.
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- ▶ Juvenile Polyps
 - ▶ Peutz-Jeghers Syndrome



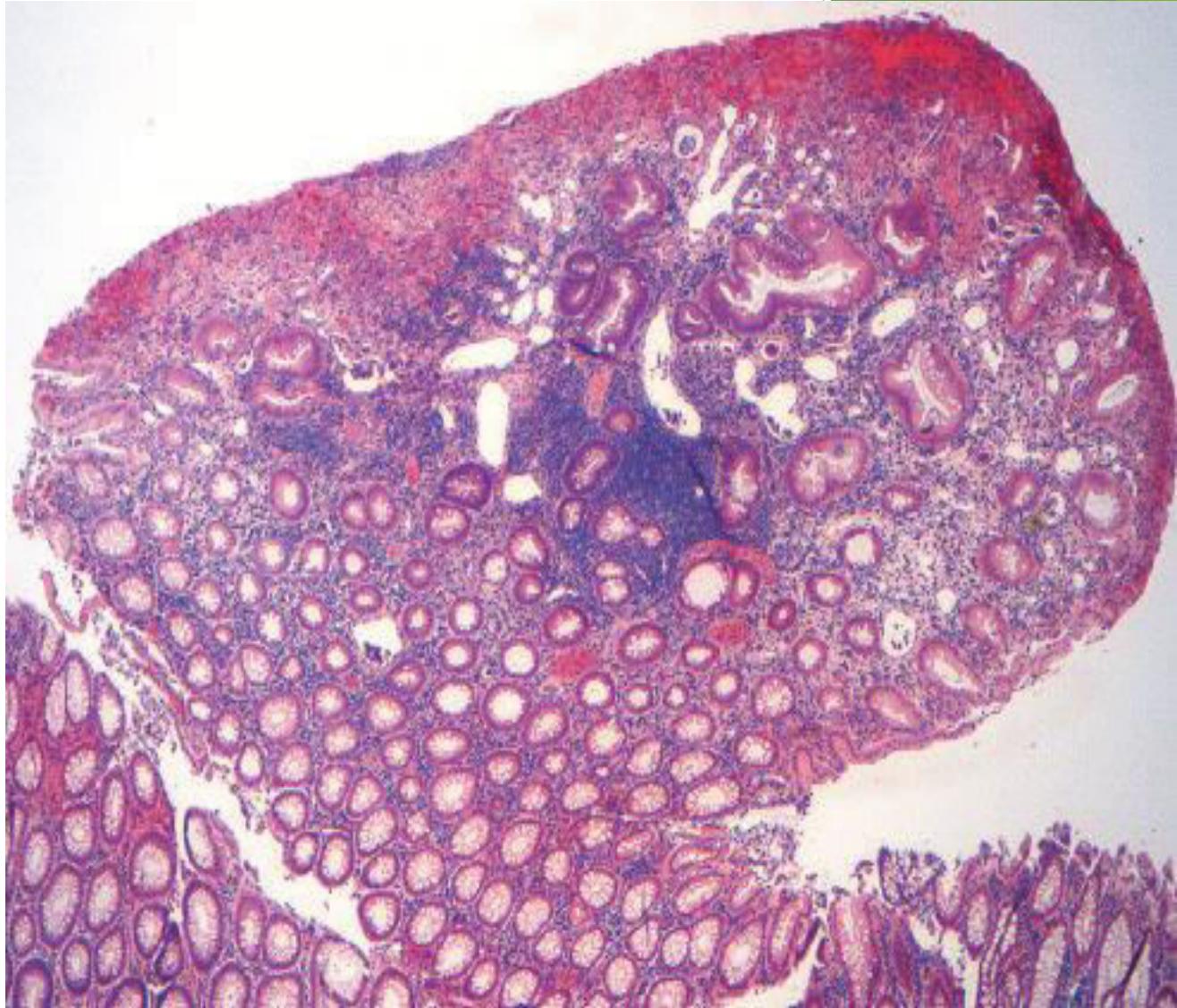
Juvenile Polyps

- ▶ 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

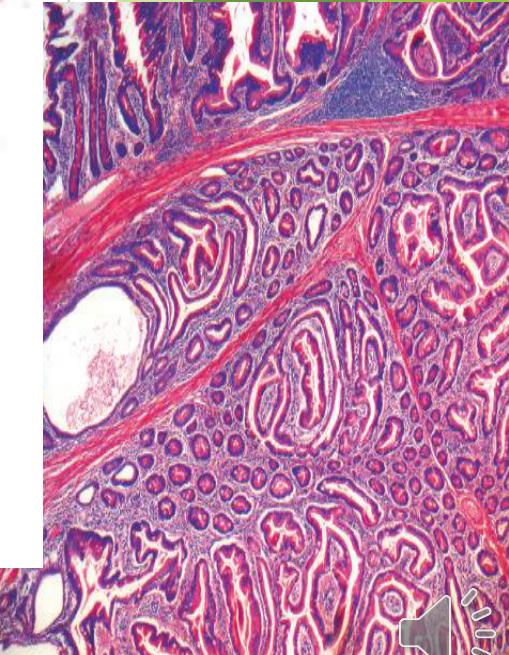
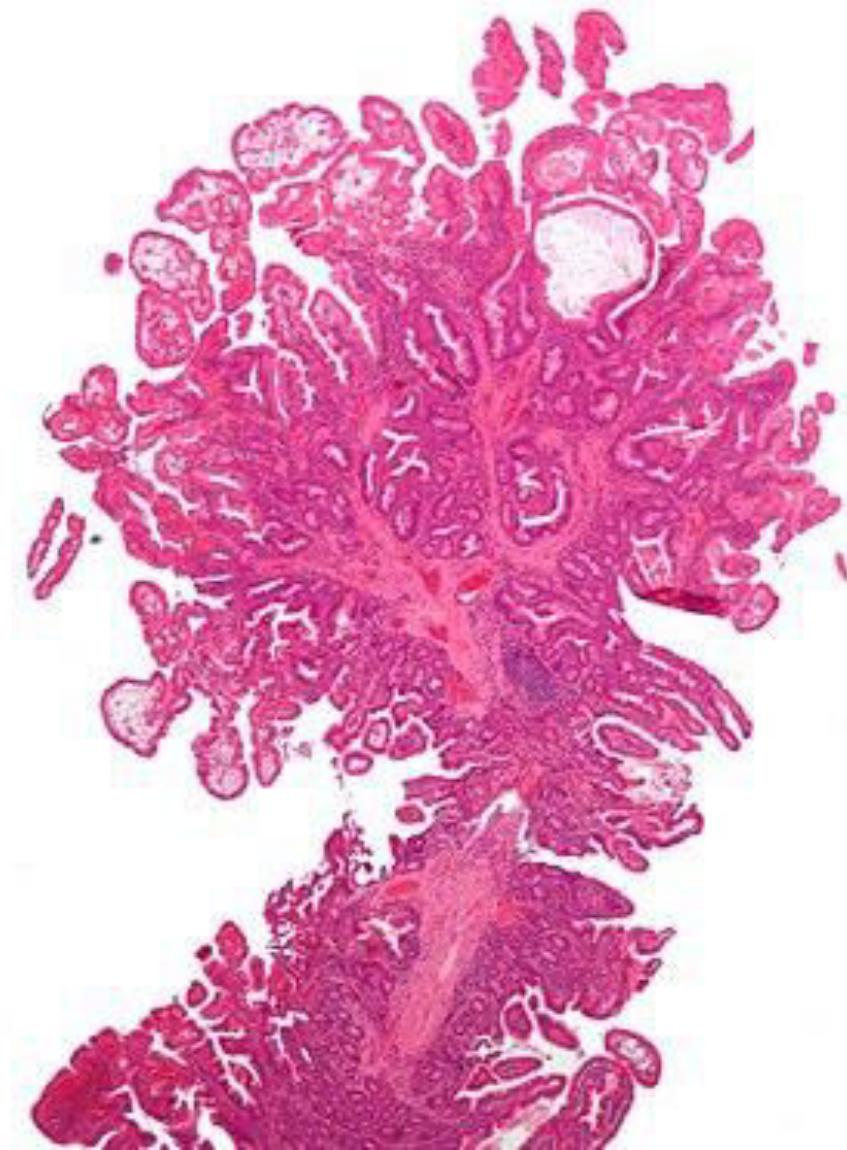
- ▶ Autosomal dominant, rare
- ▶ Mean age: 10-15 years.
- ▶ Multiple gastrointestinal hamartomatous polyps
- ▶ Most common in the small intestine.
- ▶ Mucocutaneous hyperpigmentation
- ▶ Increased risk for several malignancies: colon, pancreas, breast, lung, ovaries, uterus, and testes,

- ▶ *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



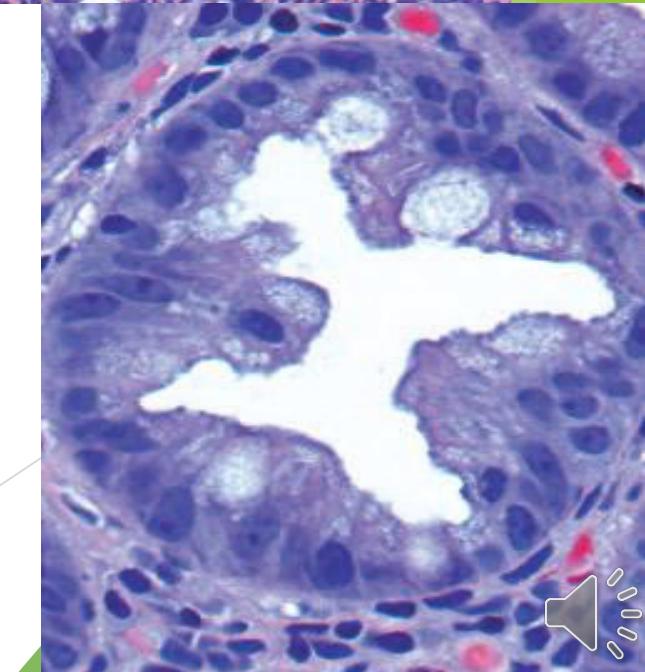
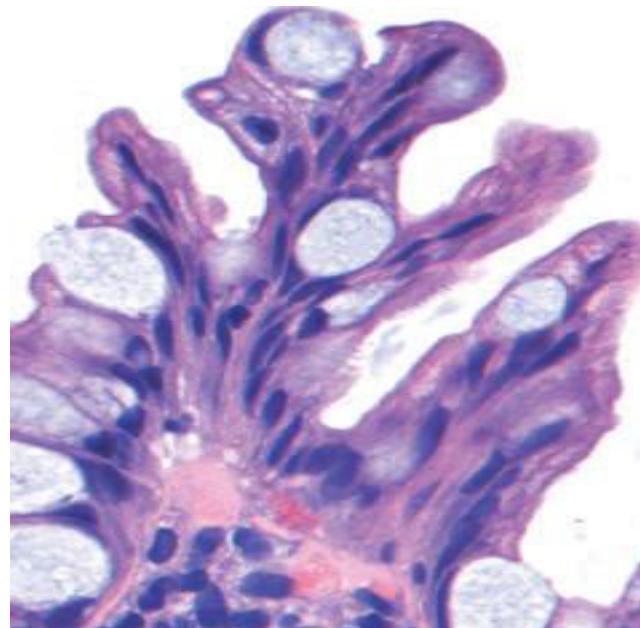
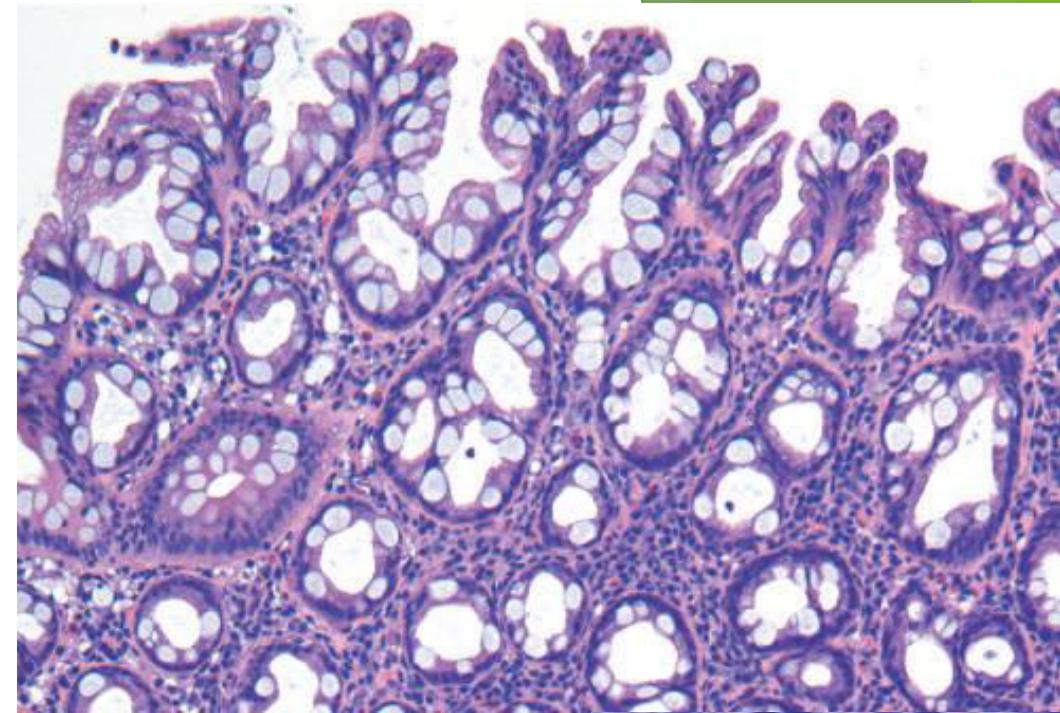
Hyperplastic Polyps

- ▶ Common
- ▶ 5th-6th 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.**

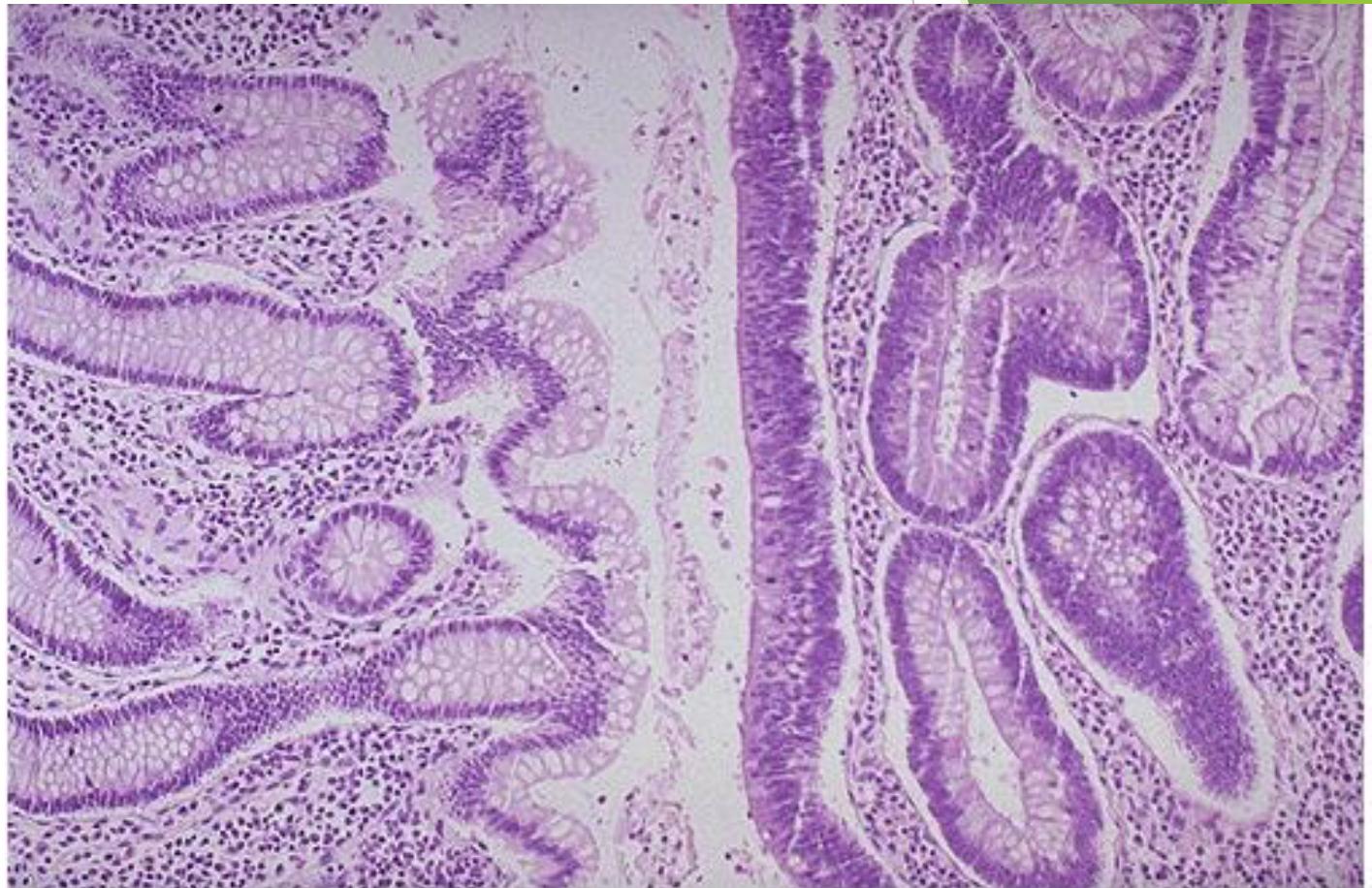


Pedunculated or sessile

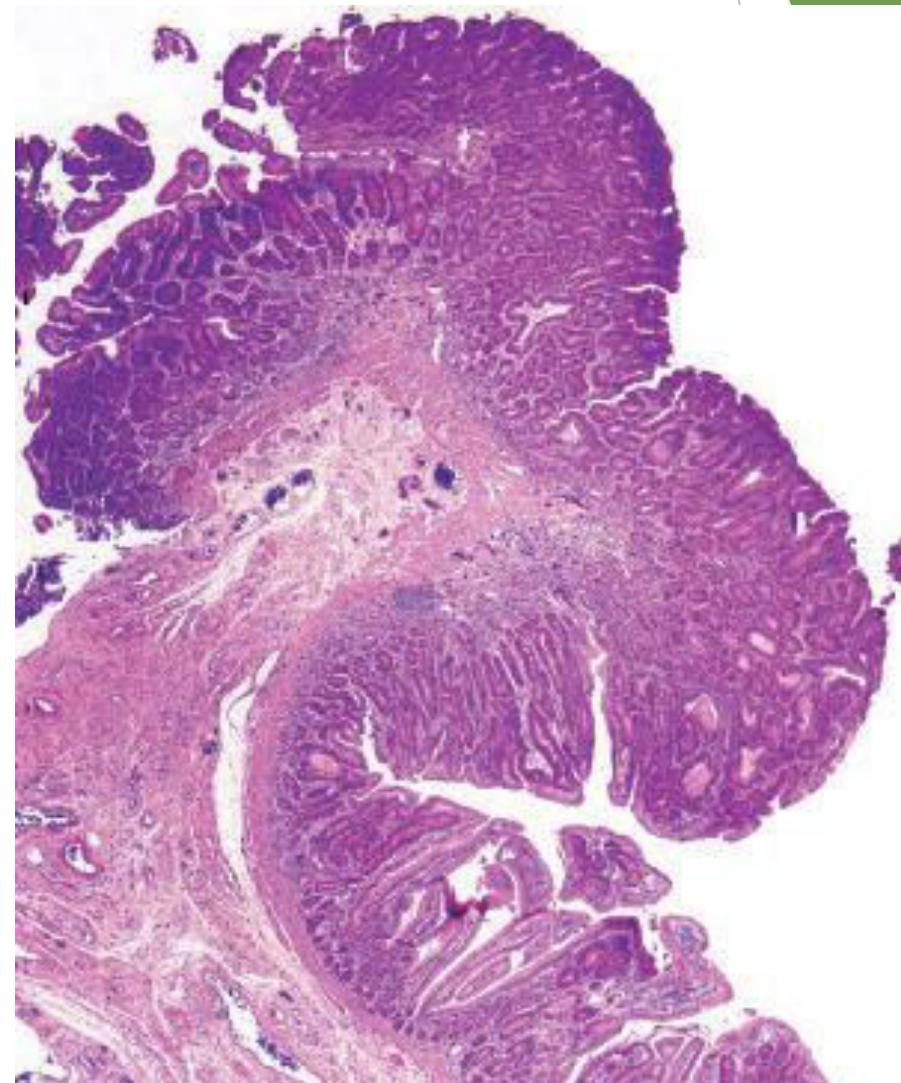
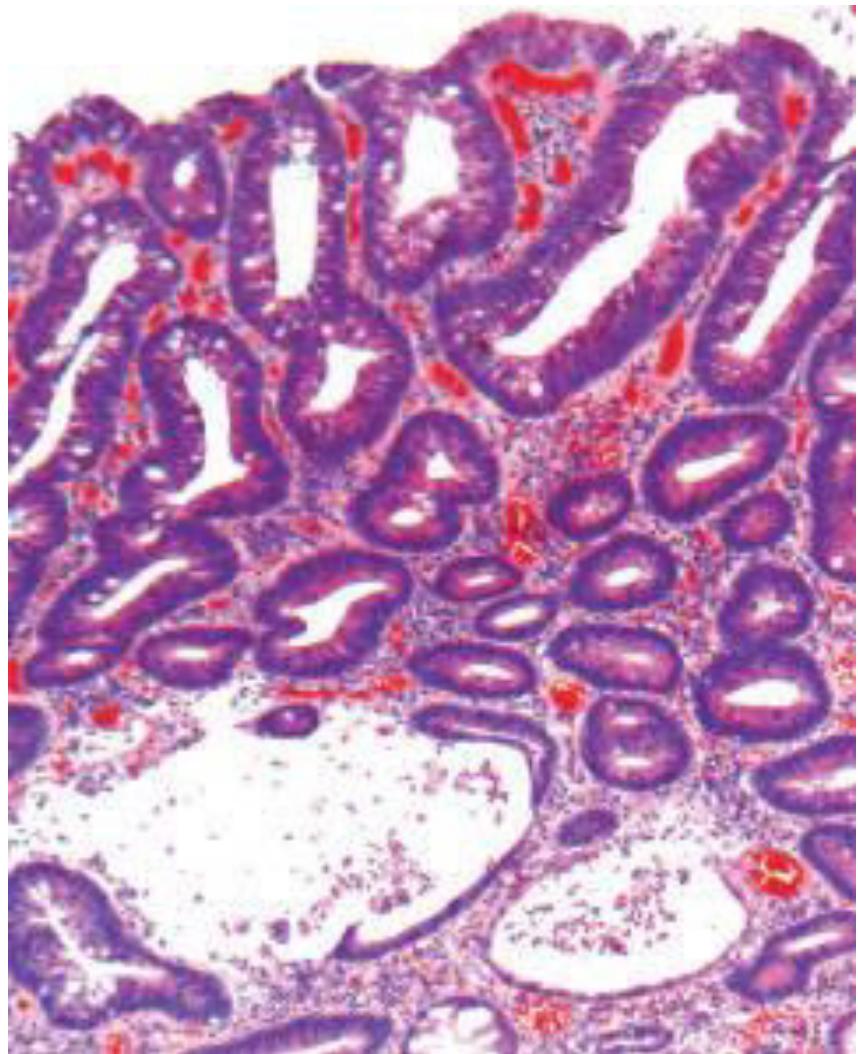


Colon adenoma

- ▶ 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

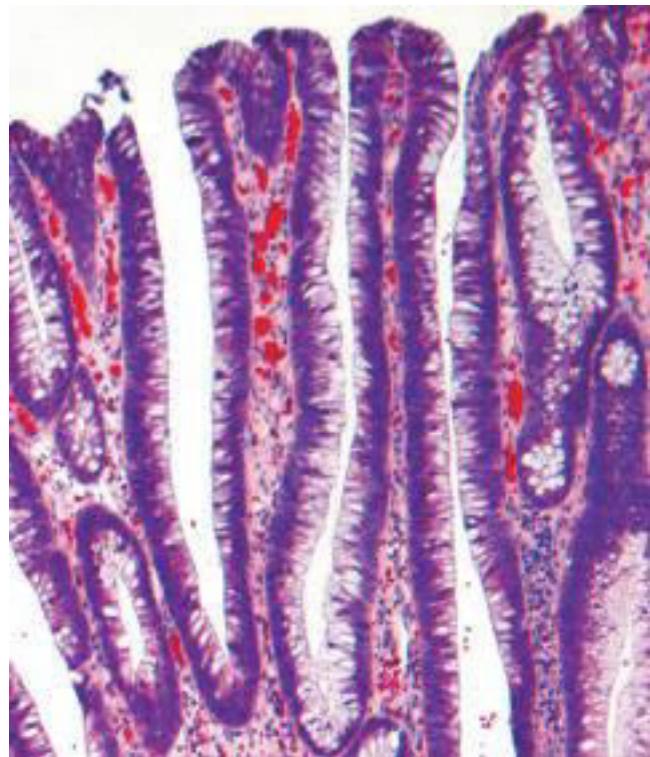


Tubular adenoma





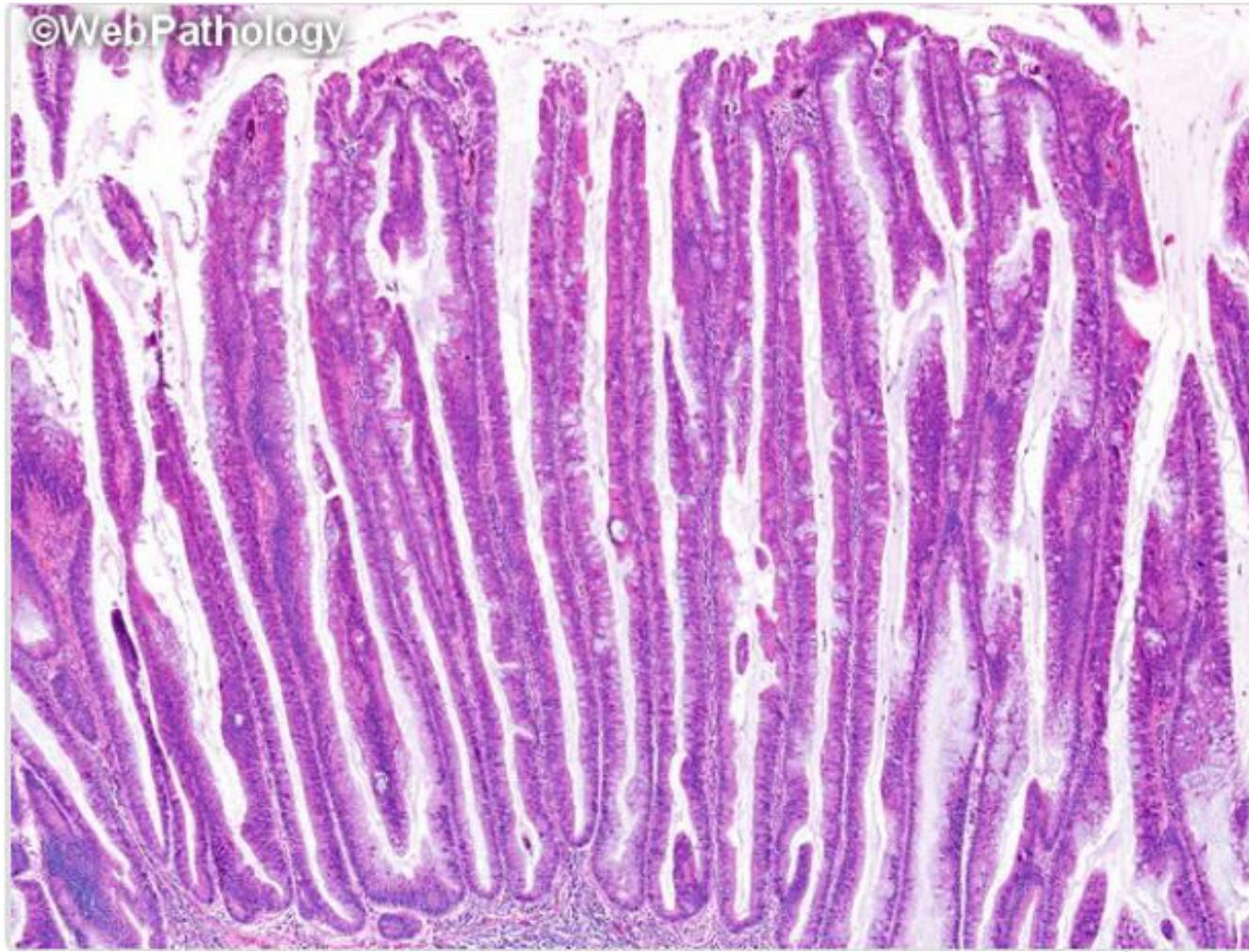
Villous adenoma.



- ▶ Long slender villi.
- ▶ More frequent invasive foci
- ▶ **Architecture:**
 - ▶ Tubular.
 - ▶ Tubulovillous.
 - ▶ Villous.



Villous adenoma



Familial Syndromes

- ▶ Syndromes associated with colonic polyps and increased rates of colon cancer
 - ▶ Genetic basis.
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- ▶ **Familial Adenomatous Polyposis (FAP)**
 - ▶ **Hereditary Nonpolyposis Colorectal Cancer (HNPCC)**



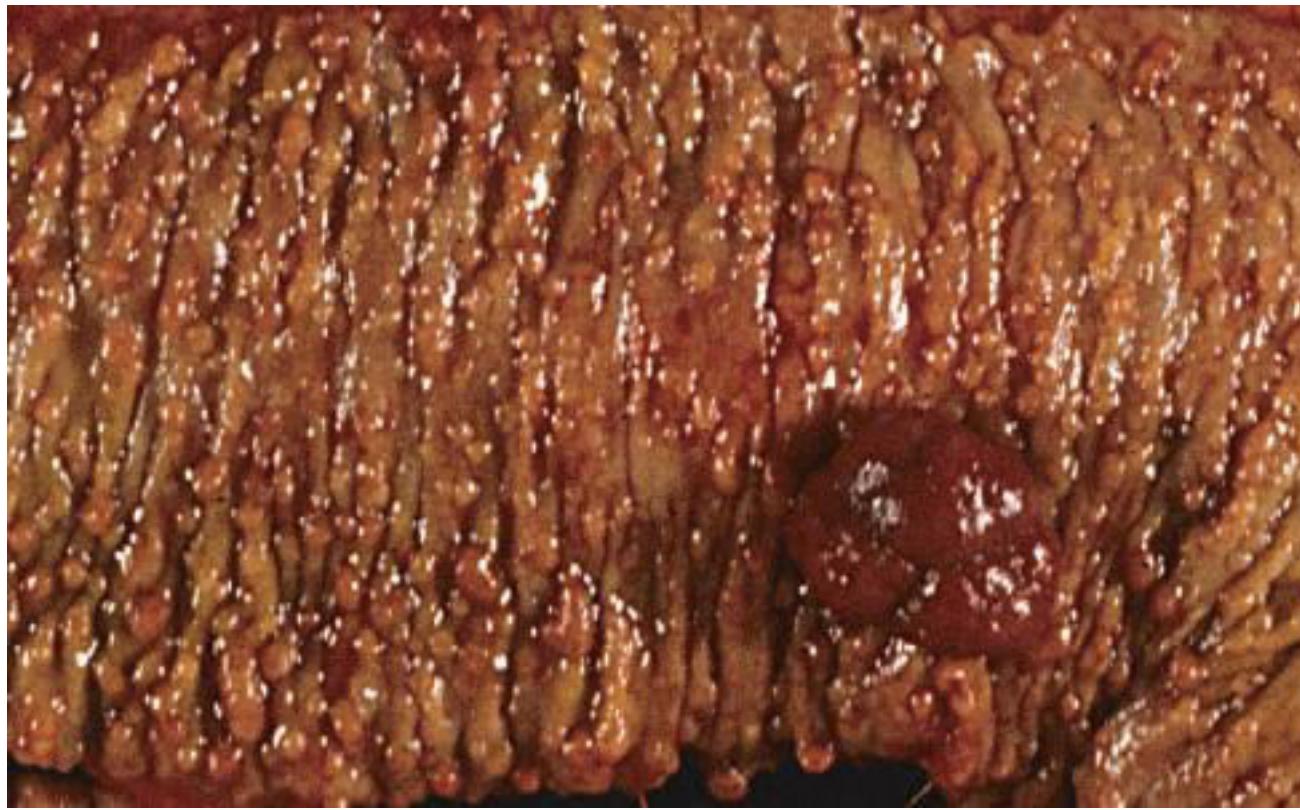
Familial adenomatous polyposis FAP

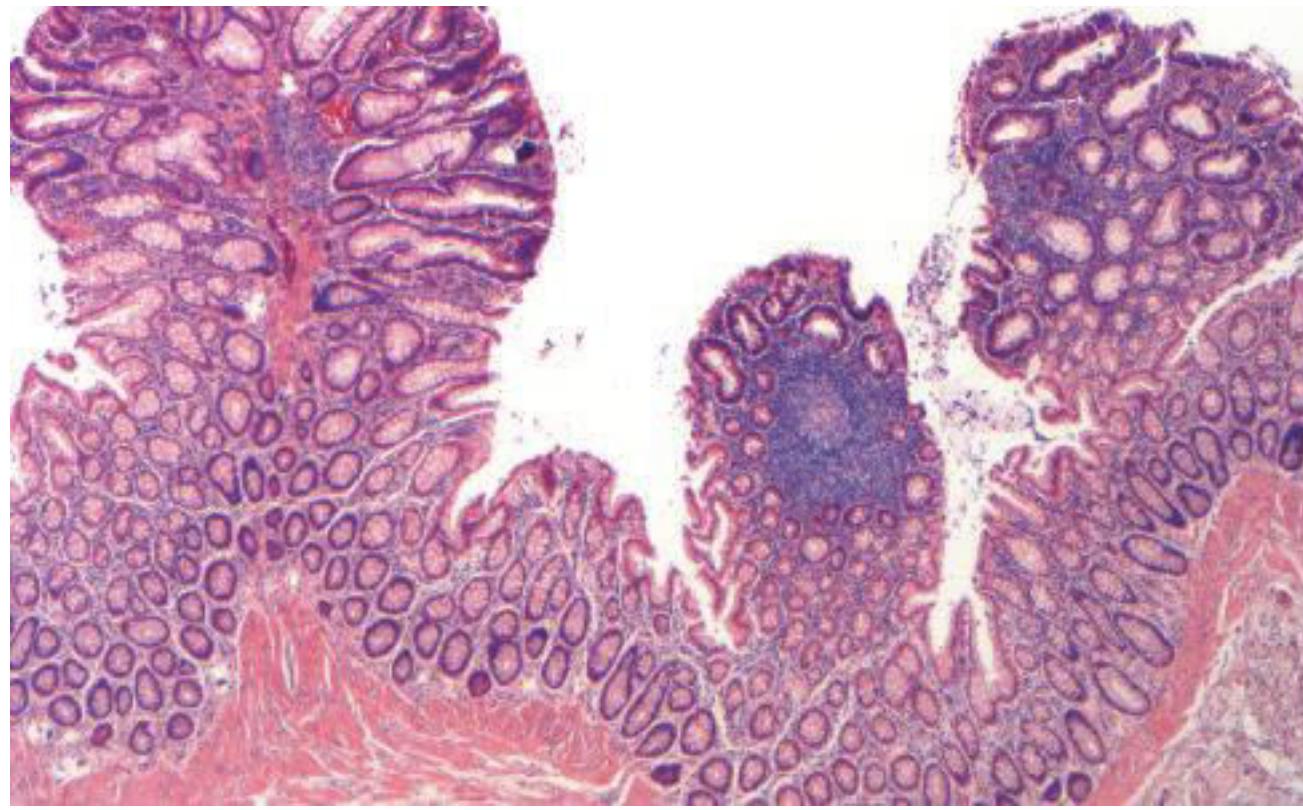
- ▶ Autosomal dominant.
- ▶ Numerous colorectal adenomas: teenage years.
- ▶ Mutation in APC gene.
- ▶ At least 100 polyps are necessary for a diagnosis of classic FAP.
- ▶ Morphologically similar to sporadic adenomas
- ▶ 100% of patients develop colorectal carcinoma, IF UNTREATED, often before age of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

