Polyps & neoplastic disease

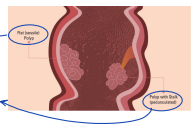
Polyps

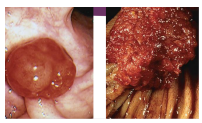
Neoplasms

* Colon is the most common site for polyps

Polyps → Sessile: No stalk

Polyps → Pedunculated: Stalk





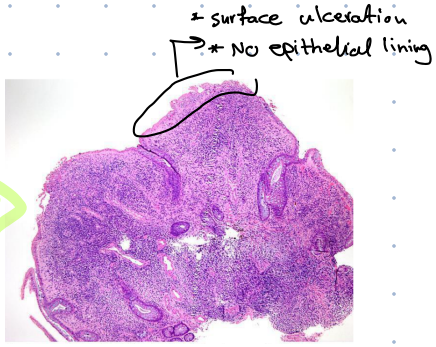
Polyps → Neoplastic polyps

Polyps → Non-Neoplastic polyps

Non-Neoplastic polyps → inflammatory, hamartomatous, 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.



Hamartomatus polyps:-

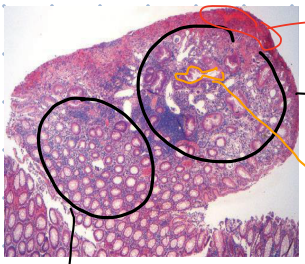
- disorganized, mature cells, present in that site.
- Sporadic, syndromatic

Hamartomatus polyps → Juvenile polyps, Peutz-Jeghers Syndrome

- Most common

hamartomatus polyps

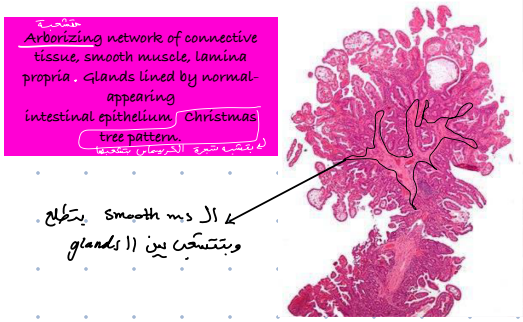
Juvenile polyps

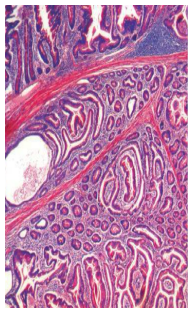


- pedunculated
- Reddish lesion
- cystic spaces in cut section
- dilated glands filled with → mucin, inflammatory depris
- granulation tissue on surface.

Juvenile polyps
- Sporadic (solitary) → children younger than 5 years. → rectum
- Syndromatic (multible) → 3-100 ، Mean age = 5 years → Autosomal dominant → TGF-B mutation → increased the risk of colonic adenocarcinoma

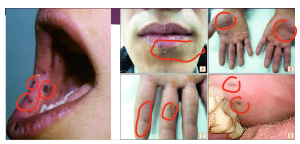
peutz-jeghers syndrome





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, genital organs [ovaries, uterus, and testes,
- LKB1/STK11 gene mutation.

* mucocutaneous pigmentation.



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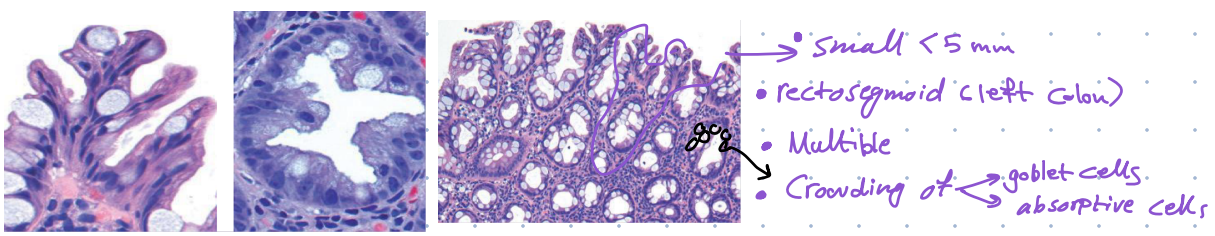
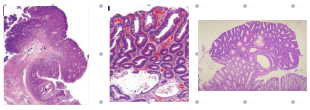
hyperplastic polyps:-

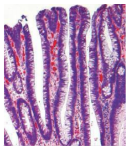
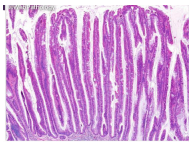


- small <5 mm
- rectosegmoid (left colon)
- Multible
- Crowding of → goblet cells, absorptive cells

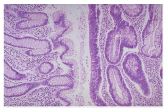
- 5th - 6th decade (50-60 years age)
- No malignant potentiond
- ↓ epithelial turnover
- delay shedding of surface epithelium → lead to → pile up of goblet cells & epithelial overcrowding



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



Colon adenoma

Long slender villi. More frequent invasive foci. Architecture: Tubular, Tubulovillous, Villous.

Villous adenoma

Adenoma

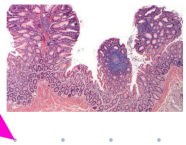
- increase with age
- western diet & lifestyles increase risk
- presence of epithelial dysplasia (low - high)
- USA: screening colonoscopy at 50 years.
  - earlier screening with family history
- precursor of majority of colorectal adenocarcinoma
- Most adenoma Dont progress to carcinoma

** Familial syndromes ↔ Familial adenomatous polyp (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,

FAP

Gardner syndrome

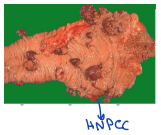
- intestinal polyps
- osteoma → mandible, skull, long bone
- epidermal cysts
- dismoid & thyroid tumors
- dental abnormality

Turcot syndrome

- intestinal adenomas
- CNS tumors (medulloblastoma, glioblastoma)

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. ** *



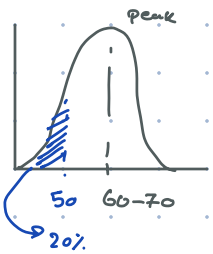
HNPCC
* مستوى FAP يدن الpolyp مو كتار

exception usually يدن بكونوا بال left أما هاد بعبر بال Right

* الدكتور بجيب مال mutated genes فانتبهولهم

Colonic adenocarcinoma

- Most common malignancy of GI tract
- Small intestine is uncommonly involved by neoplasia



- Aspirin & NSAIDs → has a protective effect
- Cox-2 promotes epithelial proliferation

pathogenesis:- (sporadic > familial)

Two pathways → APC/B-catenin pathway → ↑ in WNT signaling.
→ Microsatellite instability pathway

The APC/B-catenin pathway:

* APC inactivation → ↑ B-catenin → ↑ MYC & Cyclin-D1 → promote proliferation
  (both copies (1st & 2nd hit))
* KRAS (activation) → inhibit apoptosis → an additional mutation
* $SMAD_2 + SMAD_4$ mutation
* TP_{53} mutation

The microsotellite instobility pathway

mismatch repair deficiency → loss of mismatch repair genes → Mutation accumulate in microsatellite repeats

Microstellite instability

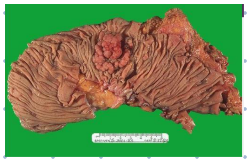
** Uncontrolled cell growth if located in coding or promoter regions of genes involved in cell growth and apoptosis (TGF-B and BAX genes)

Etiology	Molecular Defect	Target Gene(s)	Transmission	Predominant Site(s)	Histology
Familial adenomatous polyposis (70% of FAP)	APC/WNT pathway	*APC*	Autosomal dominant	None	Tubular, villous; typical adenocarcinoma
Hereditary nonpolyposis colorectal cancer	DNA mismatch repair	*MSH2, MLH1*	Autosomal dominant	Right side	Sessile serrated adenoma; mucinous adenocarcinoma
Sporadic colon cancer (80%)	APC/WNT pathway	*APC*	None	Left side	Tubular, villous; typical adenocarcinoma
Sporadic colon cancer (10%–15%)	DNA mismatch repair	*MSH2, MLH1*	None	Right side	Sessile serrated adenoma; mucinous adenocarcinoma

MORPHOLY

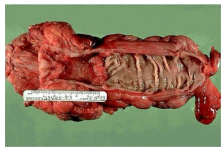
Macroscopic:

Proximal colon tumors: polypoid, (exophytic) masses



Proximal colon: rarely cause obstruction.

Distal colon: annular lesions ("napkin ring") constrictions & narrowing



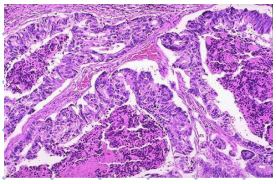
MORPHOLY

Microscopic:

Dysplastic GLANDS with strong desmoplastic response.

Necrotic debris are typical.

Some tumors give abundant mucin or form signet ring cells.



→ Adenocarcinoma with necrosis

Clinical features of adenocarcinoma

early is asymptomatic

iron deficiency anemia in an older male or postmenopausal female is GIT Cancer until proven otherwise

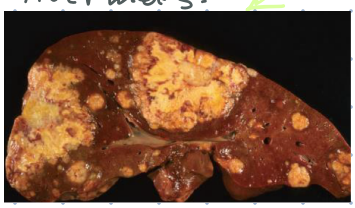
Cecal & right Cancer:
Fatigue & weakness (iron deficiency anemia)

left sided carcinoma:-
- Occult bleeding
- Changes in bowel habits
- Cramping (LLQ) discomfort

Most important two prognostic factors are Depth of invasion Lymph node metastasis.

Distant metastases (lung and liver) can be resected.

liver mets.



Appendix

acute appendicitis

- May occur in any age but most common in young adults

Normal true diverticulum of the cecum

Tumors of the appendix

Diagnosis:

Mesenteric lymphadenitis,
Acute salpingitis,
Ectopic pregnancy,
Mittelschmerz (pain associated with ovulation),
Meckel diverticulitis.

luminal obstruction (50-80)%.
↳ (ILPI) → impaired venous drainage.

- inflammatory response rich in neutrophils & edema
- stasis associated with bacterial proliferation
- ischemic injury

by fecalith
less commonly
gallstone, tumor, worms

* Diagnosis requires neutrophilic infiltration of the muscularis propria
Acute suppurative appendicitis >> more severe >> focal abscess formation. Acute gangrenous appendicitis >> necrosis and ulceration.

Clinicol Features

Early acute appendicitis: periumbilical pain

Later: pain localizes to the right lower quadrant,

Nausea, vomiting, low-grade fever, mildly leukocytosis. A classic physical finding is McBurney's sign (McBurney's point).

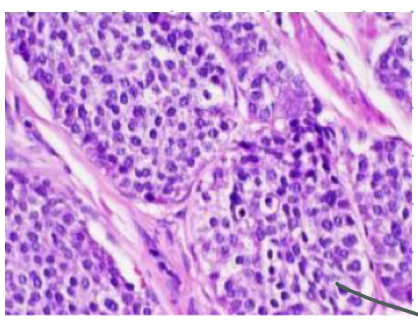
Signs and symptoms are often absent, creating difficulty in clinical diagnosis.

Tumors of the appendix.

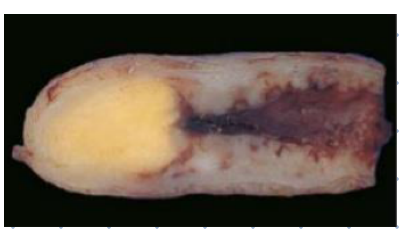
* Most common tumor → carcinoid (neuroendocrine)

Found during → surgery
→ on examination of resected appendix

- Nodal metastases & distant spread are rare



microscope → Nests salt & pepper



Grossly :-

لجنة الطب والجراحة

بالتوفيق،بارك الله في وقتكم و إنجازكم وهمتكم🩶