POLYPS.

- polyps are most common in colon and rectum, but can also be in stomach, esophagus, small intestine.

- most, if not all, polyps begin as small elevations of the mucosa.

Classification

- By gross appearance.
  - Sessile
    - no stalk
  - pedunculated.
    - well defined stalk.
- By histopathological appearance.
  - Non-Neoplastic
    - Inflammatory
    - Hamartomatous
    - Hyperplastic.
  - Neoplastic
    - adenoma (benign)
    - malignant

Hamartomatous Polyps.

- hamartomatous polyps occurs sporadically or as part of various genetically determined or acquired syndromes.

- They consists of mature tissues that are normally present at the site in which they develop.

- Juvenile polyposis
- Peutz Jeghers syndrome
- Cowden syndrome (PTEN).
  Bannayan – Ruvalcaba – Riley syndrome.
- Tuberous sclerosis.
- Familial adenomatous polyposis. (APC)

Juvenile polyposis

- they are focal malformations of epithelium and lamina propria that can be sporadic or syndromic.
- mc mutation : SMAD4. other : BMPR1A
- age group < 5 years.
- location : rectum
- commonly presents as rectal bleeding.
- Sporadic JP → called as retention polyps → solitary.
- (AD) Juvenile polyposis syndrome → 3-100 polyps → ↑ risk of colonic adenocarcinoma.

Morphology

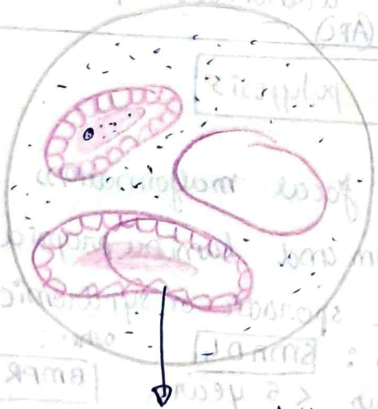
Gross: • < 3cm diameter.
• pedunculated
• reddish colour.
• smooth surface.

Cut section: cystic spaces.

M/C

• cystically dilated glands filled with mucin & inflammatory debris.

• Lamina propria is expanded due to mixed inflammatory infiltrates.



• cystic spaces filled with mucin
• inflammatory infiltrates in lamina propria.

Peutz - Jeghers

• autosomal dominant Sx.
• median age of presentation: 11.
• loss of function mutation in STK11 is seen (50% cases).

→ Multiple polyps.
→ muco cutaneous hyperpigmentation.

Brown macules: lips, buccal mucosa, palmar - hands, genitalia

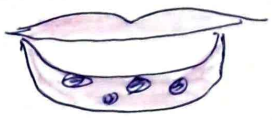
• Increased risk of several malignancies.
• sex cord Tx of testes
• gastric & SI cancer.
• colon, pancreatic, breast etc..

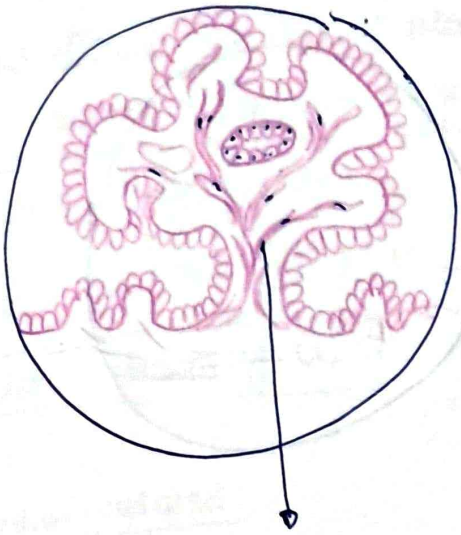
Morphology

Site: Small intestine. M/C
number: many.

Gross:
• large.
• pedunculated. with lobulated contour.

M/C.
• arborizing network of connective tissue, smooth muscle, lamina propria.
• glands lined by normal appearing Intestinal epithelium.





→ arborizing network of connective tissue, smooth muscle, lamina propria.

→ gland lined by normal-appearing intestinal epithelium.

Inflammatory polyps.

- raised nodules of inflamed, regenerating epithelium.
- not precancerous.
- commonly found in ulcerative colitis.

Hyperplastic Polyp.

- Benign epithelial proliferations.
- results from - ↓ epithelial cell turnover.
  - delayed shedding of surface epithelial cell.
  - ↓
  - piling up of goblet & absorptive cells
- No malignant potential.
- must be distinguished from sessile serrated

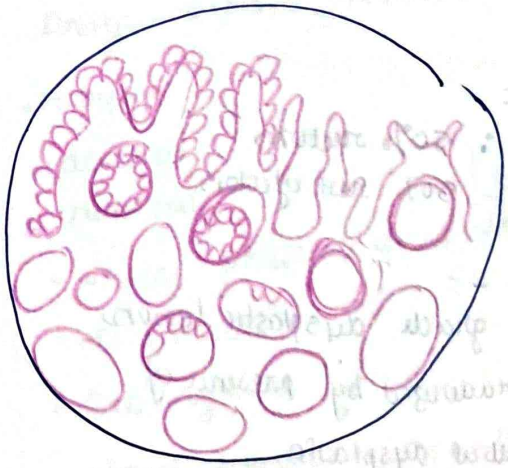
Morphology

Site : left colon.

- small, smooth, nodular protrusions of mucosa.
- sessile.
- Number: single / multiple.

M/C

- elongated colonic crypts with pseu.
  - ↓
  - Saw toothed / serrated appearance.
- serration is limited to upper 1/3rd.



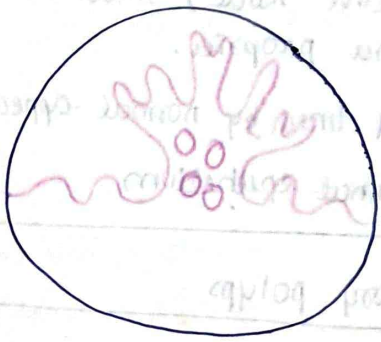
- polyp surface with irregular tufting of epithelial cell.
- tufting due to epithelial overcrowding.
- serrations - only in upper 1/3rd.

Neoplastic polyp

- most common : colonic adenomas.
  ↓
  benign with malignant potential.

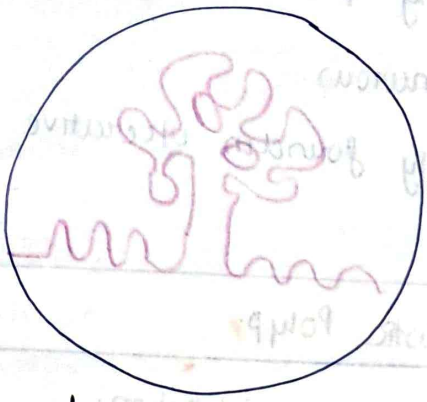
Neoplastic polyps - adenomas (architectures)

- tubular adenoma (adenomatous polyp).
- tubulovillous
- villous.



GROSS.

- site : 50% rectum
  50% rest of colon.



- m/c
  - low grade dysplastic lesions.
    characterized by presence of epithelial dysplasia.

Tubular → smooth surface.

Sessile serrated adenoma

→ abnormal crypt architecture. (L, T,).

→ lateral crypt growth.

(23 KD) Discuss the type of ulcers in Intestine - (small & large Intestine).

- short essay.

Ulcerative lesions of GIT.

① Infection related.

- Amoebic ulcer. {large intestine only}.
- Typhoid ulcer. {ileum}.
- TB ulcer.
- hyperplastic TB.
- Bacillary dysentry → C. Jejuni & difficile.

② IBD → ulcerative colitis (large intes only), crohn's disease.

③ chronic gastric ulcers - PUD. gastric & duodenal ulcers.

④ Acute - stress ulcers → curling's cushings.

⑤ Drug induced ulcers. → NSAIDs

⑤ Non - stress related.

- Gastric antral vascular ectasia

⑥ Carcinoma.

⑦ others : GVHD, Behcet's syndrome.

Gastritis.

- chronic gastritis
- H. pylori gastritis. (SE)

- Gastritis ⇒ Inflammation of gastric mucosa.

acute gastritis.

- acute gastritis is a transient inflammation of gastric mucosa.

Etiology.

- Drugs - aspirin, NSAIDs.
- H. pylori
- alcohol
- chemicals.
- severe physiological stress → {burns}
- BIle reflux
- viral infections (eg: CMV)

→ Same diagram as ← PUD

ie, imbalance between damaging & protective factors.

Chronic gastritis

Chronic inflammation of stomach mucosa associated with mucosal injury.

optional dysfunction... idk if it's correct.

Causes.

1. H. pylori infection — m/c.
2. Autoimmune Gastritis.
3. Chronic Bile reflux.
4. radiation injury
5. mechanical injury.
6. Systemic D/s (IBD, ~~GVHD~~).

H. pylori Gastritis.

- chronic gastritis caused by colonization of H. pylori

→ H. pylori → • gram negative
• spiral shaped
• curved bacilli

→ resides in foveolar cells.

→ seen in most patients with duodenal/gastric ulcers & chronic gastritis.

→ m/c site — antrum.

Risk factors.

- poverty
- Household crowding.
- rural areas.
- limited education
- age > 60 years

Route: feco-oral route of transmission.

Virulence factors

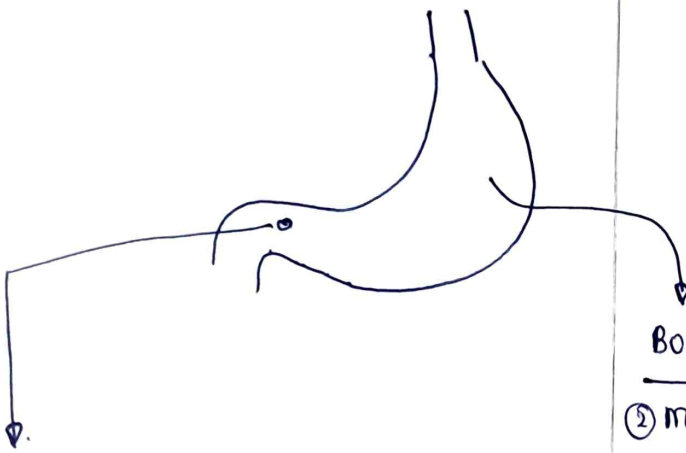
1. flagella : motility in viscous mucous.
2. urease : urea → Ammonia
   pH ↑, survival ↑ of bacteria
3. adhesins : adherence to foveolar cells
4. cag A toxin : alters cell signalling.
   - proinflammatory
   - pro proliferative.
   ↑ inflammation.
   ↑ cancer risk.
5. vac A gene : cytotoxin.

Host factors

- ↑ expression of proinflammatory cytokines — TNF, IL1β
- ↓ expression of anti-inflammatory cytokine IL-10.
- all increase risk of
  - pangastritis
  - atrophy
  - gastric carcinoma.

Pathogenesis

Two major patterns.



① Antral predominant gastritis.

- H. pylori M/C affects antrum.
- ↑ Gastrin production.
  ↓
  ↑ parietal cell mass.
  ↓
  ↑ acid production.
  ↓
  ↑ risk of gastric / duodenal PUD @ antrum.

Body / Fundus.

② multifocal atrophic gastritis.

↓

long standing gastritis.

① atrophic gastritis.
+
② Intestinal metaplasia.
↓
↑ risk of gastric adenocarcinoma.

↓ parietal cell mass
↓
↓ acid production.
↓
↓ risk of PUD.