INTESTINAL TUBERCULOSIS

- TB can involve any part of G.I.T. from mouth to anus , peritoneum and pancreatobiliary system.
- TB of G.I.T. is 6th most common extra-pulmonary site.
- M. Tuberculous is the pathogen in most cases.
- M. bovis in some part of the world.

ETIOPATHOGENESIS :

- By ingestion :

* Ingestion of food contaminated by mycobacteria
  - Primary Intestinal Tuberculosis.
* Ingestion of selfsputum,containing mycobacteria
  - Secondary Intestinal Tuberculosis.
* Hematogenous spread from primary lung focus.

* Direct spread from adjacent organ like fallopian tube or other abdominal organs.
* Via lymph channels from infected lymph node.

PATHOGENESIS :

Bacilli in the depth of mucosal glands

↓

Inflammatory reaction

↓

Phagocytes carry bacilli to Payer's patches

↓

Formation of tubercle & necrosis

↓

Edema & sloughing

↓

↓

Ulcer formation

↓

Collagen accumulation, thickening & stenosis

↓

Inflammation spreads to serosa

↓

Bacilli carried via lymphatics to regional lymphnodes

PATHOGENESIS OF INTESTINAL TUBERCULOSIS

Ingestion of infected milk OR sputum

→ Entry of bacilli into the mucosa of the GI tract

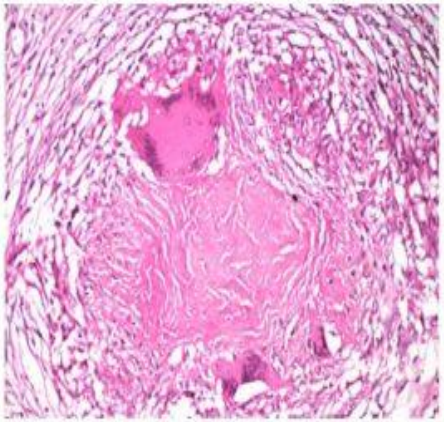
→ Granulomas in the submucosa

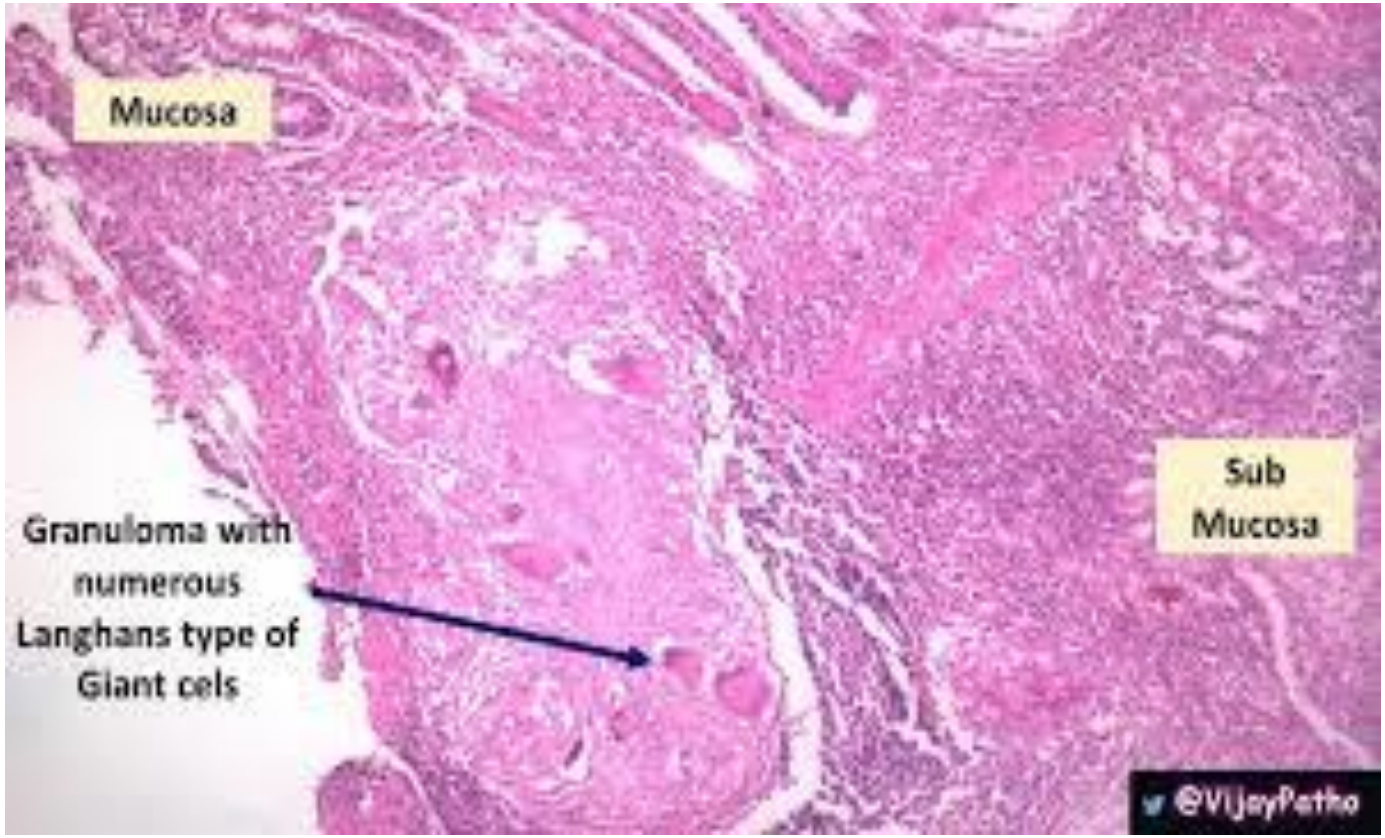
↓ Caseation necrosis

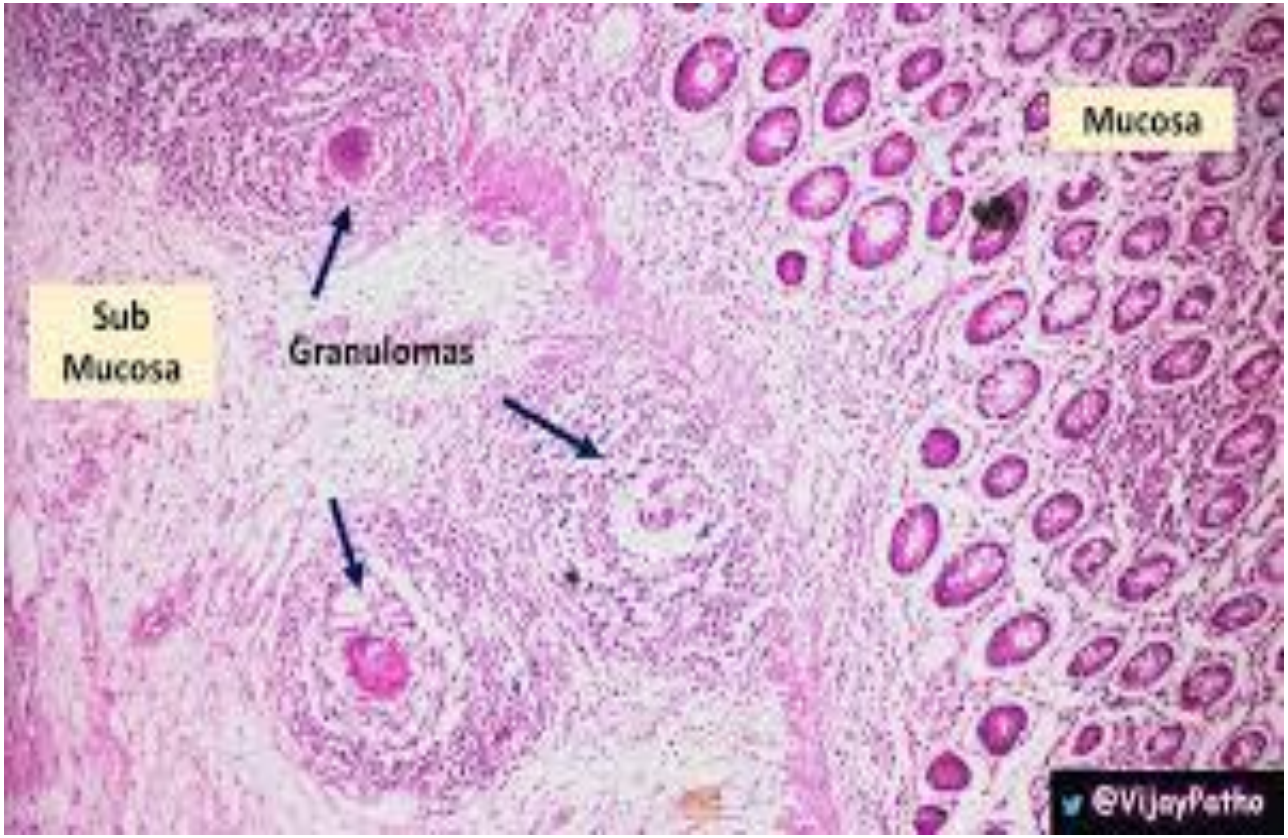
← Ulceration of the overlying mucosa

↓ Spread into deeper layers of intestine

← Spread into adjacent lymph nodes & Peritoneum




Mucosa
Granuloma with numerous Langhans type of Giant cels
Sub Mucosa
@VijayPatho


Mucosa
Sub
Mucosa
Granulomas
@VijayPatho

Tubercles on serosa :

ileocaecal tuberculosis :

- most common site.
- Characteristic lesions :
  1. Ulceration –
     * multiple circumferential transverse ulcers (Girdle ulcers) with skip lesions.
     * napkin ring strictures in long standing ulcers.(ileum)
     * Intestinal node involvement with caseation & abscess

2. Hyperplastic type :

* Fibroblastic activity in submucosa & subserosa causes thickening of bowel wall with lymphnode enlargement.

3. Stricturous type :

* Single or multiple

4. Diffuse Colitis :

* Rare.
* Similar to Ulcerative Colitis.

TB INFLAMMATION : Intestine

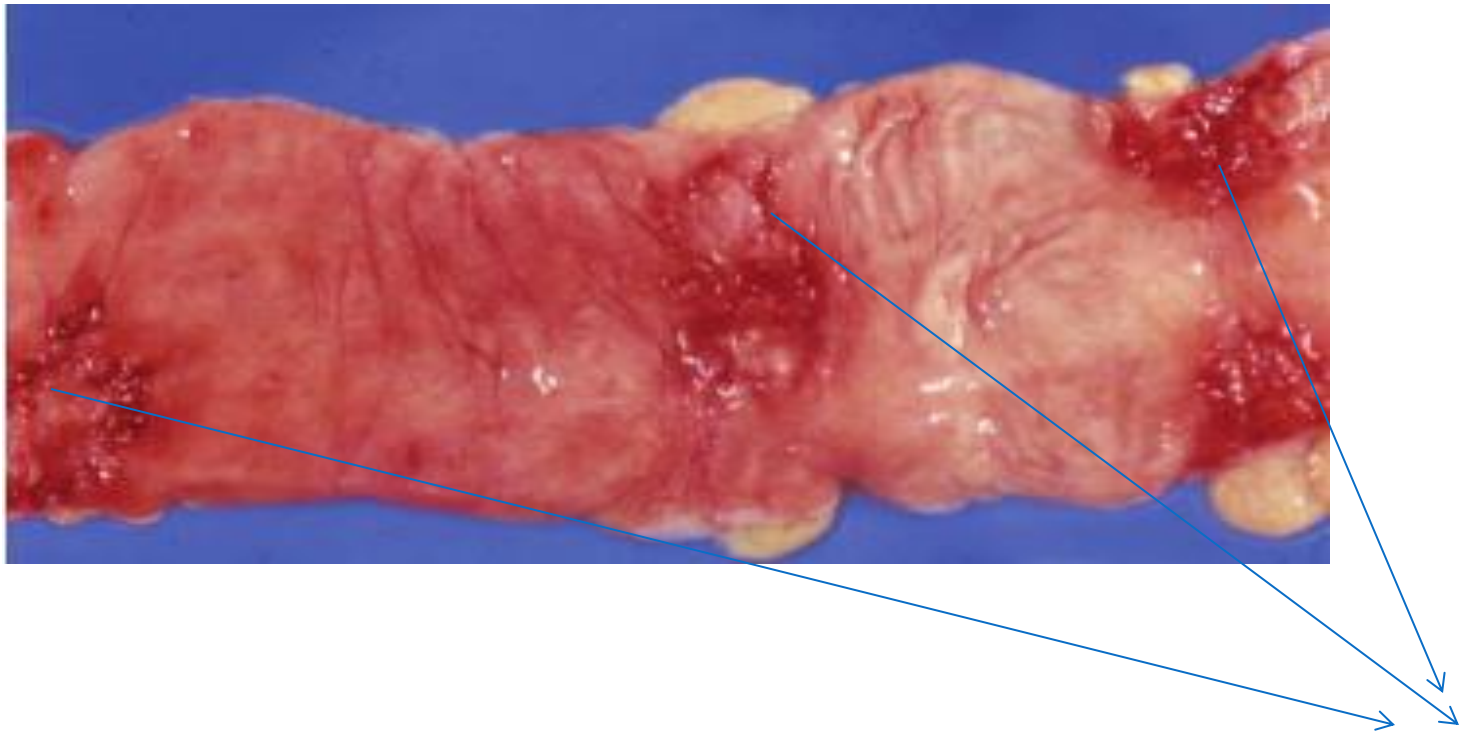


Figure 1: Transversely placed tuberculous ulcer involving entire circumference of the bowel wall.

Frequency of T.B.lesions in G.I.T. :

- ileum > caecum > ascending colon > jejunum > appendix > sigmoid colon > rectum > duodenum > stomach > oesophagus
- More than one site may be involved.

Symptoms :

1. Local symptoms depending on the site involves .
2. Constitutional symptoms like ,
   - fever
   - malaise
   - anaemia
   - weight loss
   - night sweat

3. Pain in abdomen
    * Colicky – if luminal compromise.
    * Dull & continuous , when mesenteric lymphnodes are involved.
4. Alteration of bowel habits.
5. Malabsorption.
6. Rectal bleeding , etc.

Complications :

- Intestinal obstruction.
- Malabsorption.
- Perforation. (2nd commonest cause of small intestinal perforation. 1st being typhoid fever.)
- Dissemination of T.B.
- Hemorrhage.
- Fecal fistula.

Investigations :

1. Blood investigations :
    - anaemia.
    - leucopenia with lymphocytosis.
    - raised ESR.
2. Mantoux test.
3. Chest x-ray.

4. X-ray abdomen :
   - intestinal obstruction.
   - calcified lymphnodes.
   - perforation.
5. USG abdomen.
6. Barium enema.
7. C.T. abdomen.

Treatment :

- Medical management :

-Same as for pulmonary tuberculosis.

By first & second line anti-tuberculous drugs.

- treatment to be continued for 6 months.

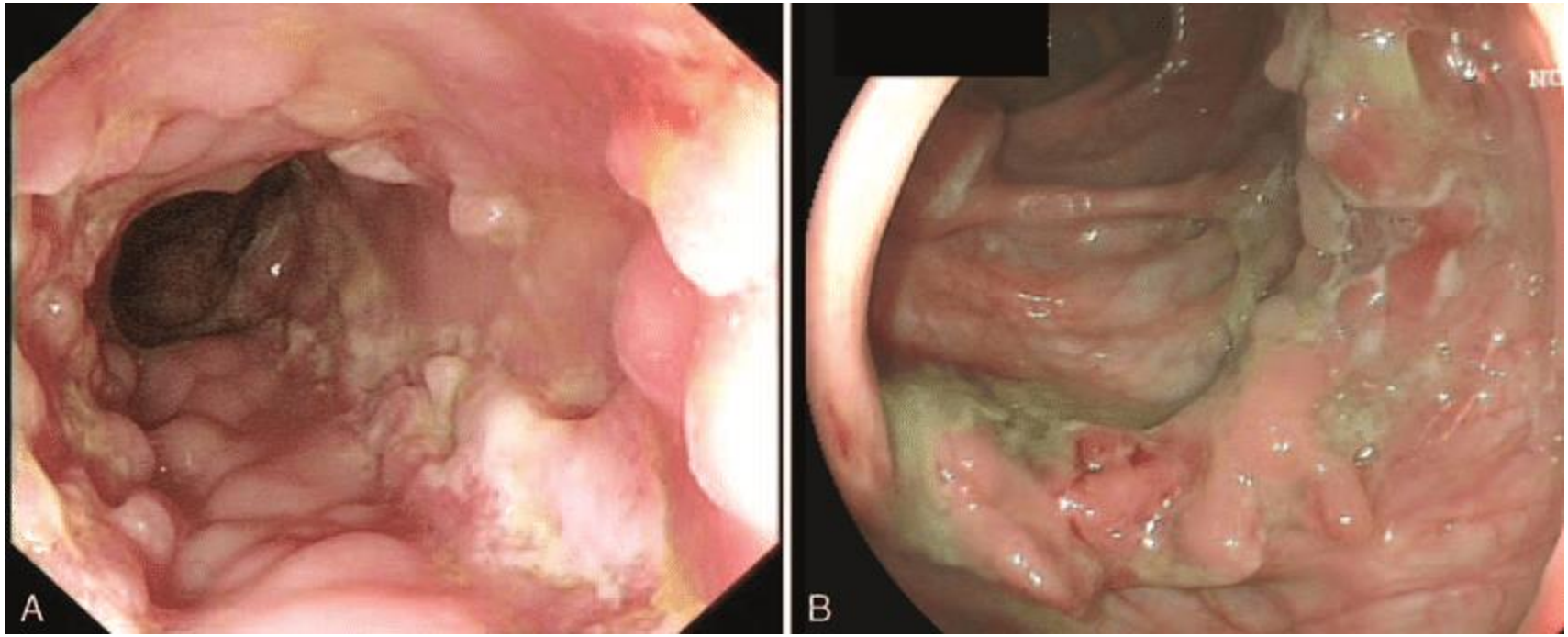
- supportive nutrition.

- Surgical management :

  Indications:

  - intestinal obstruction.
  - perforation.
  - severe haemorrhage.
  - intra-abdominal abscess & fistula.

Crohn's Versus Tuberculous Intestinal Ulcer :



COLONIC POLYPS :
(Neoplastic & Non-neoplastic)

- Polyp is defined as any growth project from mucous membrane into lumen.
- Polyp can be categorised by,

  1.size

  2.character of attachment : (a) sesile

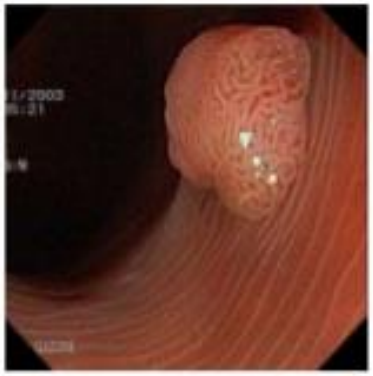
  (b) pedunculate

  3. cellular architecture : (a) adenomatous

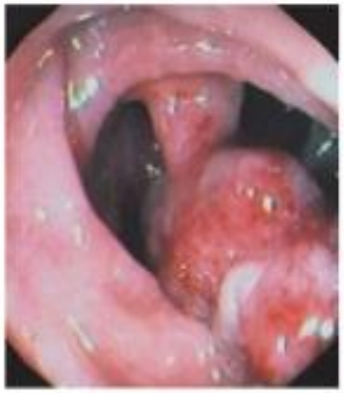
  (b) non-adenomatous

According to character of attachment

- **Sessile-** with a broad-based attachment to the colonic wall, or
- **Pedunculated-** attached to the colonic wall by way of a fibrovascular stalk



Sessile polyp



Pedunculated polyp

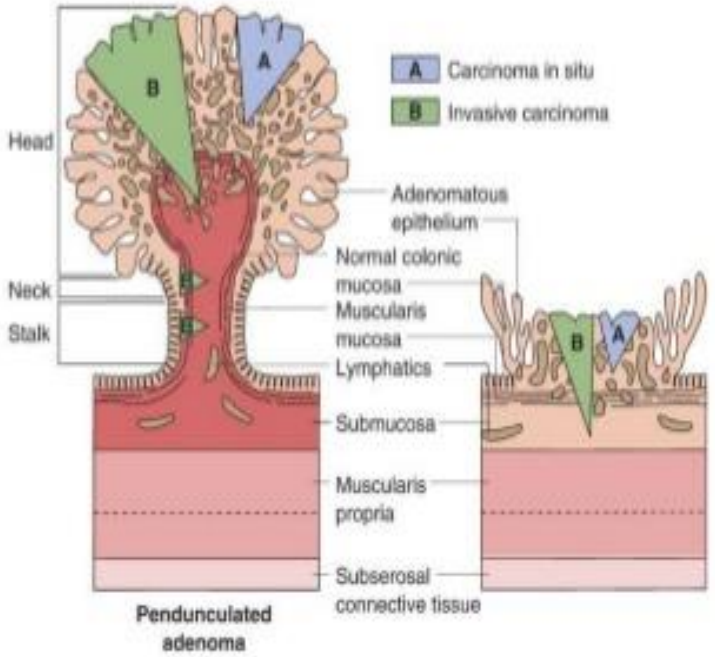


Diagram representation of cancer-containing polyps

CLASSIFICATION :

1. Inflammatory polyp.
2. Hamartomatous polyp.
3. Hyperplastic polyp.
4. Adenomas.

1. Inflammatory type :

- solitary rectal ulcer syndrome.

Clinical triad of (a) rectal bleeding.

(b) mucus discharge.

(c) inflammatory lesion of anterior rectal wall.

Etiology : Recurrent abration and ulceration of mucosa. So injury & healing leads to polypoidal mass.

2. Hamartomatous polyp :

(a) genetically determined. OR

(b) aquired.

Hamartomas are disorganised tumor like growth of mature cells.

Examples of Hamartomatous polyp are,

1. Juvenile type.
2. Peutz- Jeghers syndrome.
3. Cowden syndrome.

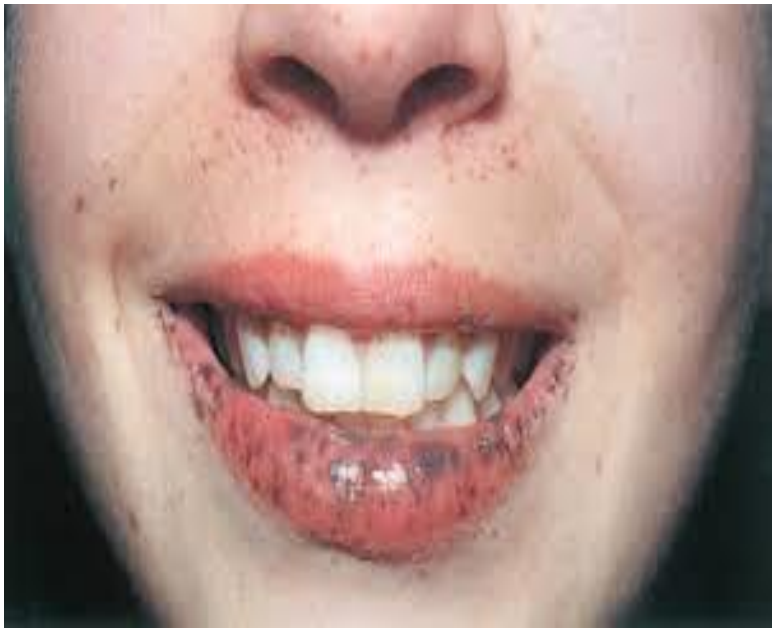
Juvenile polyps :

- most common Hamartomatous polyps.
- sporadic or syndromic.
- majority occurs in children younger than 5 years.
- located in RECTUM.
- cause rectal bleeding.
- sporadic are usually single.
- syndromic are 3 - 100 polyps.
- syndromic are autosomal dominant , associated with ↑ risk of COLO RECTAL CARCINOMA (CRC)

Peutz-Jegers Syndrome :

- rare autosomal dominant disorder.
- multiple polyps.
- muco-cutaneous hyperpigmentations.
- Christmas tree appearance at low power
- loss of function mutation in gene LKB1 / STK 11
- ↑ risk of carcinoma of colon,pancreas,breast, lungs, ovaries,uterus & testes.

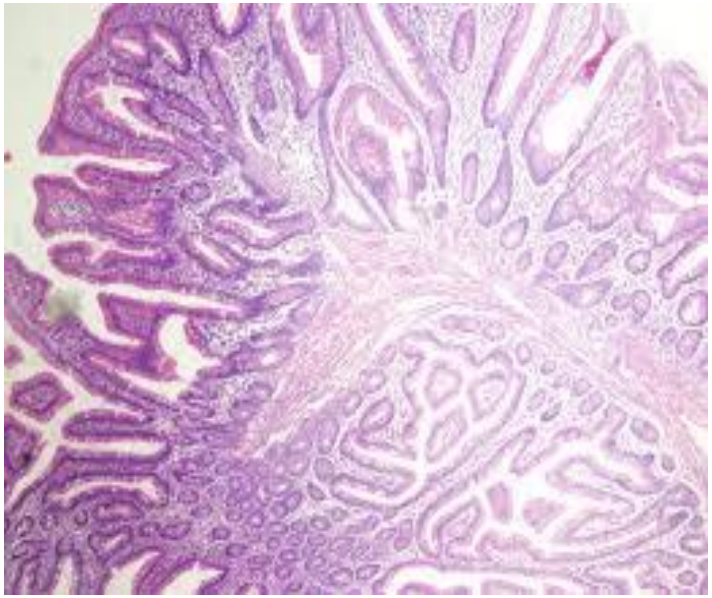
Mucocutaneous pigmentation PJ SYNDROME :



Peutz-Jeghers Polyp (PJP)

- Seen in Peutz-Jeghers Syndrome
- Most common in jejunum and ileum; colon variably involved
- Gross: variable size, small polyps sessile, large polyps pedunculated
- Micro: papillary architecture, arborizing compact bundles of smooth muscle separating glands into lobular configuration

Peutz-jegers polyp



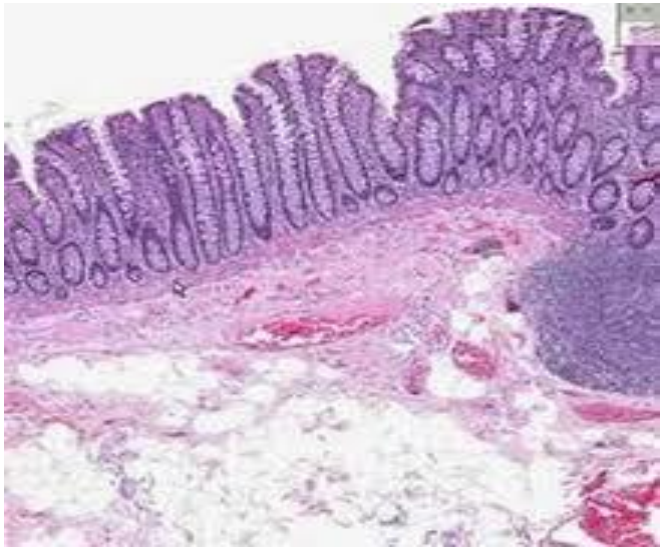
Hyperplastic polyps :

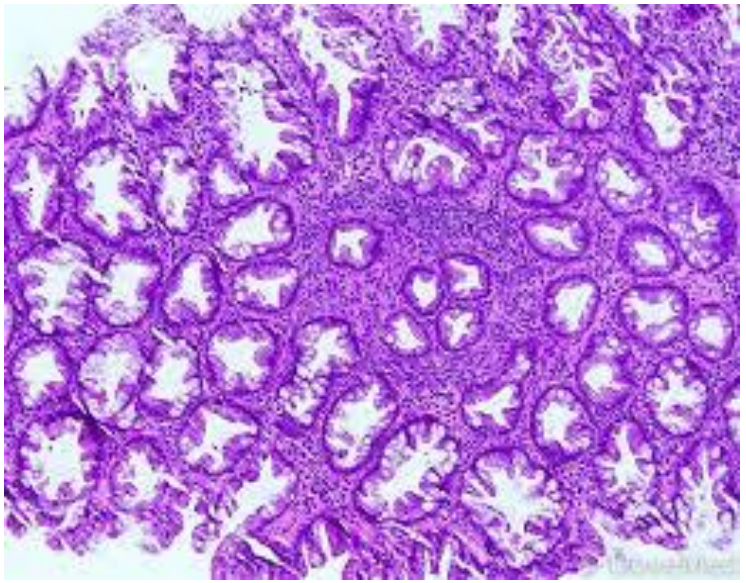
- seen in 6th to 7 th decade.
- ↓ epithelial cells turn over , so pile up of goblet cells.

Adenomas :

- they can be ,
  1. conventional adenomas.
  2. serrated lesions.
  3. Adenomatous Polyposis Coli syndrome(APC).

Normal Mucosa and hyperplastic polyp :





- Grossly they can be ,
    * sessile
    * pedunculated
- Microscopically ,
    * tubular
    * villous
    * tubulovillous

Tubular adenoma :

small,pedunculated & small ,round tubular glands.

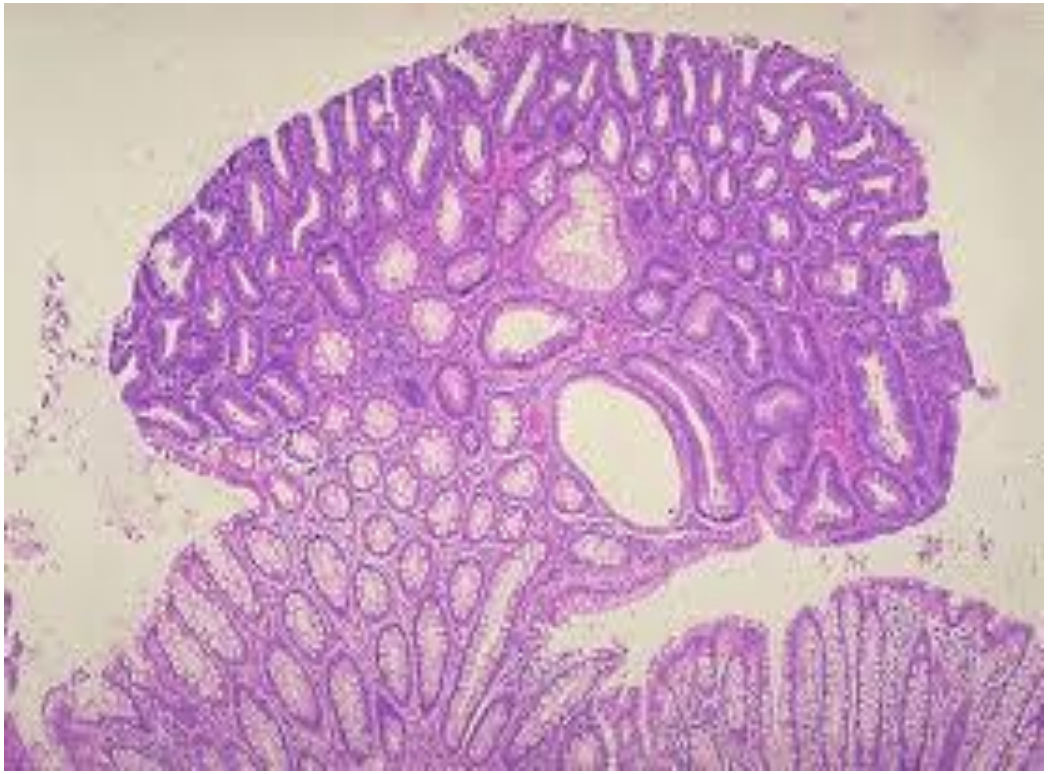
Villous adenoma :

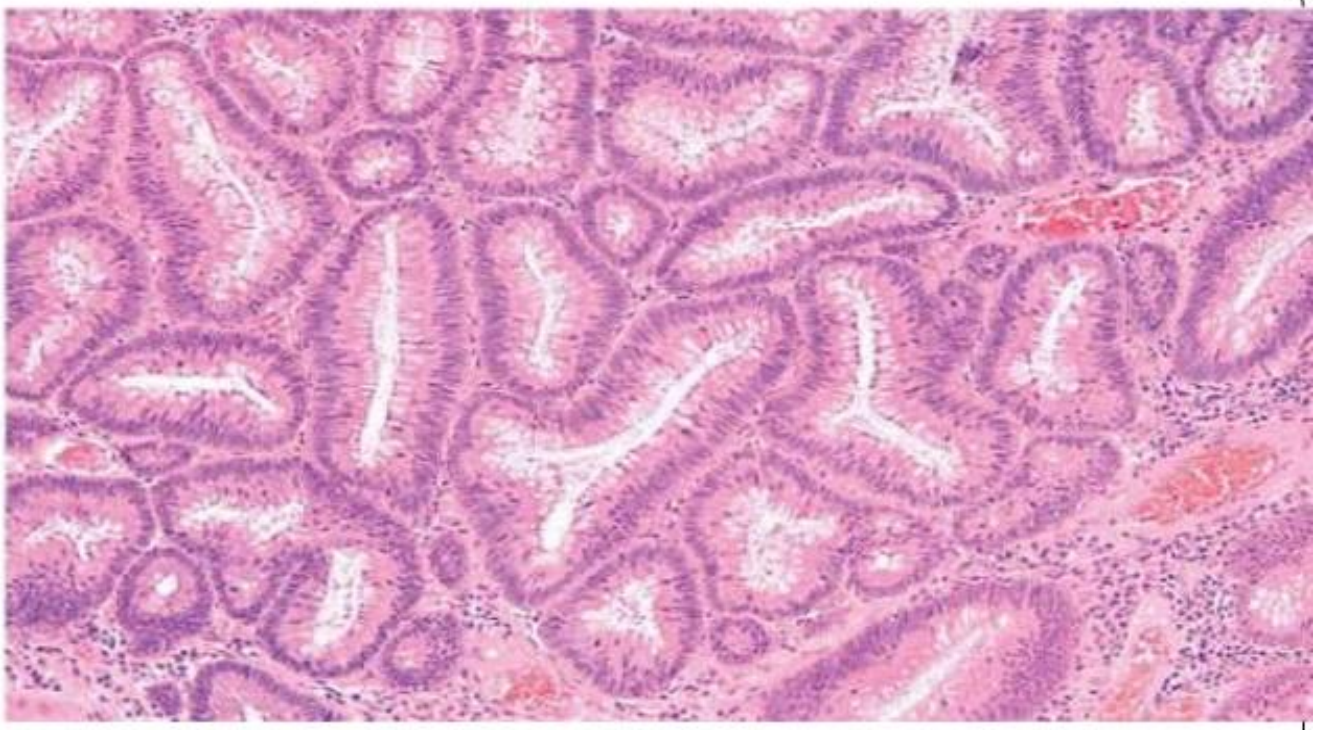
-large ,sessile & covered by slender villi.

Tubulovillous adenoma :

-mixture of two.

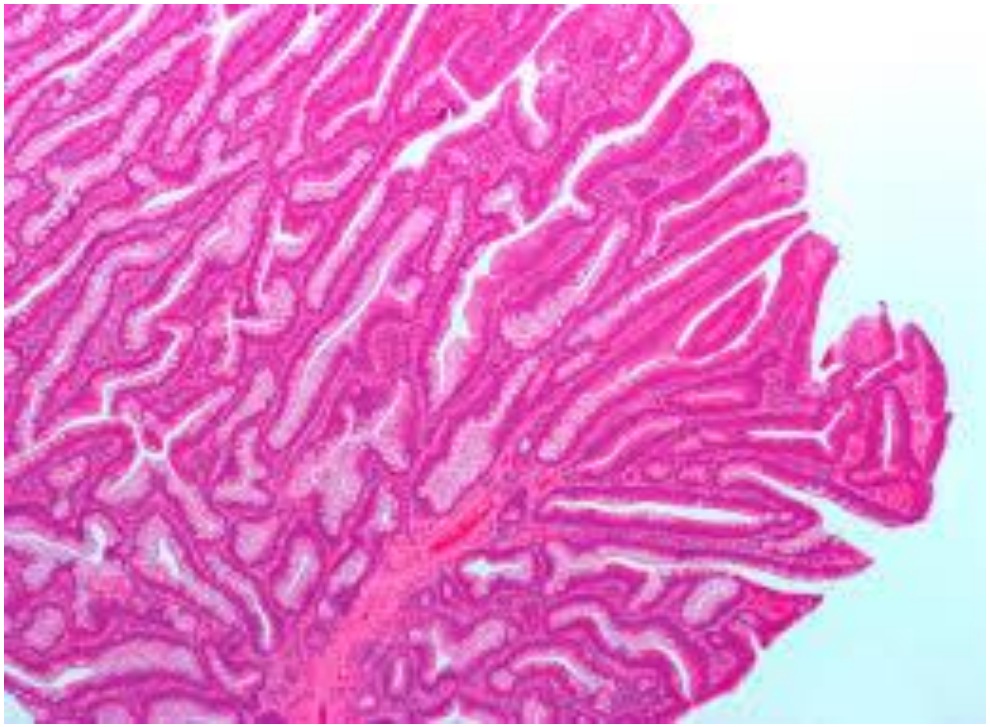
Tubular adenoma :

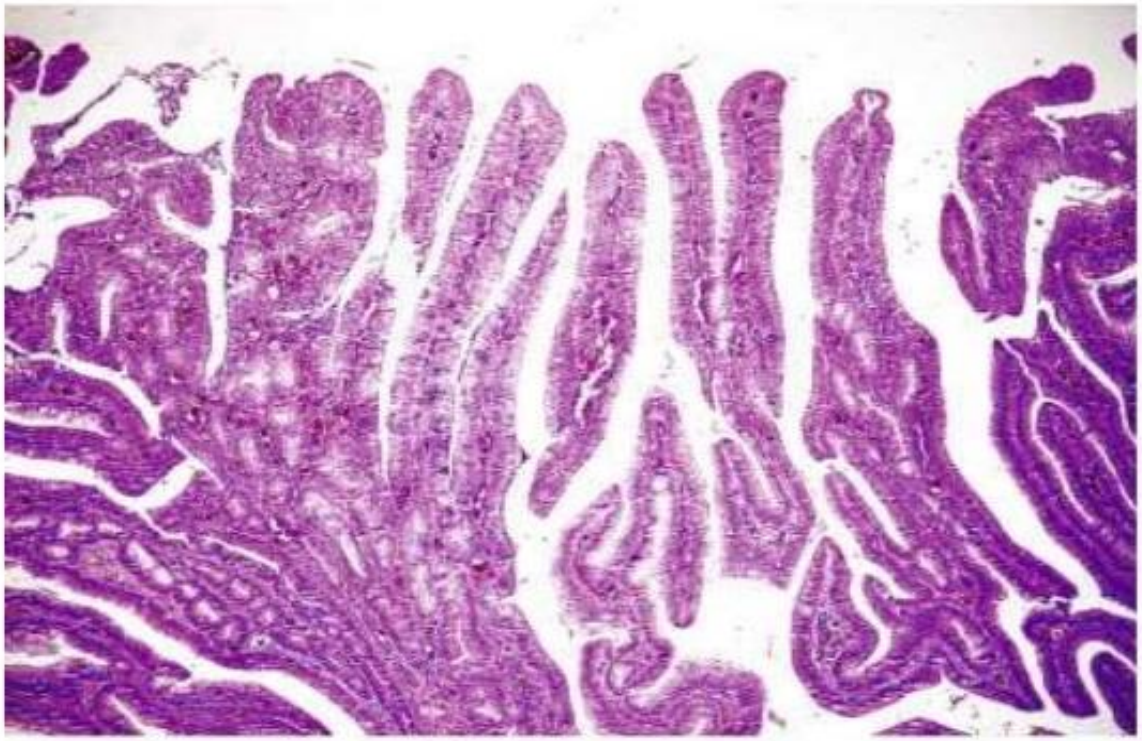




Tubular adenoma.The neoplastic glands form tubules

Villous adenoma :





- **Villous adenoma**: >75% villous component. Long villi are arranged in parallel, perpendicular to the mucosa

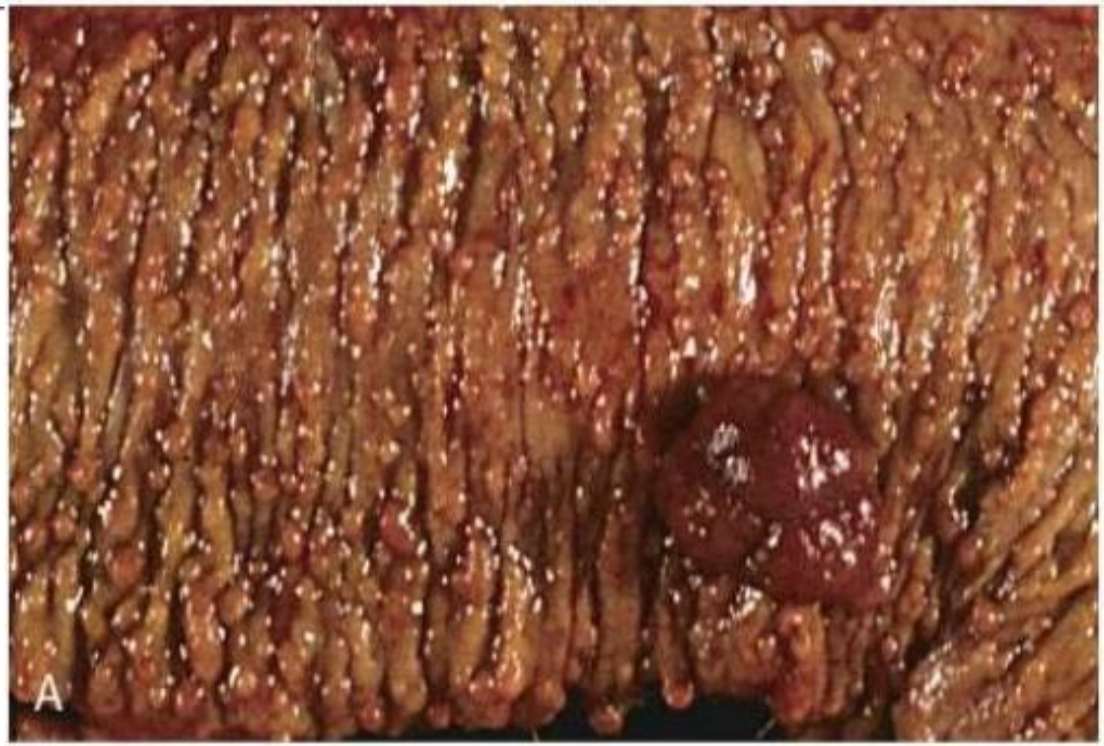
Tubulo-villous adenoma :

Familial adenomatous polyposis (FAP)

- Autosomal dominant disorder.
- Numerous (100- 1000) colorectal adenomas.
- Mutation of the adenomatous polyposis coli (APC) gene.
- In left untreated → colorectal carcinoma (100%) , before age 30.

FAP:

FAP:



- **Hundreds of small polyps are present throughout this colon with a dominant polyp *(right)*.**

Gardener Syndrome :

- intestinal adenomas
- osteomas
- epidermal cyst
- thyroid tumors
- dental abnormalities

Turcots Syndrome :

- intestinal adenomas.
- CNS tumors.(medulloblastoma & glioblastoma)

Adenoma associated with high risk of CRC :

1. Large size , > 1 cm.
2. Villous histology.
3. High grade dysplasia.
4. Number of polyps – 3 or more is a risk factor.

THANK YOU

Dr. Rajul Shah