PATHOLOGY GI PICTURES SHOW COLON-RECTUM –ANUS TUMORS

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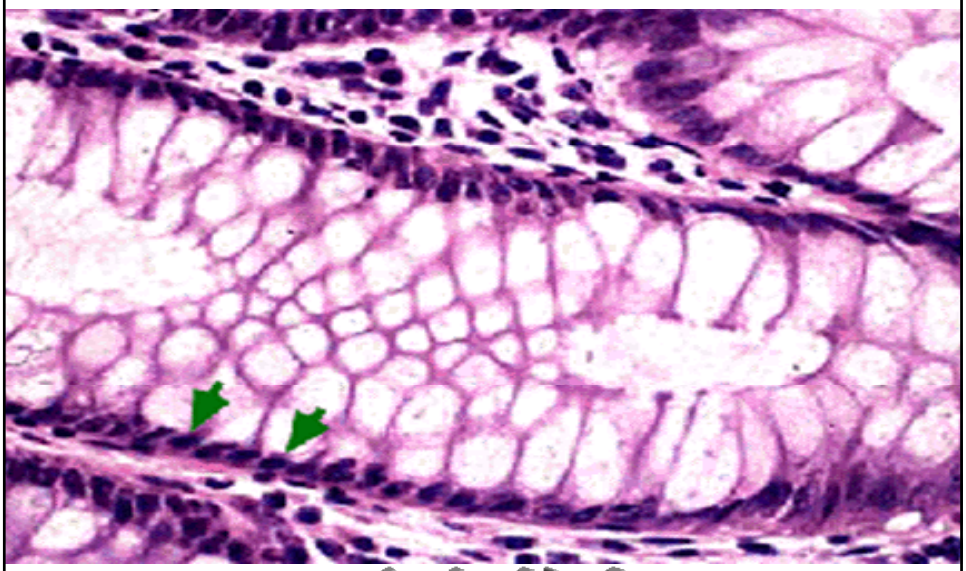
The American Cancer Society has released a public awareness campaign to encourage colon screenings for polyps

Polyp Man is the new villain in public service announcements encouraging colon cancer screening.

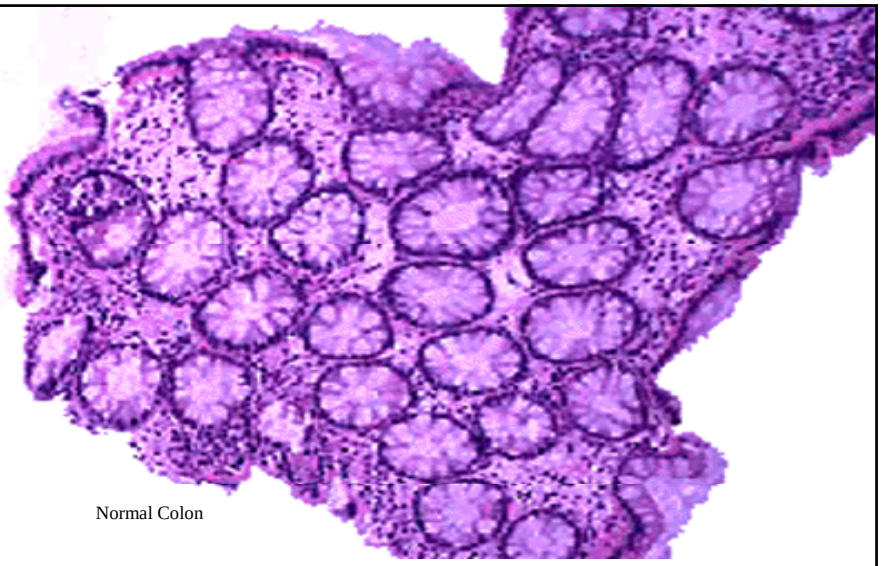
Normal Colon

•High power view of normal colonic crypts shows mucus filled cells with small regular basally placed nuclei

•Note the lack of palisading and absence of nuclear pleomorphism

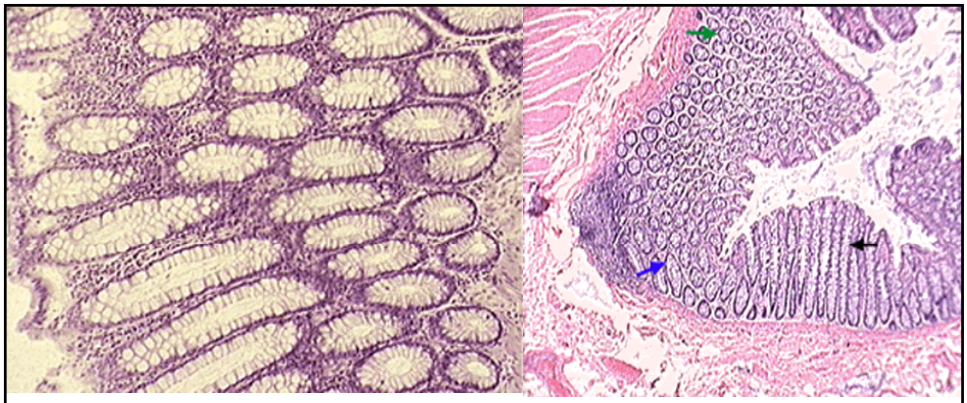


Normal Colonic Mucosa (Colon)	
Etiology	• Not applicable.
Pathogenesis	• Not applicable.,
Epidemiology	• Not applicable.
General Gross Description	• Consists of cecum, ascending colon which is attached to the retroperitoneum; hepatic flexure and transverse colon which are supported by gastrocolic ligament; splenic flexure, descending colon with retroperitoneal attachment; sigmoid colon, and rectum which have an extraperitoneal component. • All show similar mucosal surface • Supplied by superior mesenteric artery to splenic flexure, inferior mesenteric artery to distal rectum • Distal rectum supplied by branches of the internal iliacs • Venous drainage is predominantly via the portal system to the liver although systemic drainage occurs in distal rectum • Lymphatic drainage to regional nodes.
General Microscopic Description	• Epithelium composed of a mixture of columnar absorptive and goblet cells covering the surface and extending into crypts • Occasional lymphocytes, eosinophils, and apoptotic cells found in surface epithelium, as well as endocrine cells and Paneth cells in proximal right colon and cecum • Crypts extend from surface to muscularis mucosae and are surrounded by lamina propria containing plasma cells, T lymphocytes, mast cells, fibroblasts, eosinophils and macrophages • Lymphocytes may be dispersed or arranged in lymphoid follicles that may extend through muscularis mucosae to submucosa. • Lymphatics are found in mucosa just adjacent to the muscularis mucosae. • Submucosa contains Meissner's plexus of ganglion cells and nerve fibers • Inner circular layer and outer longitudinal layer of muscle with Auerbach's plexus between them • Serosa and or pericolic fat



Normal Colon

•Normal colonic crypts when transected have the apearance of gun barrels
•There is little lamina propria between the crypts, and only sparse numbers of chronic inflammatory cells
•Nuclei are basally placed and uniform



Normal colonic mucosa x10

•Note regular spacing of glands which have little lamina propria containing scant mononuclear cells
•Epithelium shows diffuse uniform mucous production with small basally placed regular nuclei

Normal colonic crypts depending on the axis of section can be elongated(black arrow), round gunbarrels(green arrow) or at an angle(blue arrow)
•Note the tight spacing of the normal mucosal crypts with little lamina propria seen
•Crypts extend from the surface to the muscularis mucosa

ADENOCARCINOMA OF THE COLON

ADENOCARCINOMA of the COLON:

- **PATHOGENESIS:**
  - **Classically, step-wise progression to cancer. Multiple genetic mutations are thought to have to occur, before a polyp becomes malignant. Genes involved:**
    - **ras gene: Oncogene that becomes cancerous when a point mutation is introduced.**
    - **p53 gene: Tumor-suppressor gene.**
    - **DCC gene: Tumor suppressor gene; "deleted in colon cancer"**
  - **PREDISPOSING CONDITIONS:**
    - **Inflammatory bowel disease**
    - **Congenital polyposis syndromes**
- **PATHOLOGY:**
  - **Napkin-Ring Tumors: Constricting-type tumors, most common in the left colon. The wall thickens, narrowing the colonic lumen.**
    - **Often present with constipation due to narrowed lumen.**
  - **Cauliflower Tumors: More prevalent in the right colon. Tumors may ulcerate.**
    - **Usually presents with rectal bleeding.**
- **CLINICAL:**
  - **Progression of disease**
    - **EARLY: Asymptomatic. May find occult blood in stool if you test for it.**
    - **MIDDLE: Rectal bleeding and change in bowel habits. Possible palpable mass.**
    - **LATE: Fatigue, abdominal pain, pallor, cachexia, hepatomegaly**

Adenocarcinoma of the colon is a primary cause of mortality and morbidity in North America and Western Europe.

Colonic cancers are the most common GI carcinomas and have the best prognosis.

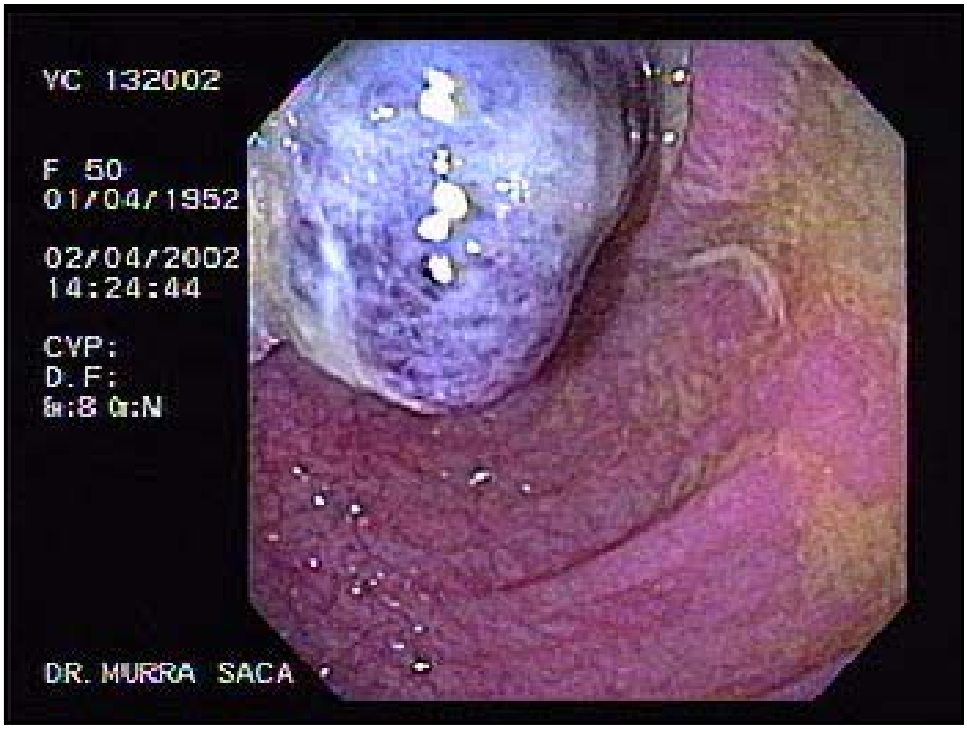
The 5-year survival rate is approximately 50%.

Survival rates may be improved by screening and removal of adenomatous polyps.

Almost all colonic cancers are primary adenocarcinomas

factors increase the risk for colonic cancer.

- •High-fat low-fiber diet
- •Patient age older than 50 years
- •Personal history of colorectal adenoma or carcinoma (3-fold risk)
- •First-degree relative with colorectal cancer (3-fold risk)
- •Familial polyposis coli, Gardner syndrome, and Turcot syndrome (all patients develop colorectal carcinoma unless they undergo a colectomy)
- •Juvenile polyposis syndrome, Peutz-Jeghers syndrome, and Muir syndrome (risk increased slightly)
- •Hereditary nonpolyposis colorectal cancer (as many as 50% of patients are affected)
- •Inflammatory bowel disease
  - •Ulcerative colitis (risk is 30% after 25 y)
  - •Crohn disease (4- to 10-fold risk)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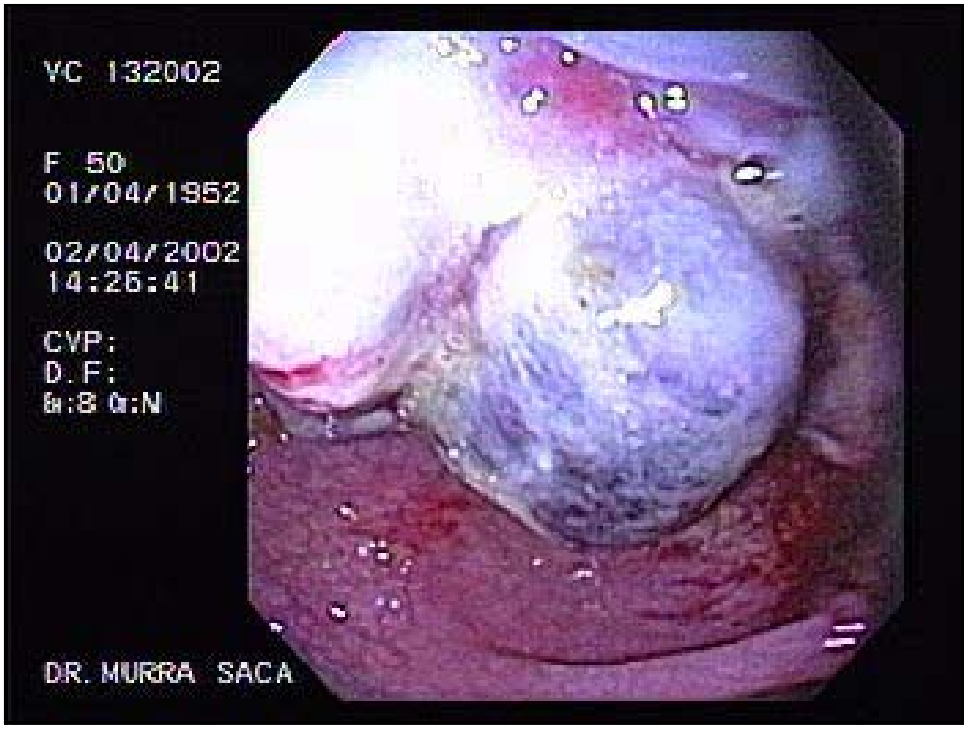
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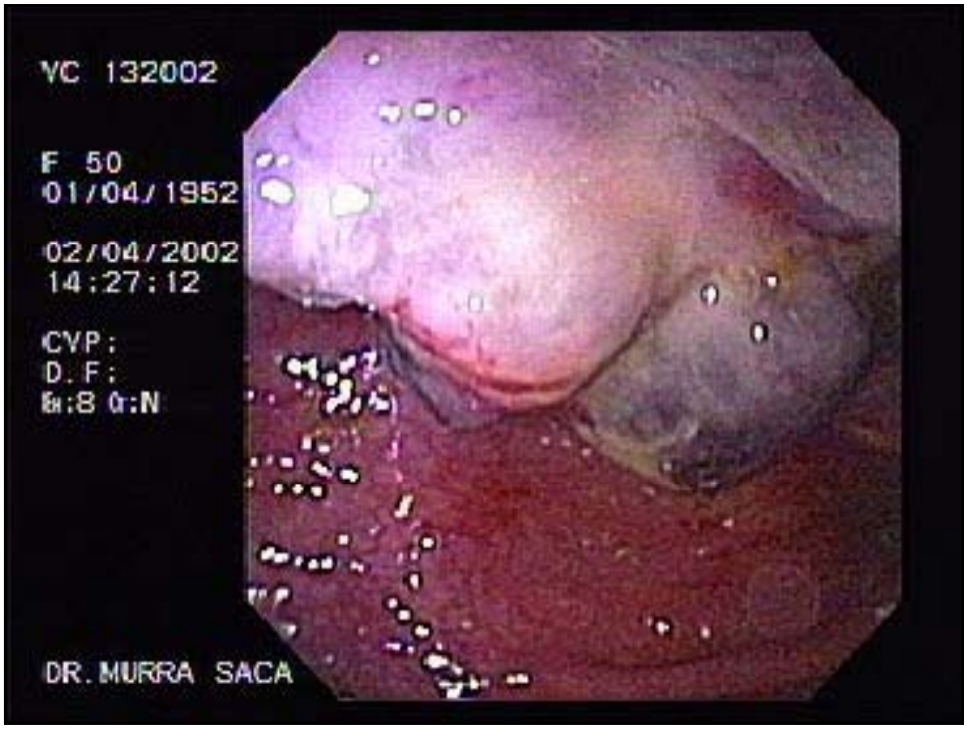


Types of colon cancer:

adenocarcinoma of the colon –
the most common type of colon cancer.
Adenocarcinoma of the colon and rectum develop in the glands of the inner lining or mucosa of the intestine and comprise 95% of colorectal cancer.

Subtypes of adenocarcinoma of the colon are:
mucinous (colloid)
signet ring

Other types include:

neuroendocrine
lymphomas
melanomas
squamous cell
sarcomas
carcinoids

polyps - common tumors found in about half the population over 40 years of age. These mushroom-like growths are usually benign, but at least one type, *adenomatous polyps*, may be a precursor to cancer. About 90% of colon cancers are thought to arise from these polyps.

•**Stages of colon cancer:**
Stage I (Dukes' A) - cancer is limited to the lining or muscular wall of the colon and has not spread anywhere else.

Stage II (Dukes' B) - cancer has spread through muscular wall of the colon or has extended into adjacent organs through the intestine but has not entered the lymph nodes.

Stage III (Dukes' C) - cancer has spread outside the colon into lymph nodes nearby.

Stage IV - cancer has spread beyond colon to distant organs (such as the liver, lungs, or bones)

Recurrent - cancer has recurred after it has been treated. It may recur in or on the colon (e.g., in the suture line) or in another part of the body (metastatic colon cancer).

Metastatic Colon Cancer:

Colon cancer with bone metastases

Colon cancer with brain metastases

Colon cancer with liver metastases

Colon cancer with lung metastases

Colon cancer with liver and lung metastases

Colon cancer with skin metastases

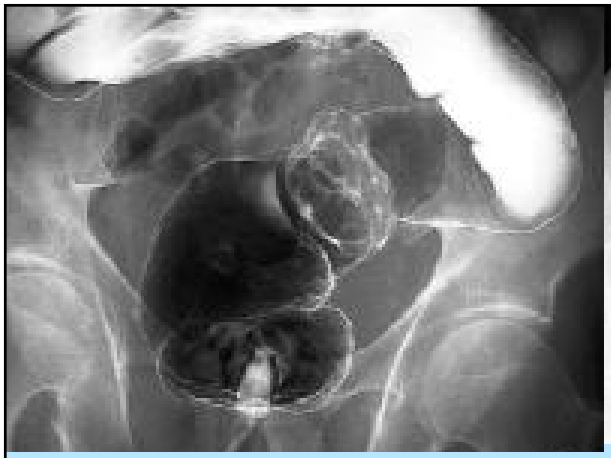
Colon cancer metastasized to other sites such as the ovaries, buttocks, etc.

Clinical Presentation

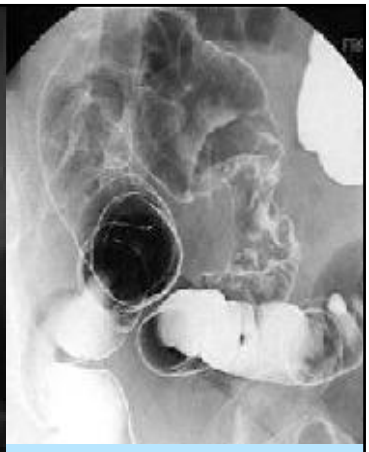
- •May be asymptomatic
- •Microcytic anemia (fatigue, shortness of breath, angina)
- •Vague abdominal discomfort
- •Change in bowel habit
- •Palpable mass
- •Rectal bleeding (overt or occult)
- •Large bowel obstruction
- •Perforation (rare)
- •Jaundice
- •Ascites

Preferred Examination:

- •Begin the evaluation with a history and physical examination, including a digital rectal examination.
- •Inspect the stool and test for occult blood.
- •Perform blood tests, including a full blood count, liver function tests, and carcinoembryonic antigen level.
- •Perform either a sigmoidoscopy (rigid or flexible) and a double-contrast barium enema or a colonoscopy

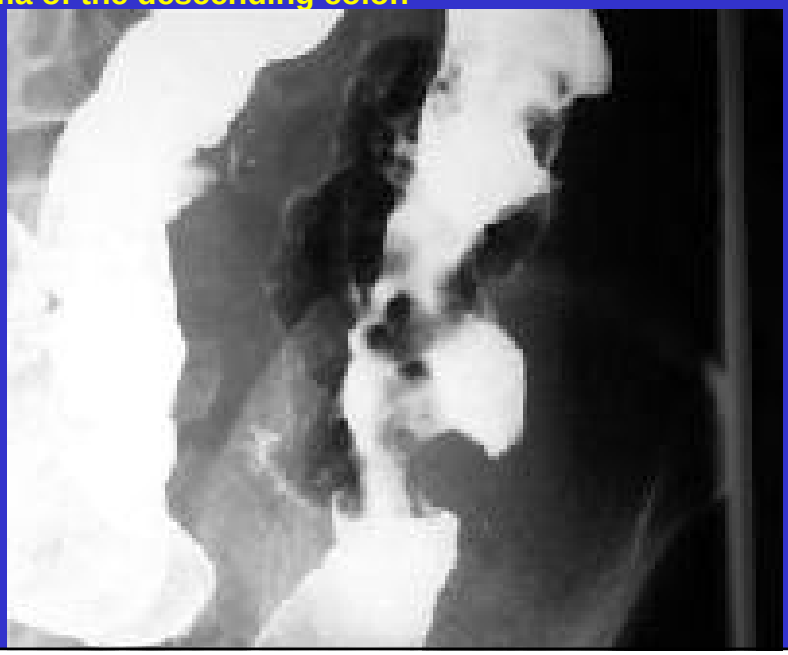


Polypoid carcinoma. A large, irregular lobulated mass is present in the rectosigmoid junction.



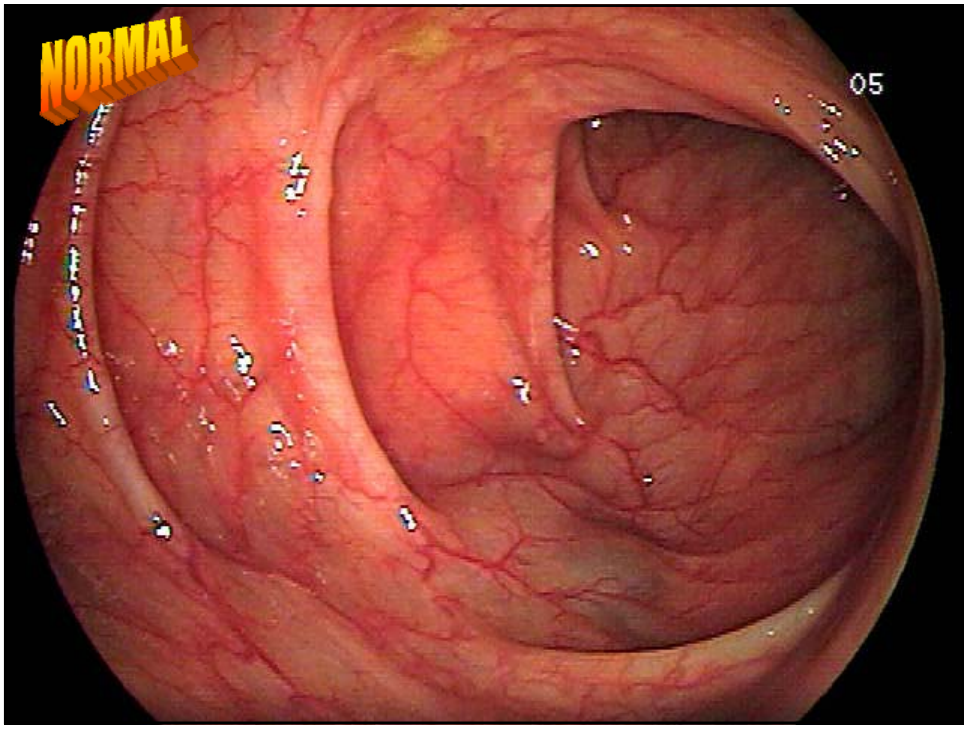
Annular carcinoma of the sigmoid colon. The lumen of the sigmoid is narrowed severely by the circumferential mass with mucosal destruction and the overhanging edges or shouldering at the tumor margins

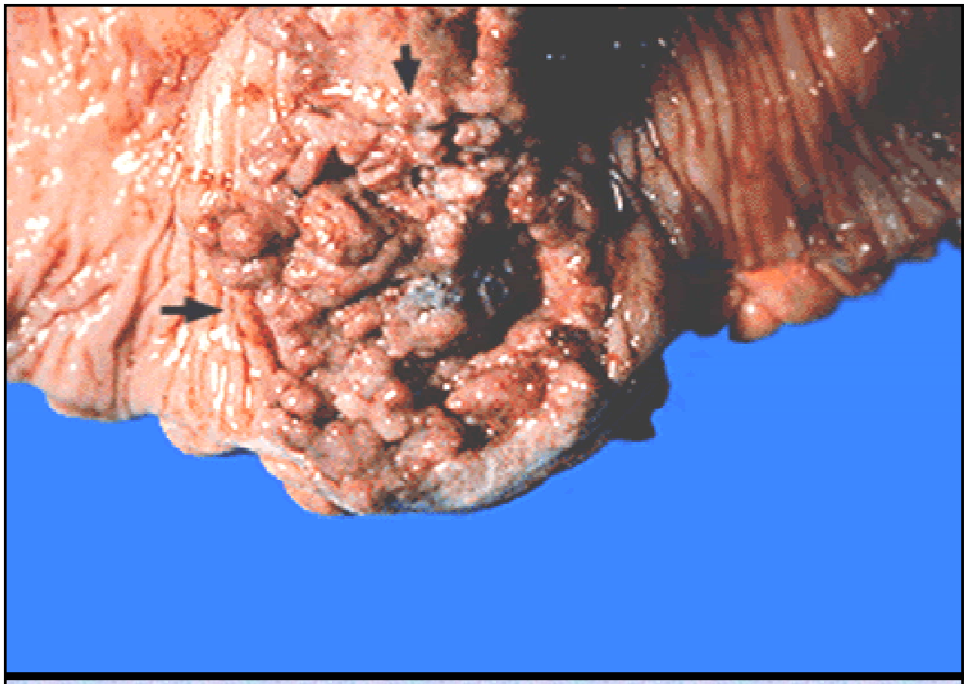
Local perforation and paracolic collection in an annular carcinoma of the descending colon



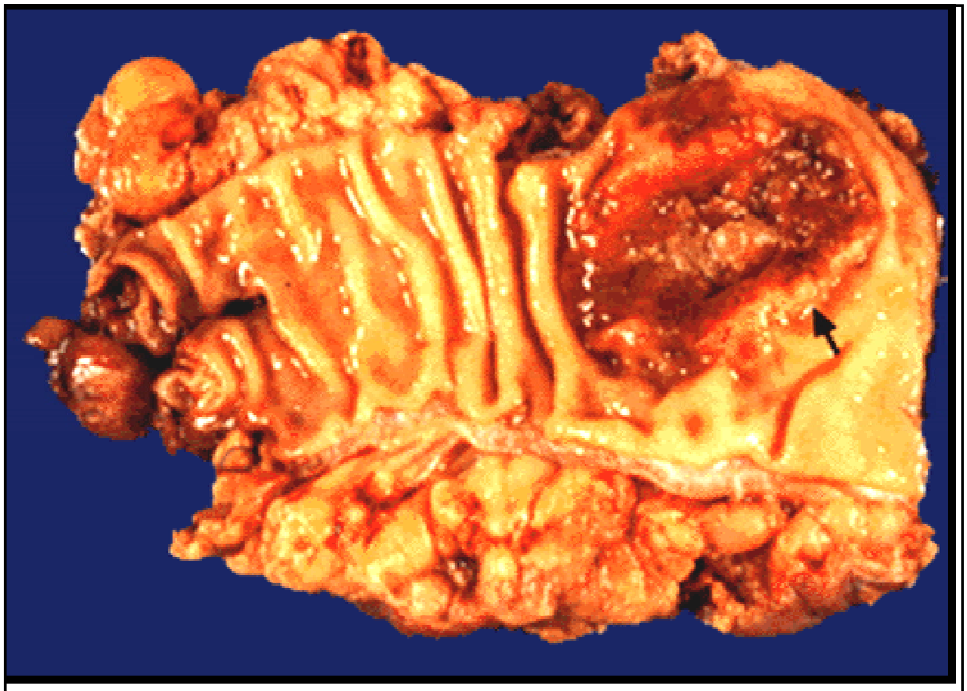
Colon carcinoma

	Right colon 30%	Left colon 70%
type	Polypoid Fungating mass	Annular ring Apple core lesion Napkin ring configuration circumferential growth
symptoms	Bleeding Melena producing iron deficiency anemia Malaise weight loss	Obstruction Constipation Thin caliber stool Sometimes bleeding
spread	Nodes Liver 50%>lung>bone	Nodes Liver 50%> lungs>bone

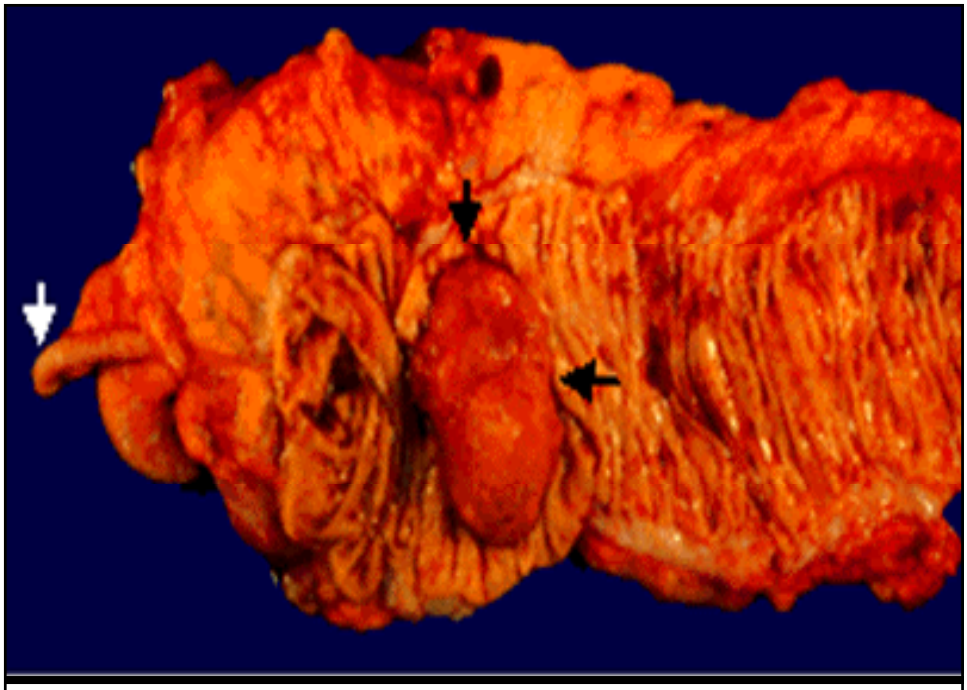




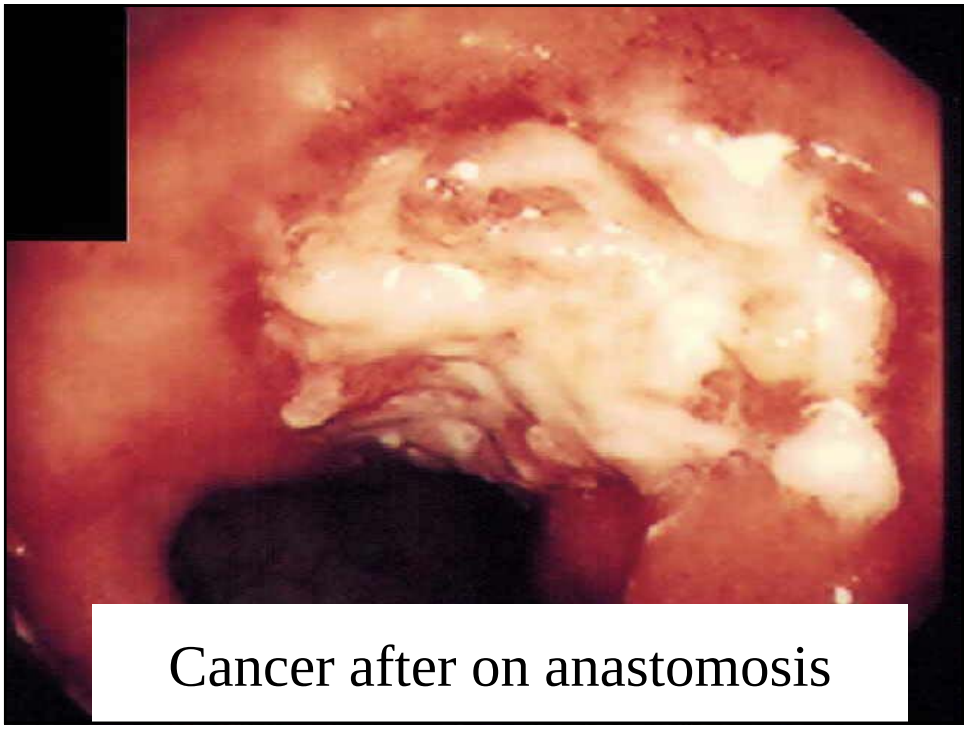
Adenocarcinoma colon



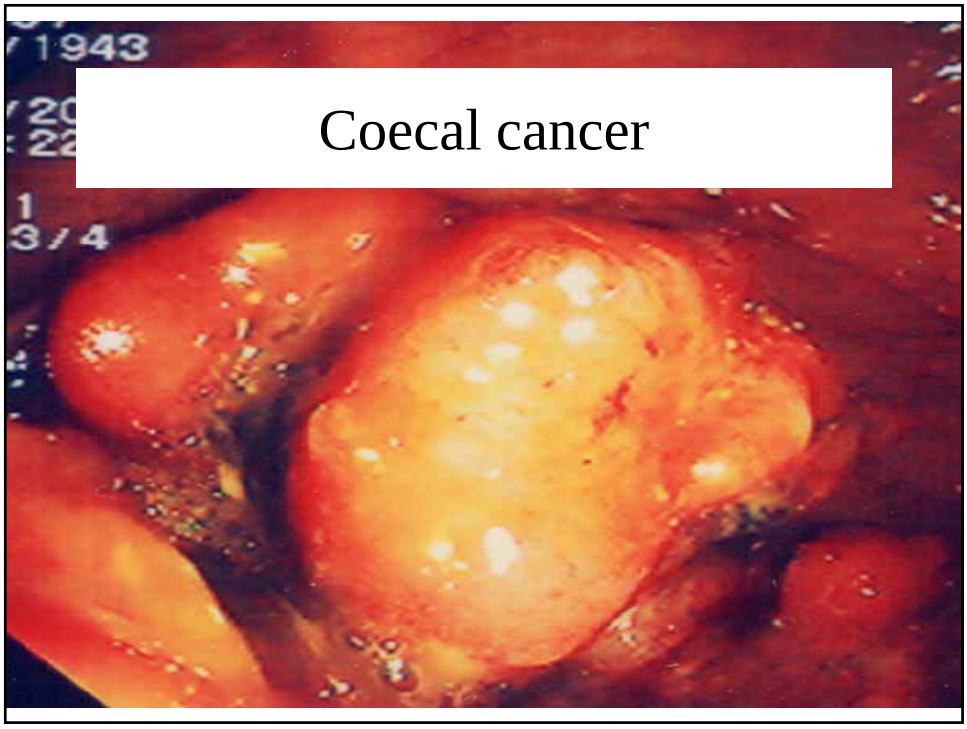
Adenocarcinoma colon



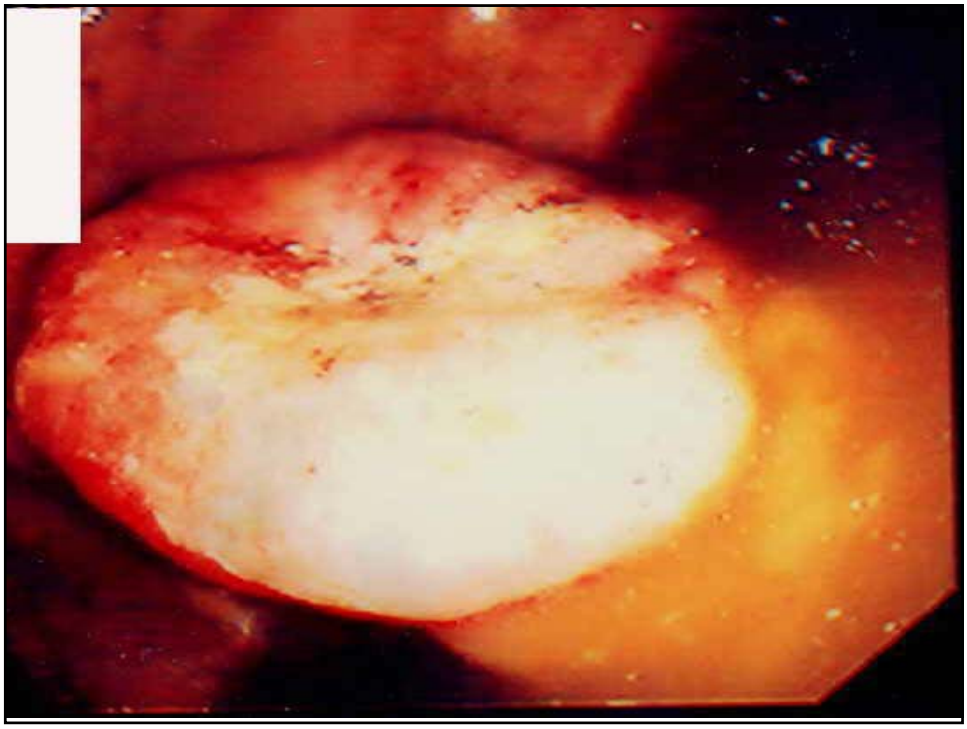
adenocarcinoma



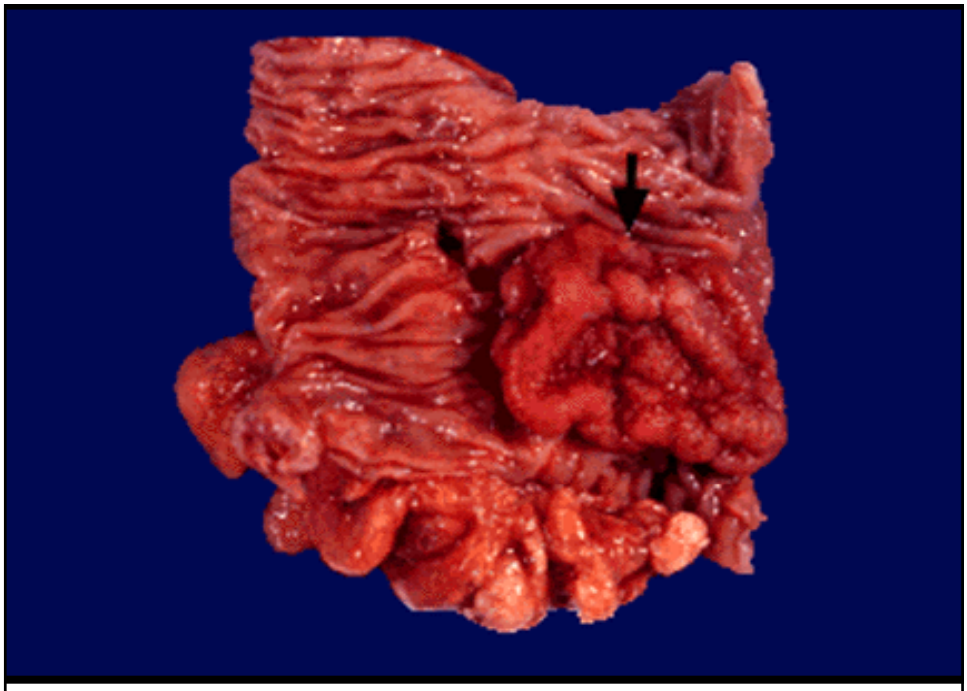
Cancer after on anastomosis



Coecal cancer



Adenomatosis coli



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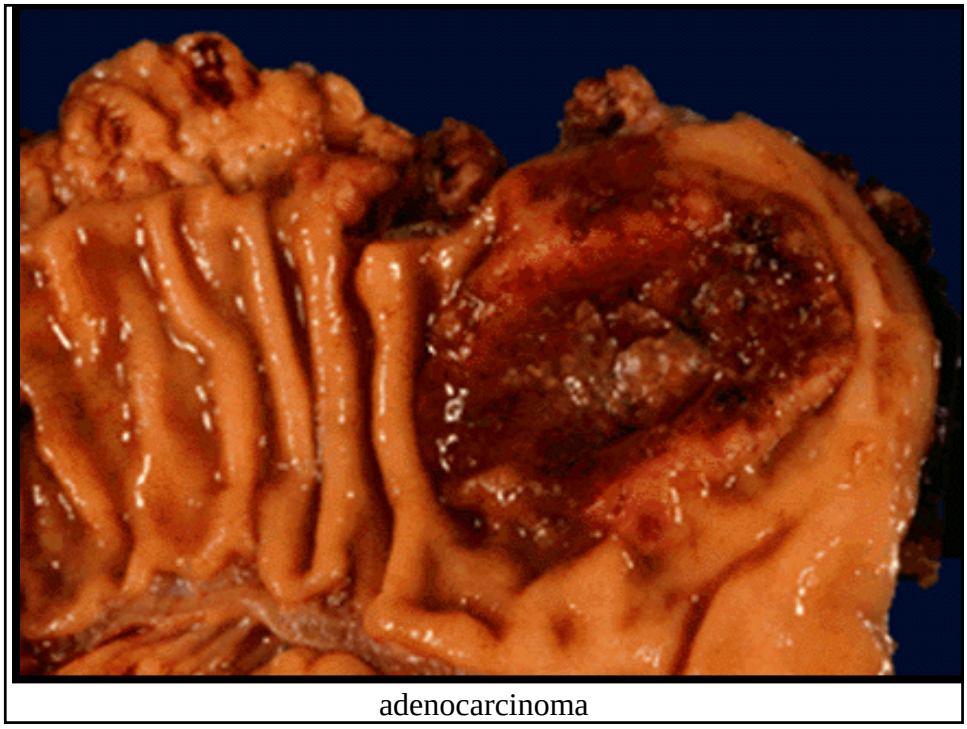

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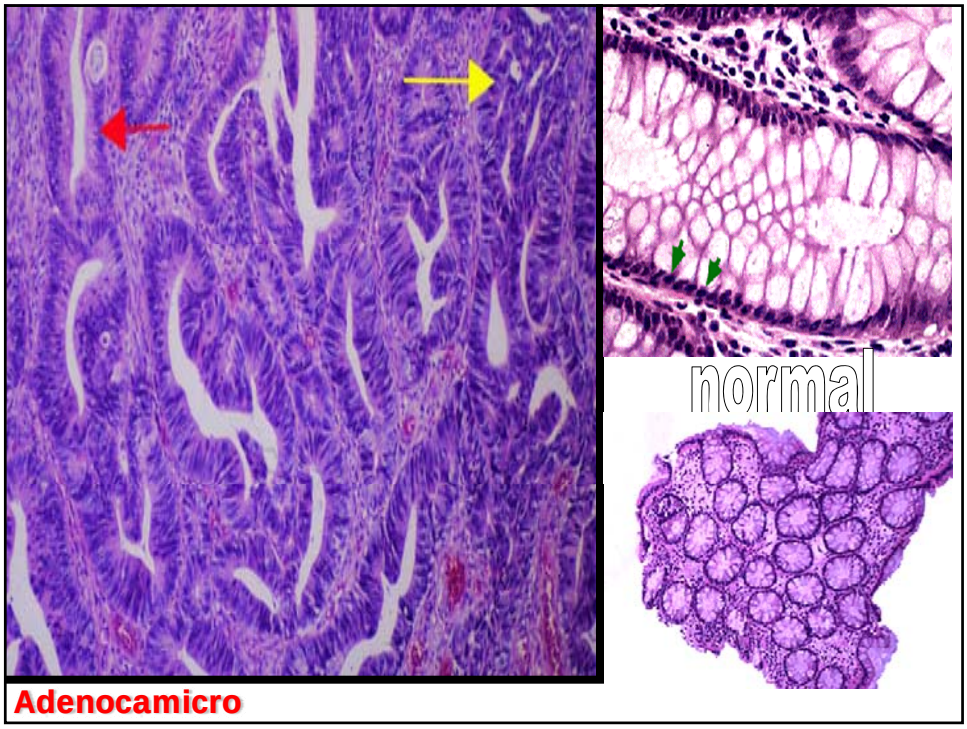

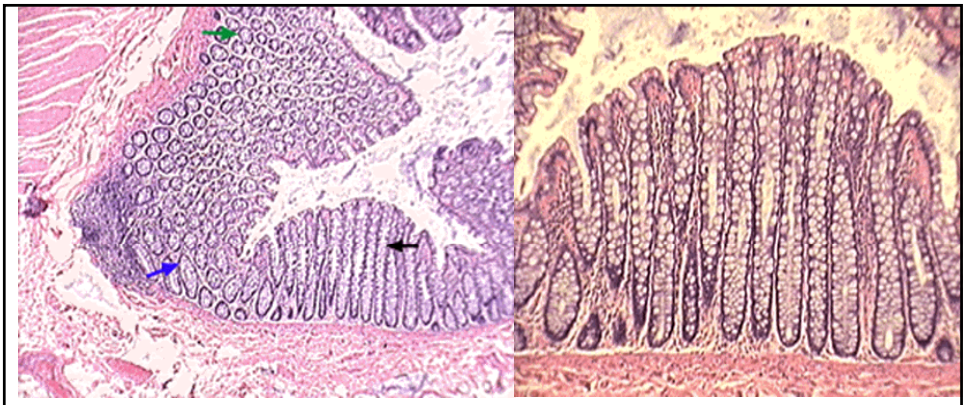

Normal colonic Mucosa

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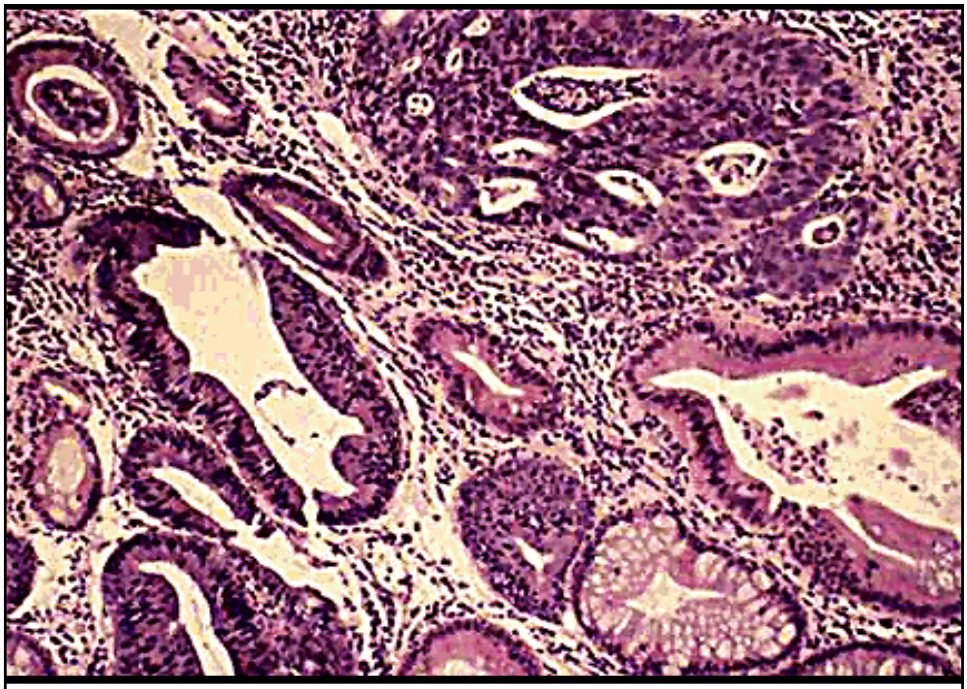
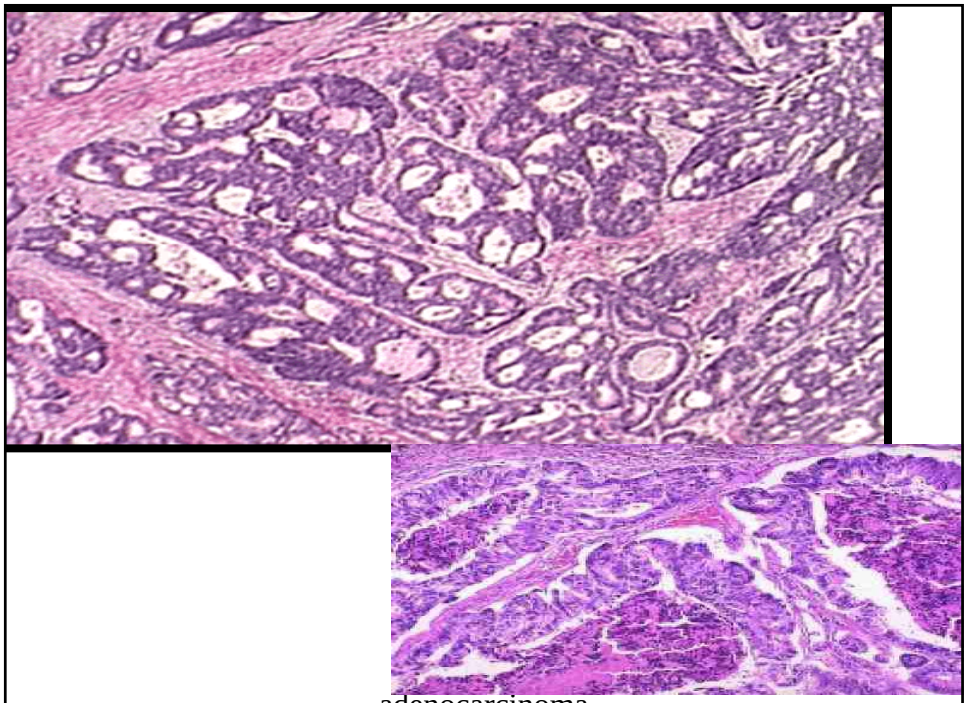
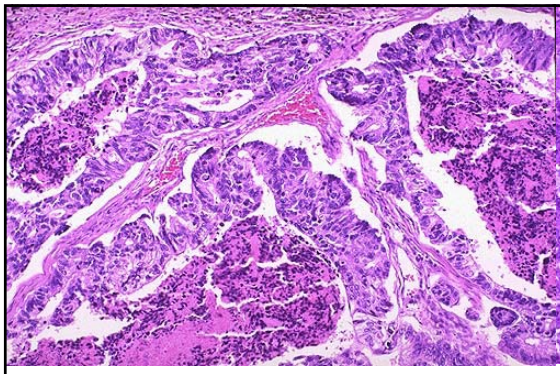
- Note the tight spacing of the normal mucosal crypts with little lamina propria seen
- Crypts extend from the surface to the muscularis mucosa



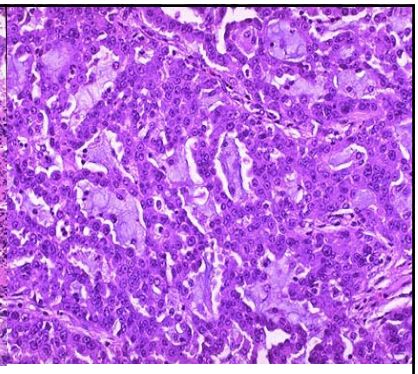
Adenocarcarcinoma



adenocarcinoma

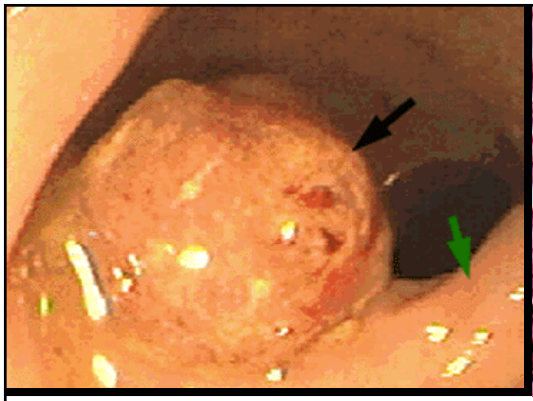


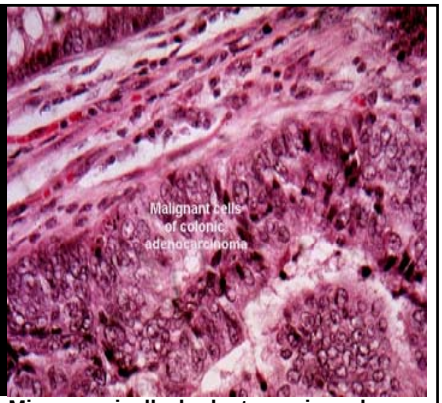
Here is an adenocarcinoma in which the glands are much larger and filled with necrotic debris.



a moderately differentiated adenocarcinoma of colon is seen here. There is still a glandular configuration, but the glands are irregular and very crowded. Many of them have lumens containing bluish mucin.

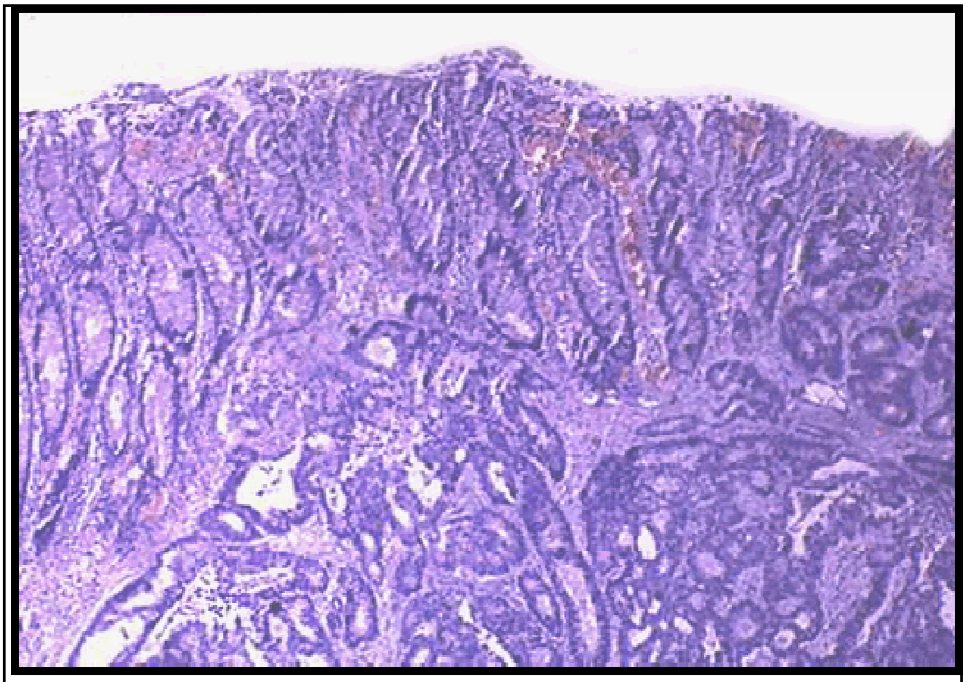
adenocarcarcinoma





Microscopically, look at a region where you can see both malignant and benign elements in the same field. The difference in gland and cellular morphology will be much more evident. The malignant glands show "gland within gland" pattern as well as disorganized arborization and branching. The usual cellular features of malignancy are here in abundance: nuclear-cytoplasmic ratio, hyperchromasia, angulated nuclear margins etc.

adenocarcarcinoma



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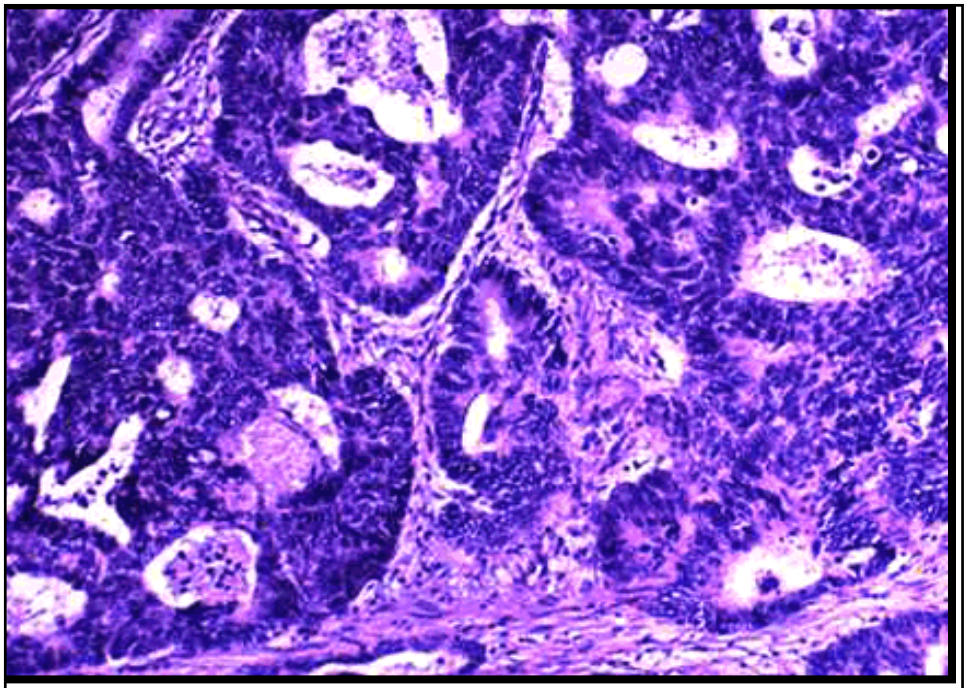

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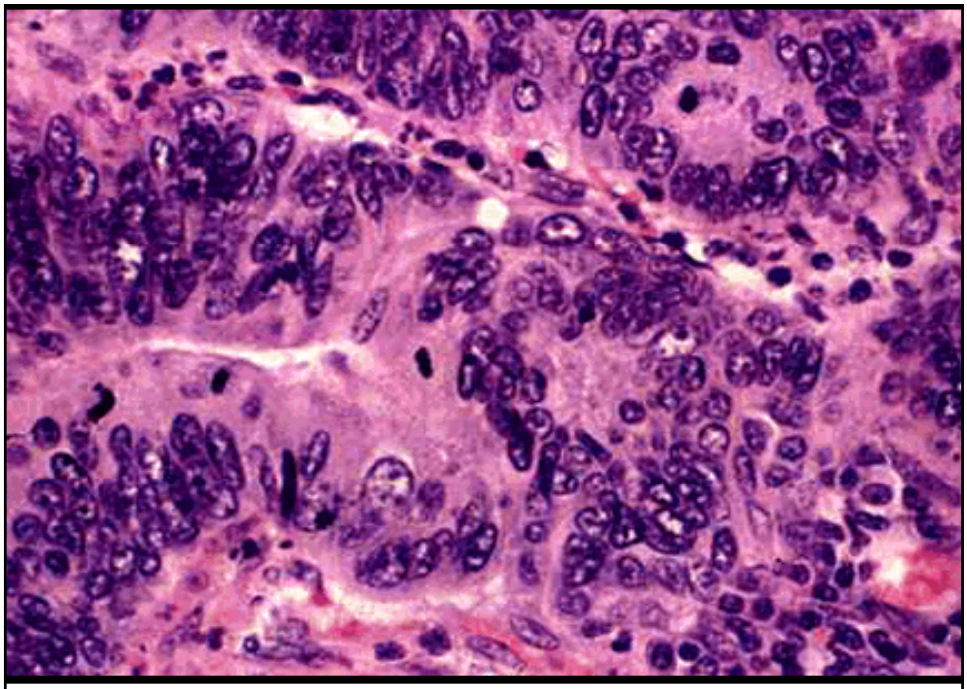

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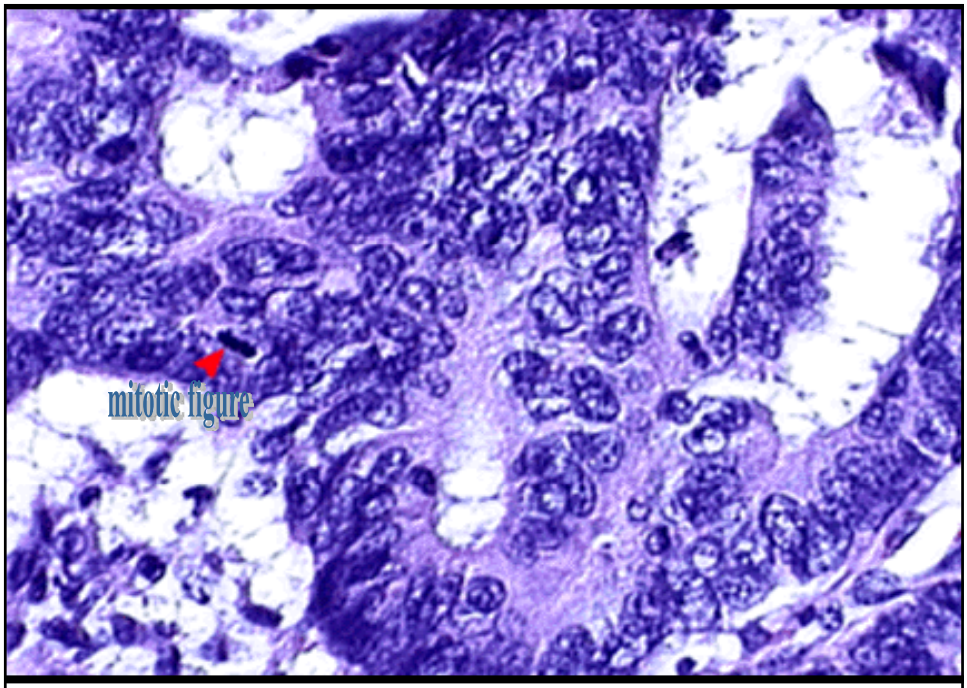

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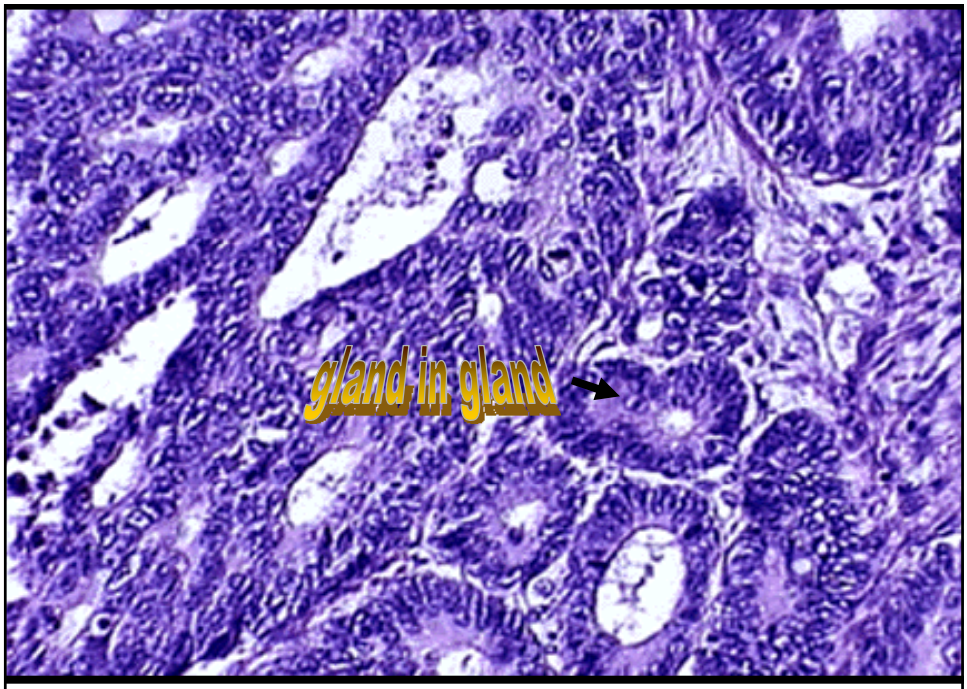

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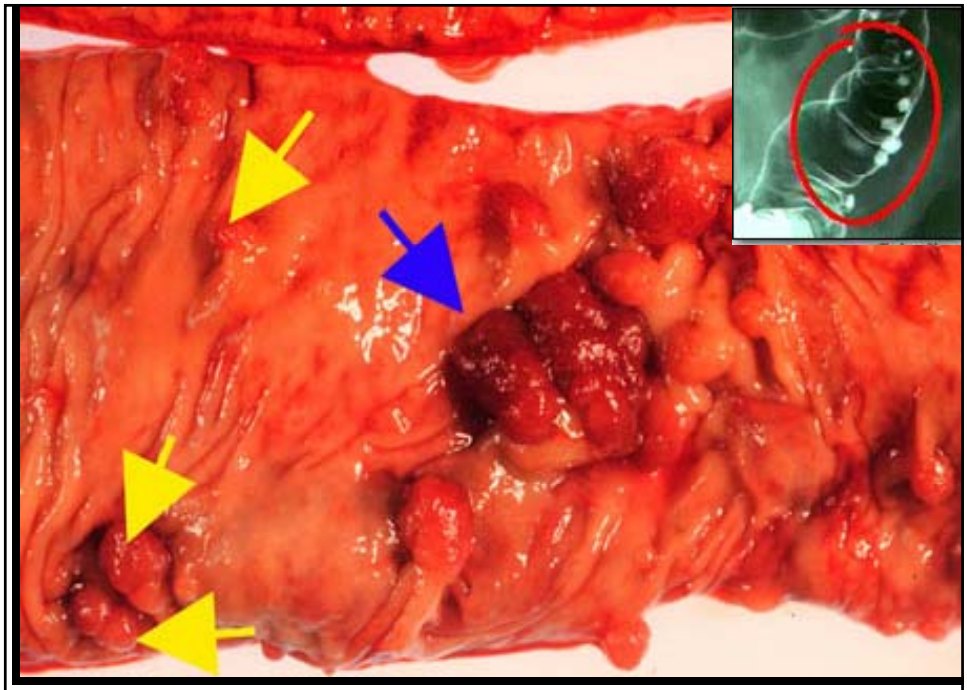

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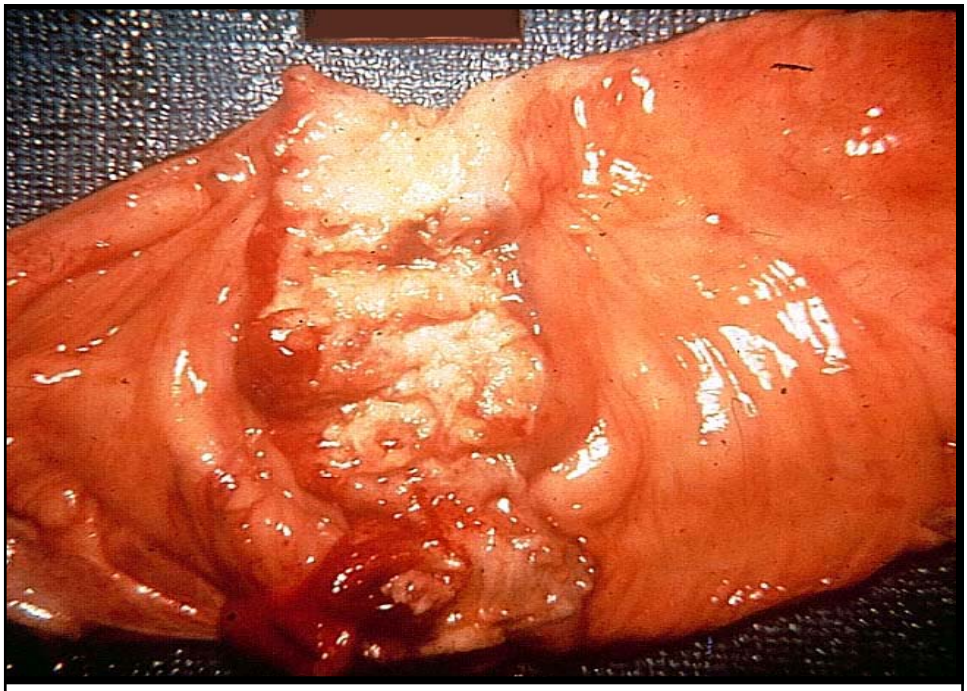

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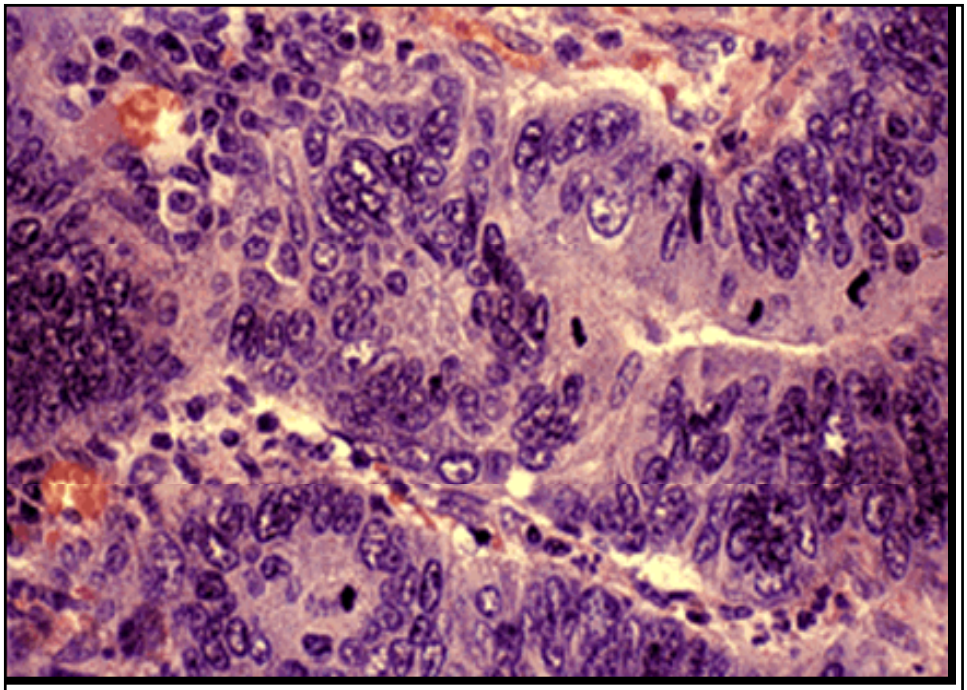

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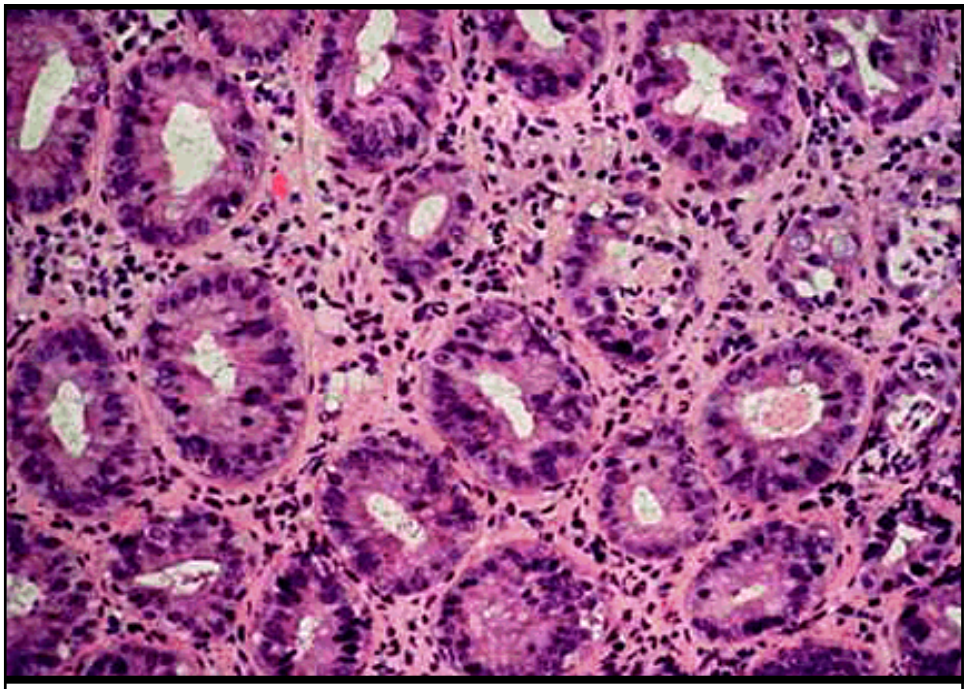

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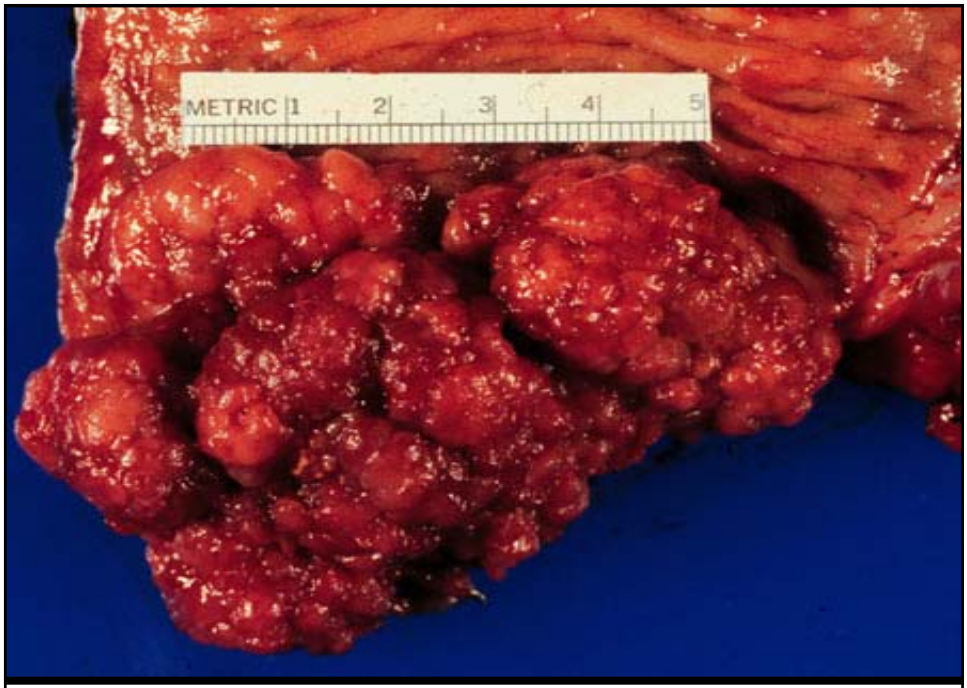

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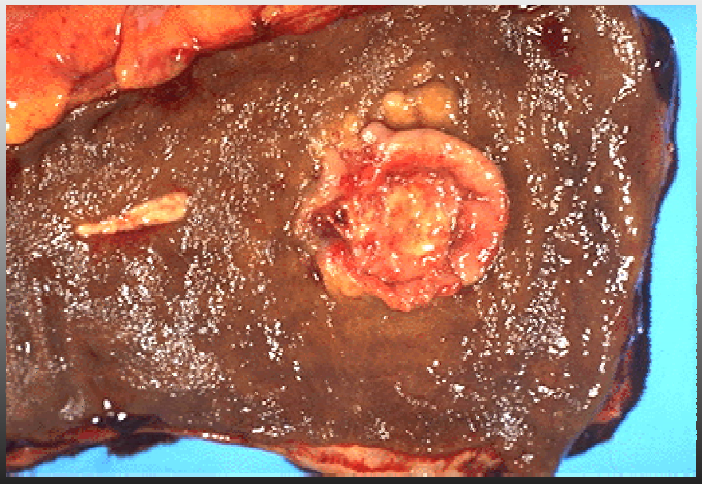
COLONIC ADENOKC



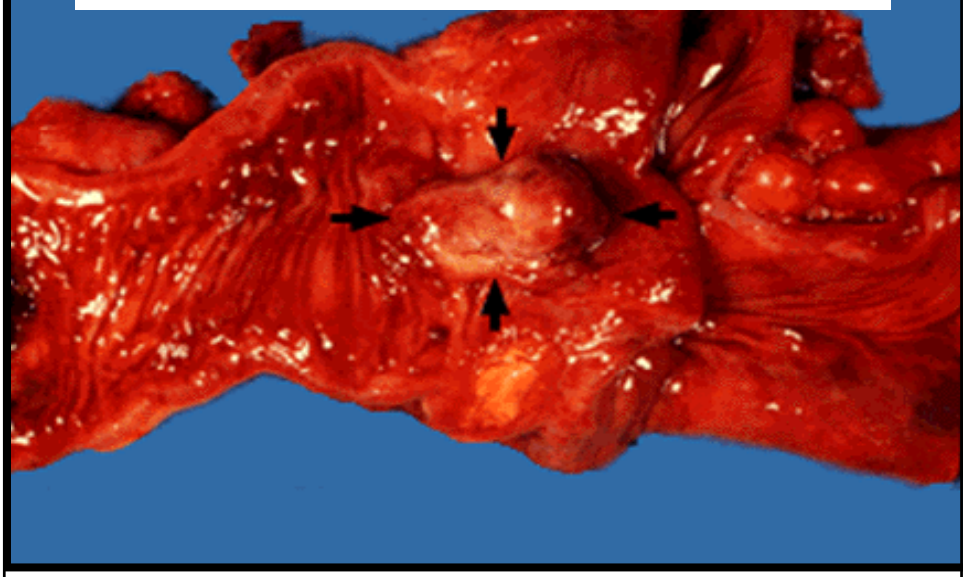
Carcinoid

- 15% rectum
- 5-7% colon

Carcinoid Tumor (Colon)	
Etiology	•The etiology of intestinal carcinoid tumors is unknown. •Carcinoid tumors arise from neuroendocrine cells of gut mucosa, but it uncertain whether differentiated or immature neuroendocrine cells are the origin. •Carcinoids arise from Neuroendocrine cells of endodermal derivation.
Pathogenesis	•The pathogenesis of intestinal carcinoids is unknown. •While the vast majority of carcinoids arise in the GI tract, they can be found wherever endodermal derived tissue is seen, such as teratomas most commonly in the Ovary,,
Epidemiology	•The peak incidence of carcinoids is the sixth decade. •No other sex, racial or geographic preference has been described.
General Gross Description	•While carcinoids are found the entire length of the GI tract, 95% arise in the Appendix, Ileum and Rectum. •These tumors are fairly well circumscribed but not encapsulated, white or pale yellow tan, and very firm due to a marked desmoplastic reaction. •When found serendipitously, 75-80% of Appendiceal and Rectal Carcinoids are less than 1cm, while over half of Ileal Carcinoids are over 1.5cm. •The overlying mucosa is usually intact, and the tumor is seen as a submucosal smooth nodule. •Large lesions can ulcerate the mucosa or penetrate the serosa. •Virtually all symptomatic carcinoids have metastasized when diagnosed, and these lesions gnerally over 2cm in size.
General Microscopic Description	•The nomenclature of Carcinoid tumors is in flux, with some restricting the term to those tumors showing the classical histologic patterns described for Carcinoids, while other prefer to use the term neuroendocrine tumor for all tumors showing a predominant neuroendocrine expressiopn. •While carcinoid tumors have many different histologic appearances, the main patterns are: solid, often with peripheral palisading; trabecular, forming ribbon-like strands; acinar or gland-like; and insular, cribiform arrangement similar to islets. •Individual tumor cells have well defined cell borders, centrally placed small nuclei with a finely stippled chromatin pattern, small or abscent nucleoli and moderate amount of cytoplasm varying from acidophilic to basophilic, •The most striking feature is the bland monotous uniform appearance of the tumor without pleomorphism or mitotic activity. •Confirmation of the neuroendocrine nature of Carcinoid tumors was their positive argyrophilic (silver) staining, with the Grimelius Stain being the most usefull. •The argyrophilic positive neurosecretory granules can also be demonstrated ultrastructurally, but these techniques have generally been replaced by immunoperoxidase identification of the neurosecretory granules using either a general marker such as chromogranin, or specific hormone markers such as gastrin, somatostatin, or serotonin,. •As with other endocrine tumors, prediction of aggressive biologic behavior is not possible on histologic grounds with the only absolute critieria being the presence of metastases.

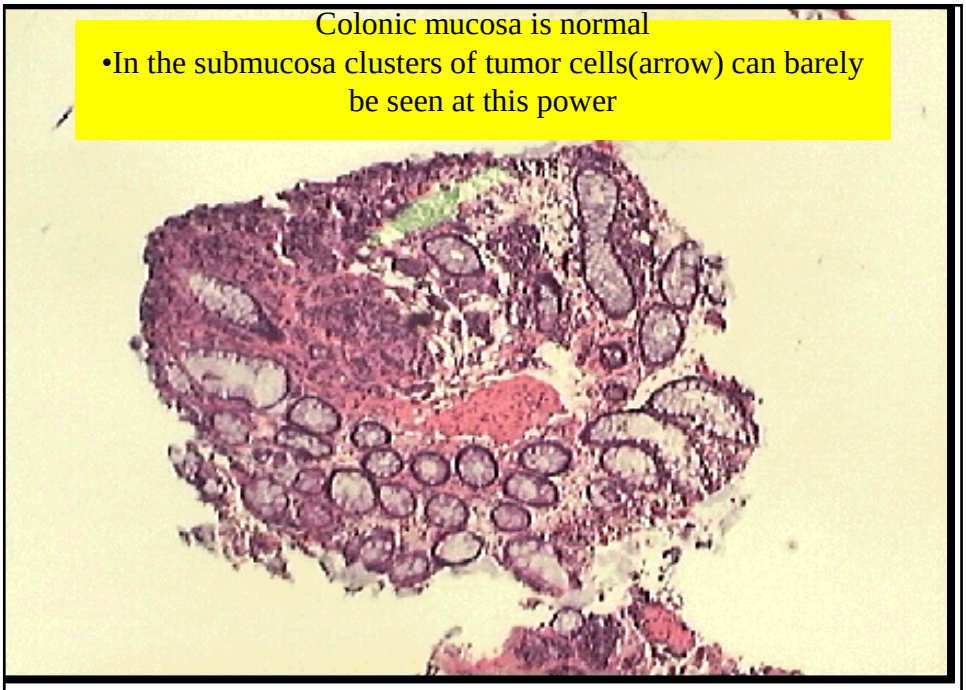
Clinical Correlation	•Carcinoids are slow growing low grade malignant tumors most of which do not metastasize and are asymptomatic at death. •Appendiceal and rectal carcinoids are virtually never metastasize, while the great majority of metastasizing carcinoids arise in the Ileum, usually distal; Ileum. •The most common symptom is non-specific abdominal pain, which has usually been present for a number of years. Bleeding is rare, and obstruction can occur due to size of the tumor, desmoplastic thickening of the intestinal wall, intestinal infarction secondary to mesenteric vessel compression by tumor, or intussusception. •Metastases first occur in regional nodes and then the liver. •When first diagnosed, only 25% of patients with Ileal carcinoids will have disease limited to the intestine, and 25% will already have liver metastases. •These tumors are slow growing so that even with demonstrated liver metastases, 5 year survival is over 50%. •The most striking presentation of these tumors is the Carcinoid Syndrome. Less than 1% of carcinoid tumors will demonstrate this, and almost all are primary in the Ileum. •The presence of the Carcinoid Syndrome indicates release of polypeptide hormones or vasoactive amines into the general circulation and in most cases denotes bulky hepatic metastases. •The two most common symptoms of the Carcinoid Syndrome are flushing and diarrhea, and these are almost always associated with an elevated 5-HIAA, although the amines responsible for the symptoms are not clearly identified. •Carcinoid tumors in other locations may secrete a variety of hormone products such as Gastrin, Insulin, ACTH, Somatostatin, VIP, etc.

This carcinoid tumor is fairly large for this type of tumor
•Note that the mucosa has remained intact in spite of the size of the tumor



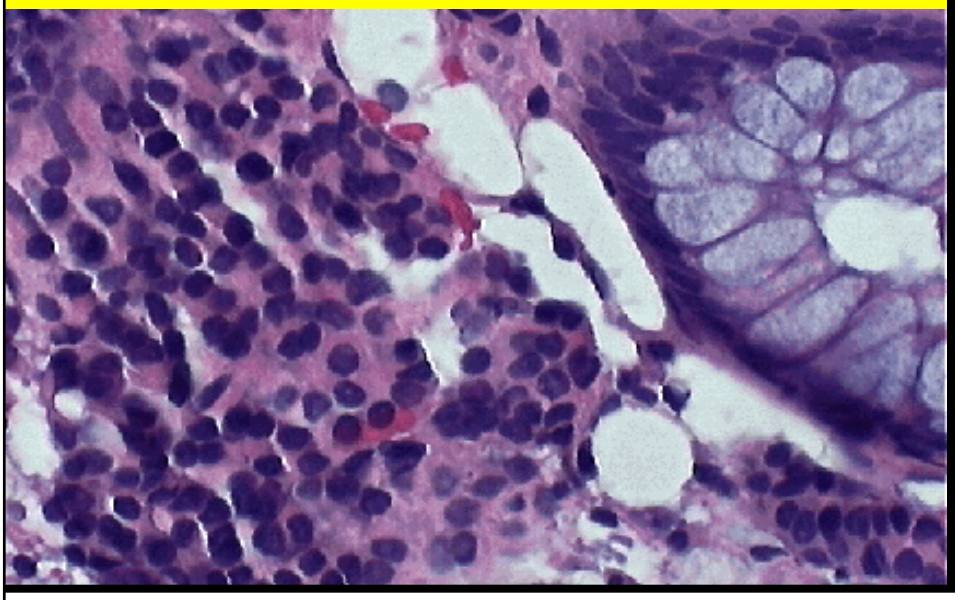
carcinoid1

Colonic mucosa is normal
•In the submucosa clusters of tumor cells(arrow) can barely be seen at this power

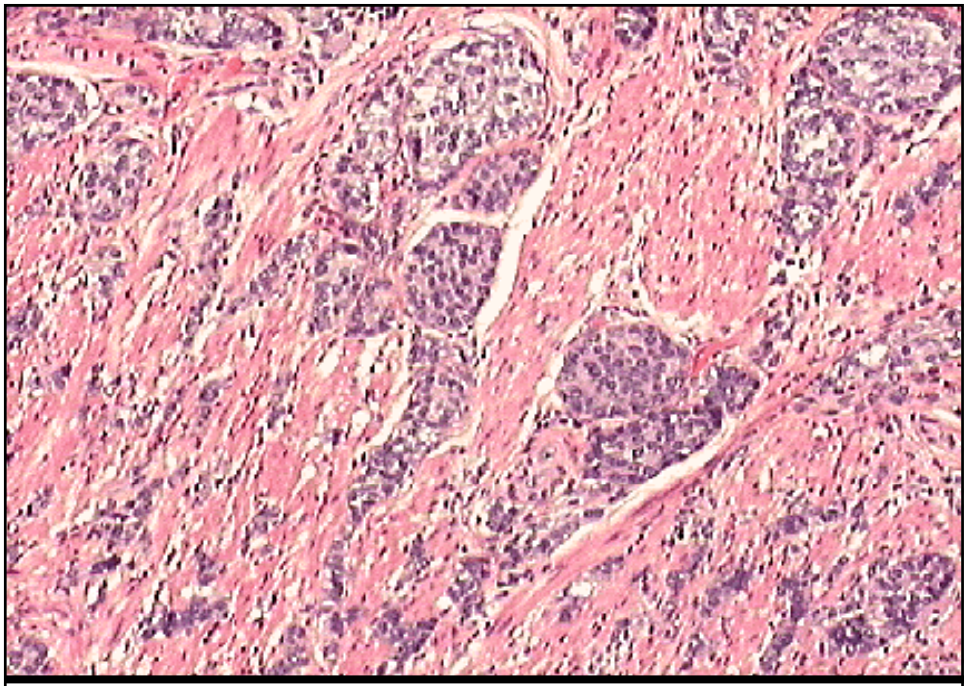


carcinoid2

High power view contains normal colonic mucosa on the right, and clusters of tumor cells on the left •The tumor cells have a very uniform appearance and occur in small clusters

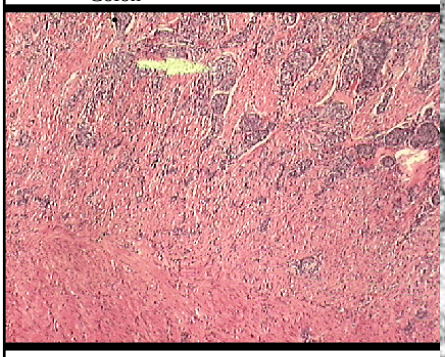


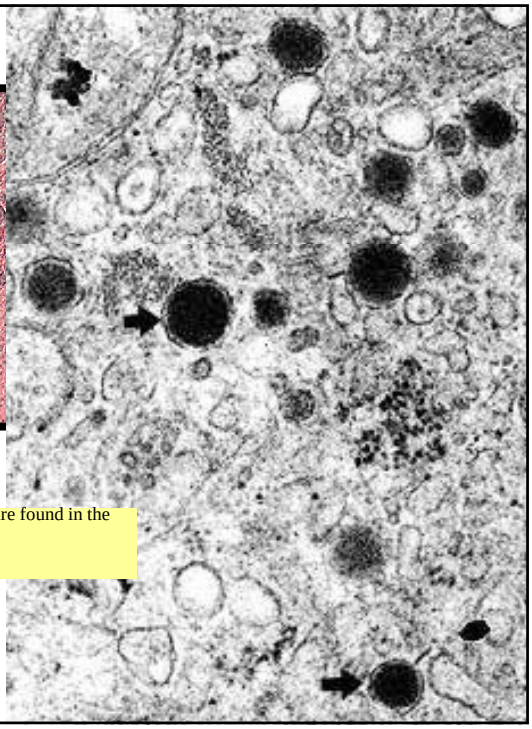
carcinoid3



carcinoid4

Carcinoid, Colon
Carcinoid Tumor of Colon

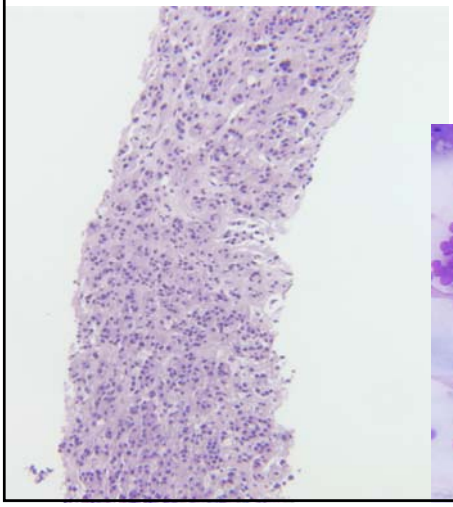


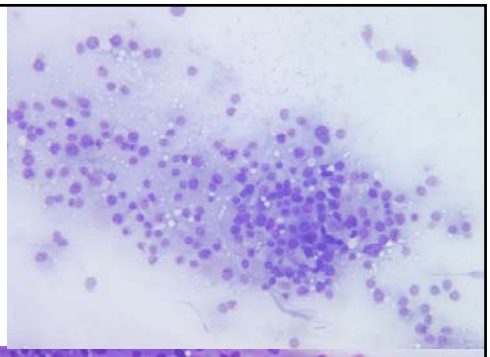


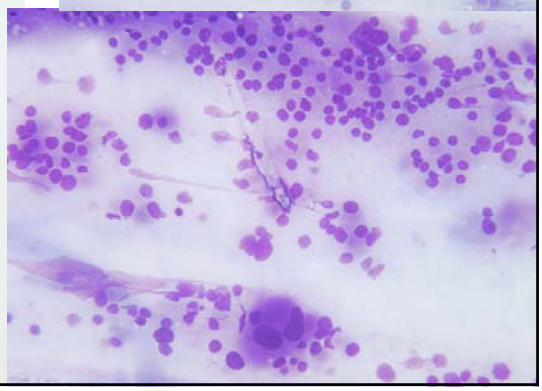
By EM, many dense-cored, membrane-bound granules are found in the cytoplasm (arrows).

carcinoid6

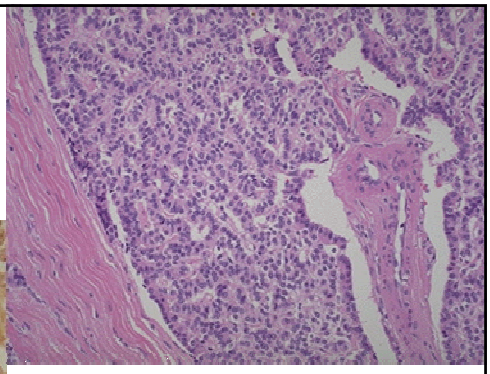
CARCINOID:The tumor has an organoid growth pattern with uniform cytologic features consisting of moderate eosinophilic cytoplasm, finely granular cytoplasm, and round nuclei with finely stippled chromatin. The histologic patterns include spindle cells (mostly peripheral), trabecular, palisading, glandular, follicular, rosette-like, papillary and sclerosing papillary. The cells of pulmonary carcinoid may have oncocytic, acinic-cell-like, signet-ring, mucin-producing, or melanocytic features. Stromal deposition of amyloid, or ossification, or calcifications may be noted.

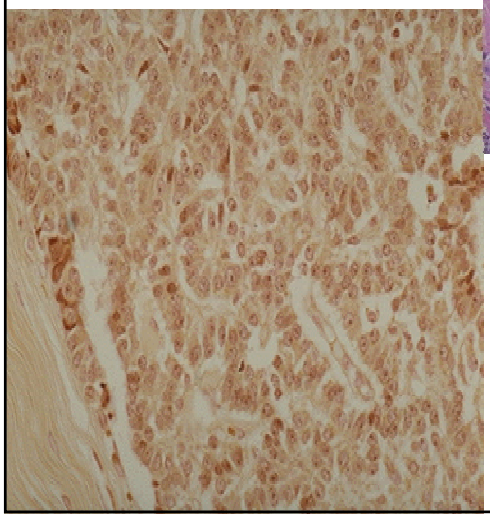






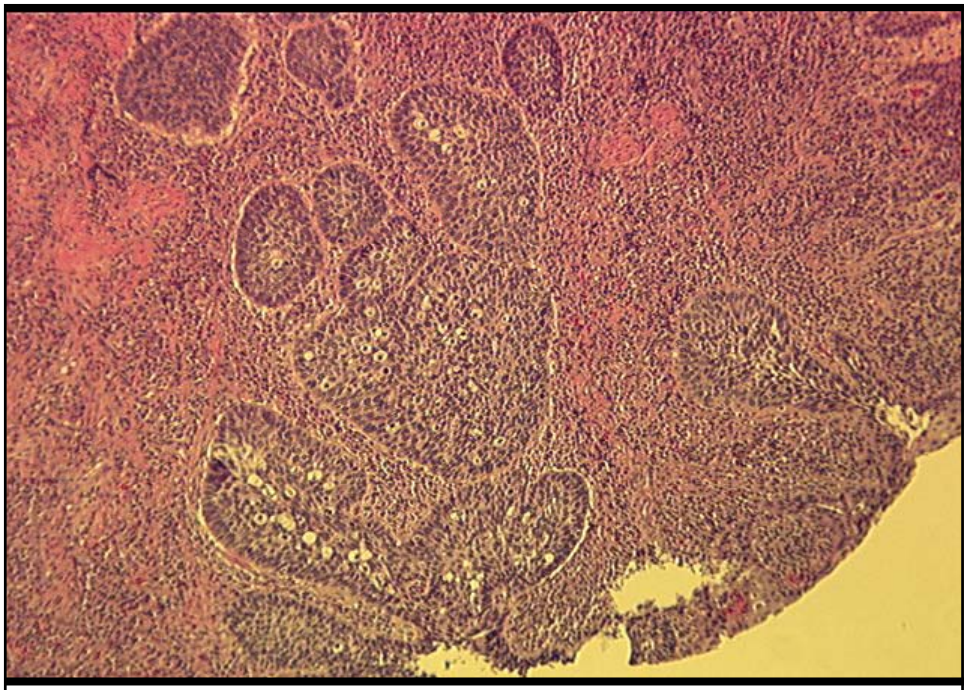
RECTAL CARCINOID



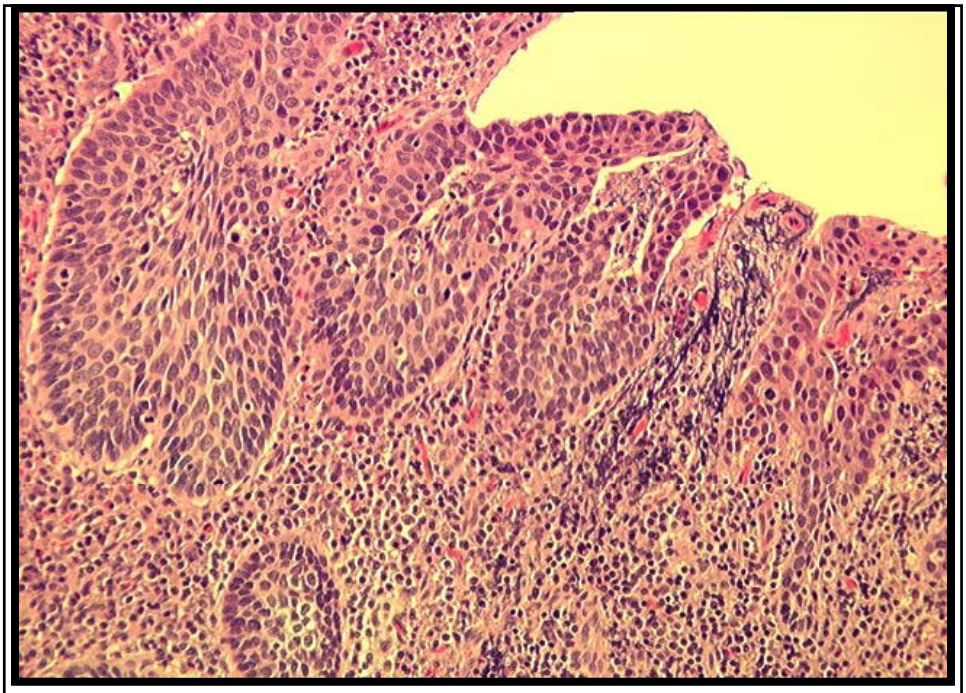


Cloacagenic_ca1

cloacagenic_ca1

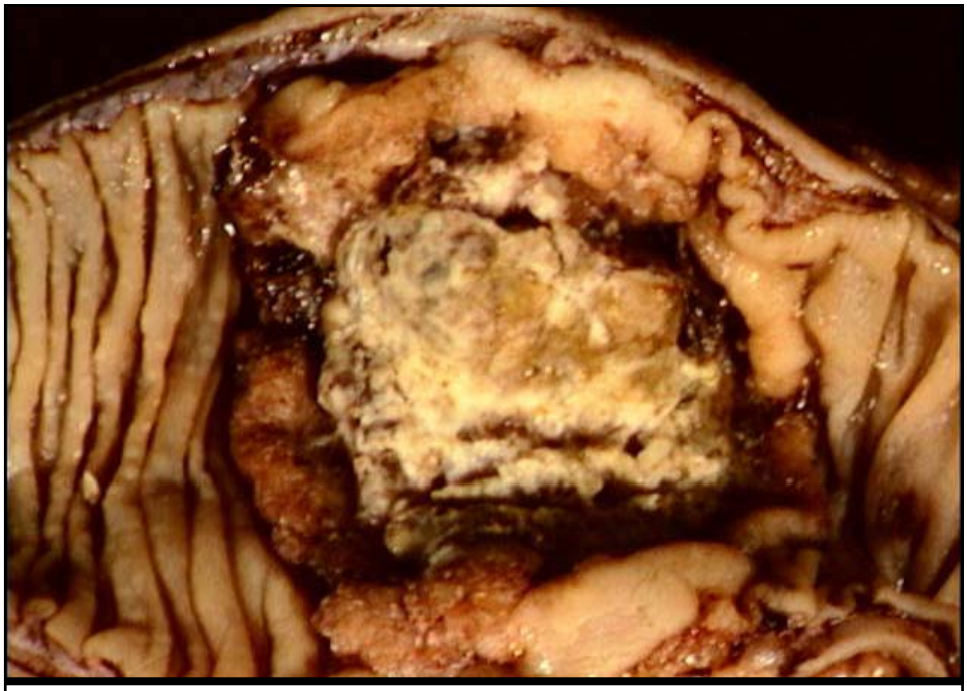


cloacagenic_ca5



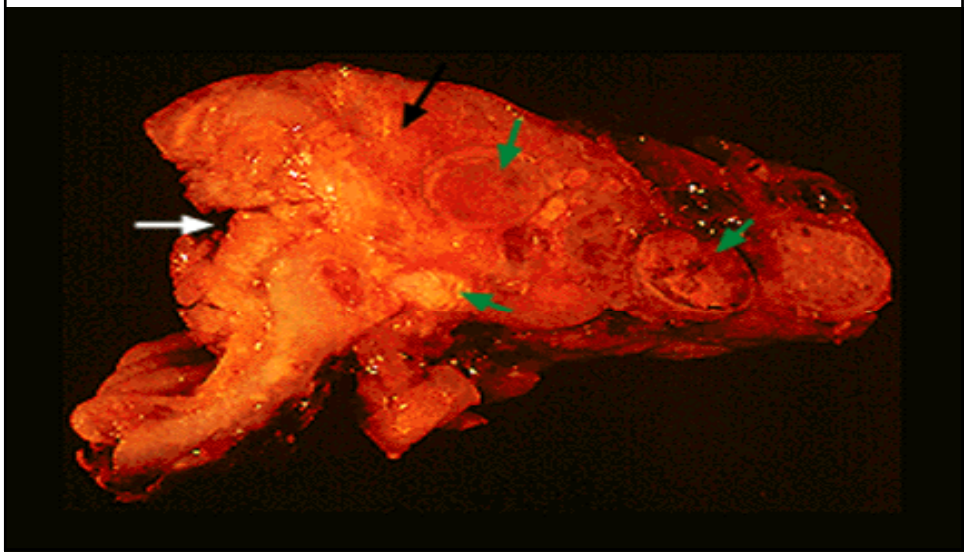
cloacagenic_ca6

MORE ADENOKC

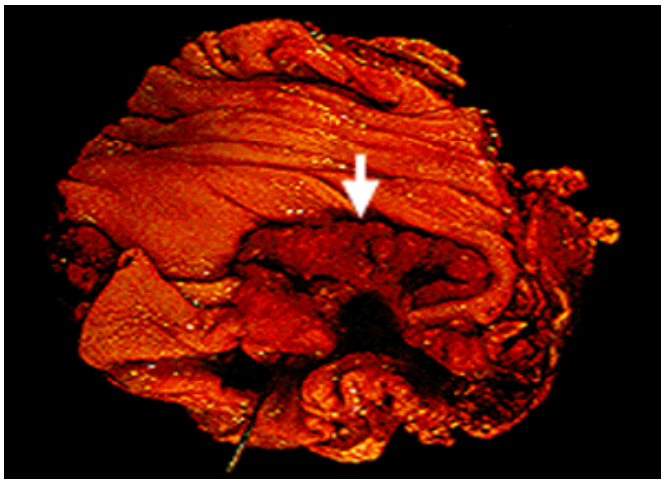


colon_ca

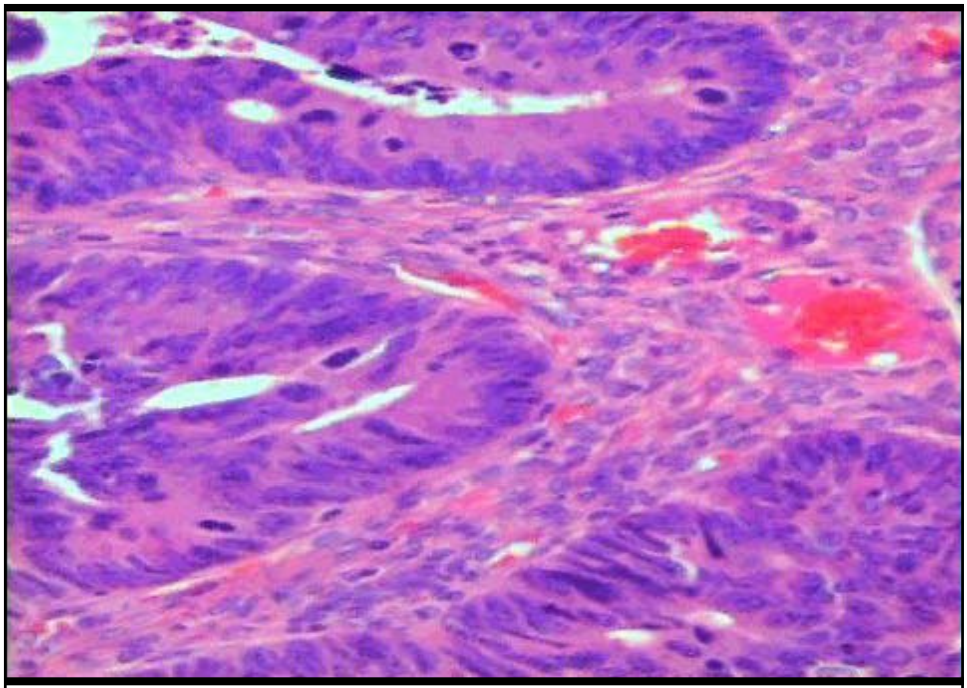
Adenocarcinoma, Colon
Adenocarcinoma with Lymph Node Metastases
•This is a view of the entire thickness of the colon wall and mesentery.
•Note the mucosal tumor(white arrow)
•Tumor involves full thickness of the muscularis propria advancing into the the pericolic fat(black arrow) •Multiple enlarged lymph nodes contain metastatic carinoma which is white to tan(green arrows)



Adenocarcinoma of the cecum



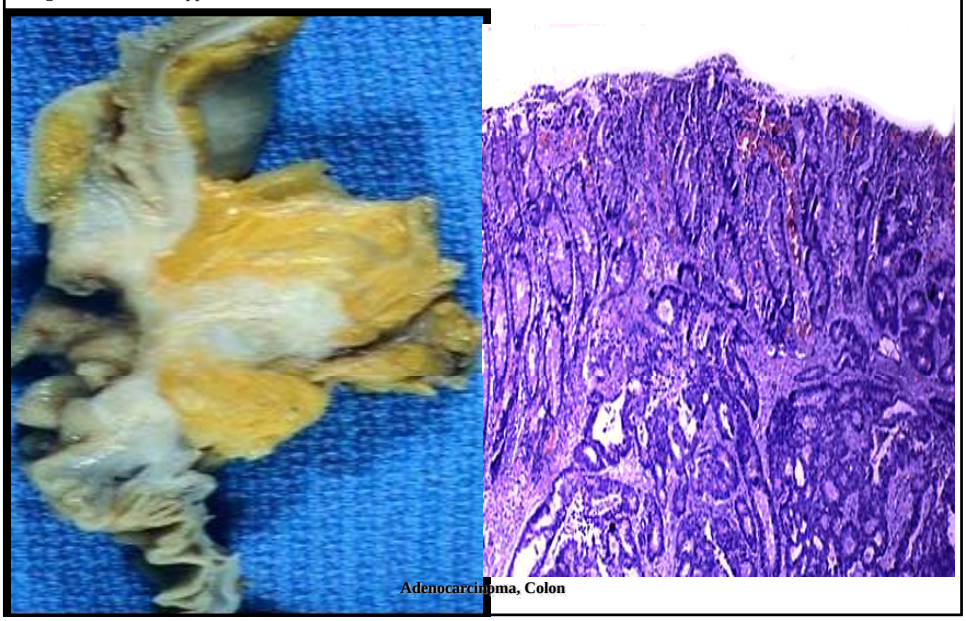
The cecum has been opened displaying the mucosal surface
•A probe is in the ileocecal valve
•The red tumor mass near the ileocecal valve has typical raised rolled clearly demarcated margins(arrow)
•Adjoining normal mucosa is smooth, tan and has normal folds



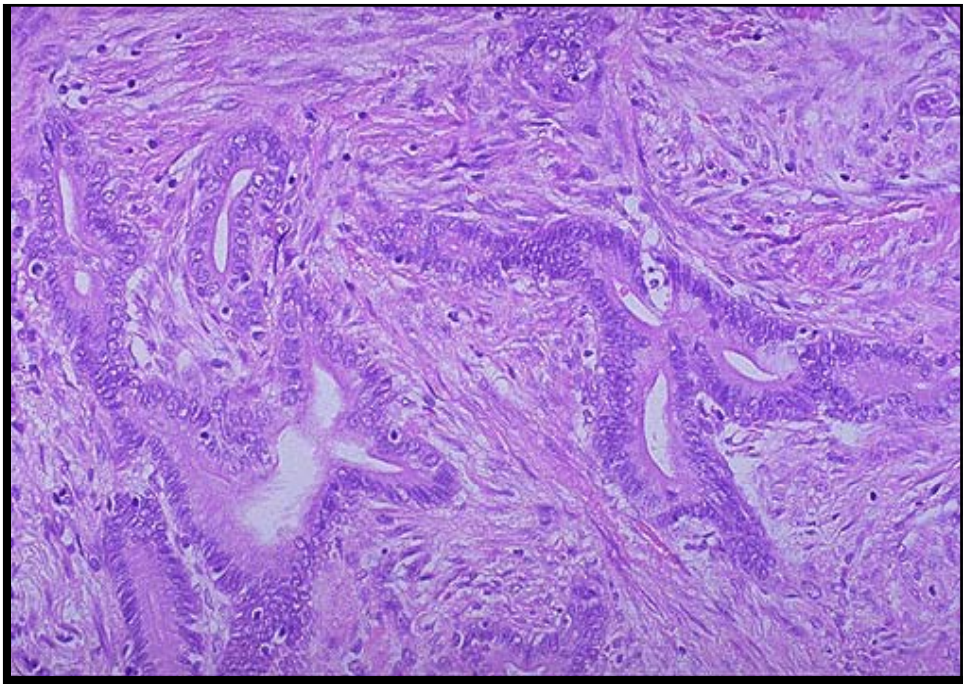
colon_ca2

Adenocarcinoma of Colon

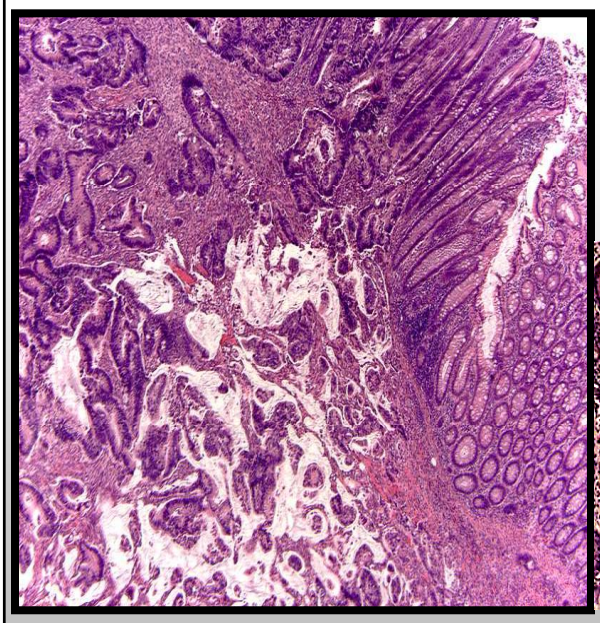
•The carcinoma penetrates the basement membrane to invade the submucosa
•The malignant cells are disorganized and crowded
•The lack of mucin within the tumor indicates loss of differentiation, in contrast to the remaining benign mucosa in the upper left



Adenocarcinoma, Colon

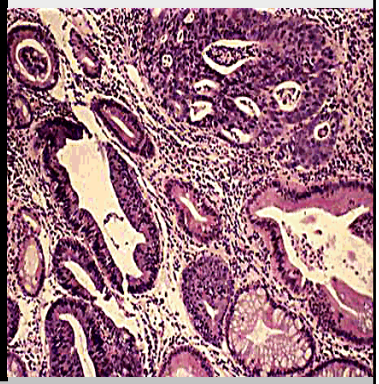


colon_ca3

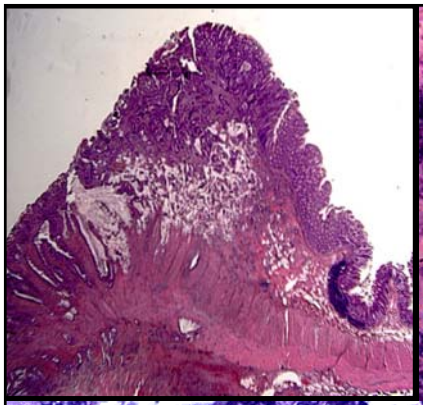


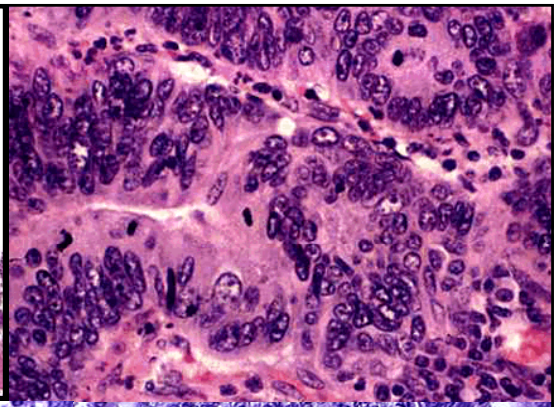
Adenocarcinoma, Colon
Adenocarcinoma of Colon (full spectrum of changes)
•Full spectrum from normal glandular tissue (bottom,right) dysplastic glands on the left and invasive adenocarcinoma seen at the top. •Note the highly irregular glandular architecture in the malignant tissue with hyperchromatic,irregular

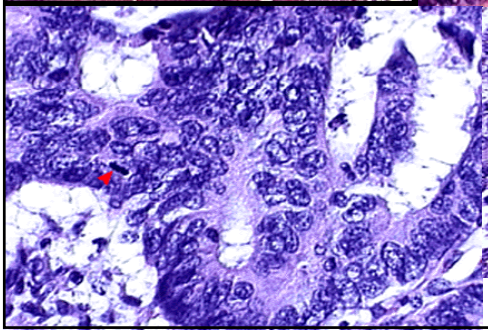
nuclei

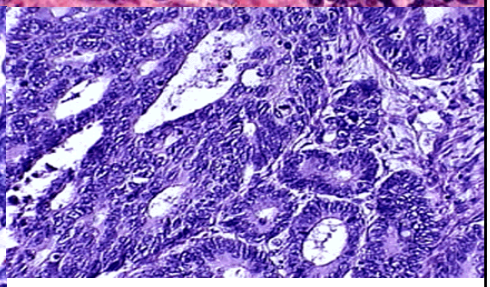


colon_ca4

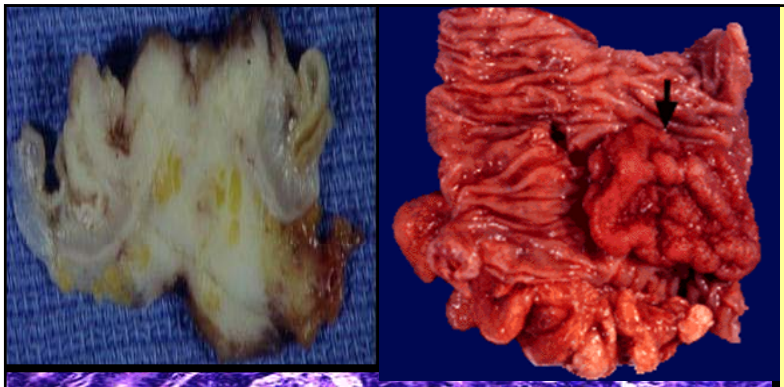








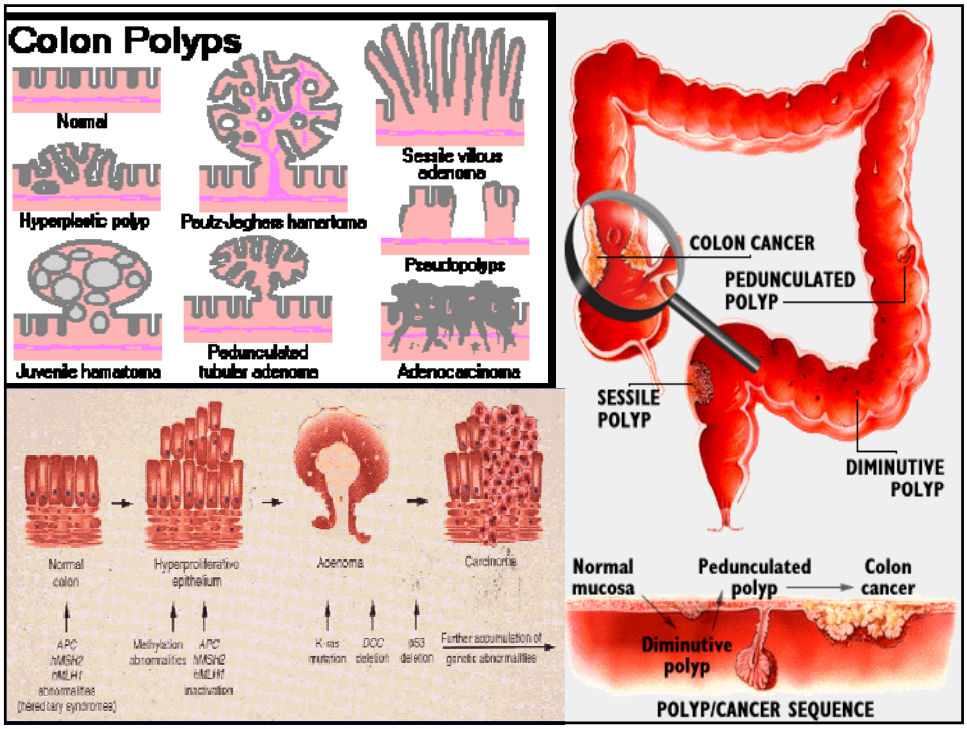
colon_ca5



colon_ca8

Adenocarcinoma, Colon
Adenocarcinoma of Colon
•The depressed central region of this raised tumor is the gross evidence that the original benign polyp has transformed to a carcinoma. The ulcerated center represents necrotic tumor •The smooth raised pink-white tissue surrounding the ulcerated tumor is viable tumor
•The lobulated edge(arrow) represents residual component of the adenomatous polyp in which this lesion originated
•The lesion is typically sharply demarcated from the adjoining normal mucosa

Polyps



Polyps
growths which develop in the colon and other parts of the body as well.
vary in size and appearance.
They may look like a wart when small and when they grow they may appear like a cherry on a stem or fig.
They are important because they can with time turn into cancer.
Sometimes they can bleed causing anemia.
A polyp is defined as a growth that projects, often on a stalk, from the lining of the intestine or rectum.
Polyps of the colon and rectum are almost always benign and usually produce no symptoms.

They may, however, cause painless rectal bleeding or bleeding not apparent to the naked eye. There may be single or multiple polyps.
The incidence of polyps increases with age.

The cumulative risk of cancer developing in an unremoved polyp is 2.5% at 5 years, 8% at 10 years, and 24% at 20 years after the diagnosis.
The probability of any singular polyp becoming cancerous is dependent on its gross appearance, histologic features, and size.

The relative risk of developing colon cancer after polyps have been removed is 2.3 compared to a relative risk of 8.0 for those who do not have the polyps removed.

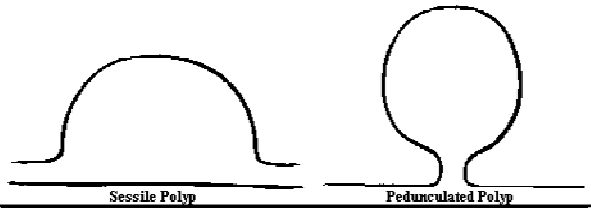
Polyps greater than 1 centimeter have a greater cancer risk associated with them than polyps under 1 centimeter.

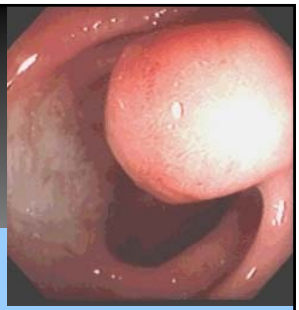
Polyps with atypia or dysplasia are also more likely to progress on to colon cancer.

The risk of cancer is much higher in sessile villous adenomas than in pedunculated tubular adenomas. Cancer is found in 40% of villous adenomas, as compared to 15% in tubular adenomas.

The good news is that 65% of adenomas are tubular, with villous adenomas accounting for only 10% of adenomatous polyps.

It has been shown that the removal of polyps by colonoscopy reduces the risk of getting colon cancer significantly.





Classification of large bowel polyps

- Epithelial
  - Adenomas - tubular, villous, tubulovillous
  - Metaplastic polyps
- Mesodermal
  - Lipoma
  - Leiomyoma
  - Haemangioma
- Hamartoma
  - Juvenile polyps
  - Peutz-Jeghers syndrome

Colonic Polyps

Persons with family members with colonic polyps are at increased risk for development of colorectal cancer.

Those patients should have individualized screening performed.

However, the screening recommendations from the American College of Gastroenterology are similar to those described above for colorectal cancer.

Surveillance of Colorectal Cancer

Persons who have had colorectal Cancer or Pre-cancerous Colorectal Polyps require continued evaluation of the colon to prevent recurrence of colon polyps and colorectal cancer. This process is called *surveillance*.

Recommendations for Surveillance of Colorectal Cancer and Polyps

Findings of Most Recent Colonoscopy	ACG Recommendations
Colorectal Cancer	Colonoscopy between 3 months and 1 yr. *
Pre-Cancerous Polyps	Colonoscopy between 3 months and 1 yr. *
Normal Examination	Colonoscopy -- usually in 5 years

Calcium

Calcium is some way regulates the growth of the cells that line the inside of the colon. This may be why medical studies are beginning to show that people who get 1000-1500 mg of calcium a day in their diet have less colon cancer. This level of calcium (from milk, dairy products, vegetables or supplements) is currently recommended for healthy bones. It may also benefit the colon.

Three Categories of Polyps

Enough is now known about polyps that physicians generally place patients in one of three categories. In each of these the end result is an adenoma type polyp:

1.The Ordinary Polyp -- Most sporadic polyps occur between the ages of 40 and 60. There may be only one or two present and they may take ten years or more to develop into a cancer. There is a hereditary link. Eventually some of these become cancer.

2.Hereditary Familial Polyposis -- This is a true hereditary condition in which the entire colon is studded with hundreds, even thousands of polyps. They begin at a very early age even under ten years old. Virtually every patient will eventually develop colon cancer. The only known preventive treatment is surgical removal of the colon. Fortunately, the condition is not common.

3.Lynch Syndrome (Hereditary Non-Polyposis Colorectal Cancer) -- This disorder is more common than familial polyposis but less so than the ordinary polyp. There is a strong tendency for adenoma type polyps to occur in close blood relatives such as sisters, brothers, aunts, uncles and children. More polyps are seen and at an early age. Polyps and even cancer occur at earlier ages, 40's, 30's and even in the 20's. In some families there is also an increased incidence of breast, ovarian, and other cancers. So a family history of this type warrants very close surveillance of all direct blood relatives

Polyps
A. Adenomatous Polyps
1. Tubular & tubulovillous adenoma
2. 2. Villous adenoma
Surgical treatment: All adenomatous polyps are considered pre-malignant and should be removed, colonoscopically if possible. Surveillance colonoscopy recommended after removal.

B. Hereditary polyposis syndromes
1. Familial polyposis: innumerable colonic polyps, early development of colon cancer. also desmoid tumors, periampullary cancers, papillary thyroid cancer.
2. 2. Gardner's Syndrome (osteomas of mandible, sebaceous cysts, lipomas, fibromas, small bowel malignancy) Surgical treatment: Total colectomy with permanent ileostomy or ileal pouch anal anastomosis (endorectal pull-through).
3. 3. Turcot's Syndrome (CNS malig. & colon polyps)
4. 4. Muir-Torre Syndrome (sebaceous adenoma & colon ca)

C. Hamartomatous polyps (not assoc. with malignancy)
1. Juvenile polyps

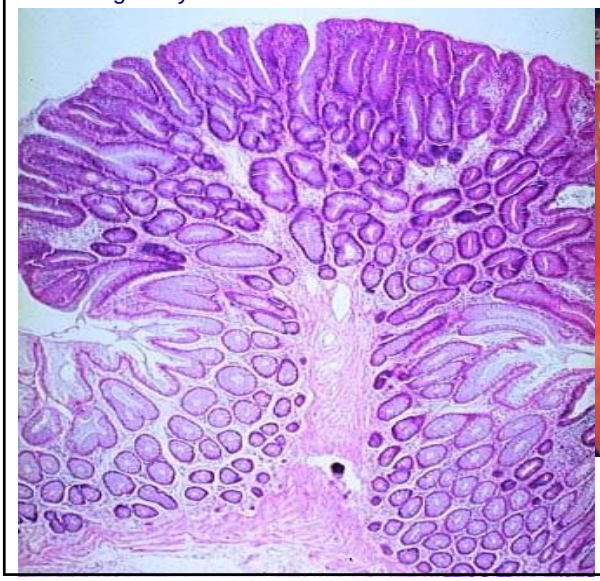
D. Other benign masses
1. Neurofibromas 2. Leiomyomas 3. Lipomas (intramural)

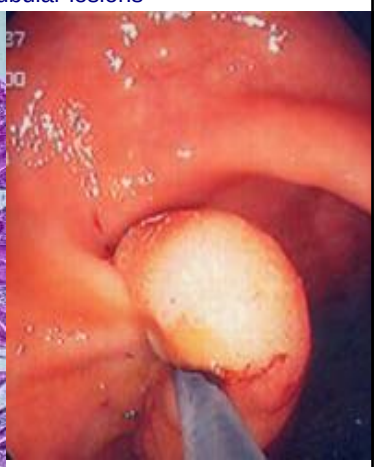
POLYPS of the COLON:
- **(A)BENIGN POLYPS:**
  - **HYPERPLASTIC POLYPS:**
    - **PATHOLOGY: Serrated appearance of crypts, frequent in rectosigmoid area. Distinguished by the proliferation in the crypts.**
    - **CLINICAL: not neoplastic, but often found concurrently with colon cancers.**
  - **JUVENILE POLYPS: Non-neoplastic polyp which can undergo torsion and cause rectal bleeding in children[SEE ALSO BELOW].**
    - **PATHOLOGY: hamartomas and they are never cancerous.**
  - **INFLAMMATORY POLYPS: "Pseudopolyps," often found in Ulcerative Colitis**

- **B)ADENOMATOUS POLYPS: Extensions of colonic epithelia, maybe cancerous.**
  - **PATHOGENESIS: They are thought to result from an over-proliferation of crypt cells. Crypts cells normally grow up the colonic villus as part of the process of normal cell-turnover. When this process grows out of control, polyps result.**
  - **PATHOLOGY: Two different forms.**
    - **TUBULAR ADENOMAS: Pedunculated, gland-like structures.**
    - **VILLOUS ADENOMAS: Extensions of mucosal surface, cauliflower-like surface. More finger-like structure.**
      - **Generally, Villous Adenomas are more likely to progress to cancer.**
  - **CLINICAL: pre-cancerous. The size of the polyp correlates with the risk for cancer.**
- **POLYPOSIS SYNDROMES:**
  - **ADENOMATOUS POLYPOSIS COLI (APC): Autosomal Dominant, progressive development of lots of adenomatous polyps in the colon.**
    - **CLINICAL: Progression to colonic adenocarcinoma is inevitable.**
  - **GARDNER SYNDROME: Autosomal dominant disease.**
    - **CLASSICAL SYMPTOMS:**
      - **Polyposis of GI tract, especially in colon, but it also occurs in stomach and the Ampulla of Vater.**
      - **Osteomas of skull, mandible, long bones.**
      - **Soft tissue tumors of the skin.**
    - **CLINICAL: Gardner Syndrome progresses to Colon Cancer**

Adenomas

- Benign epithelial neoplasm
- They are pre-malignant
- Risk of malignancy increases with size
- Malignancy more common in villous rather than tubular lesions



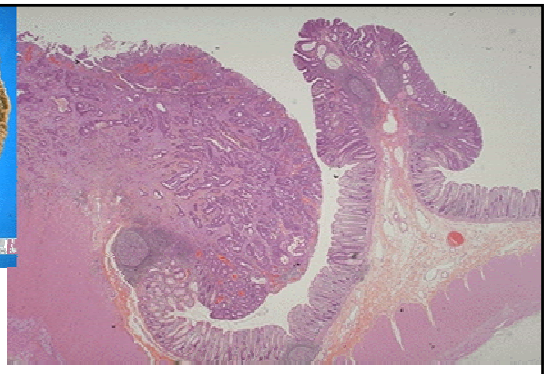


adenomatous colon polyps

prob. of malignancy by size and type

Size (cm)	< 1	1-2	> 2
Tubular	1%	10%	34%
mixed (TV)	4%	9%	45%
villous	10%	10%	54%

- most colon polyps (90%) are hyperplastic (size < 5 mm)



polyposis syndromes

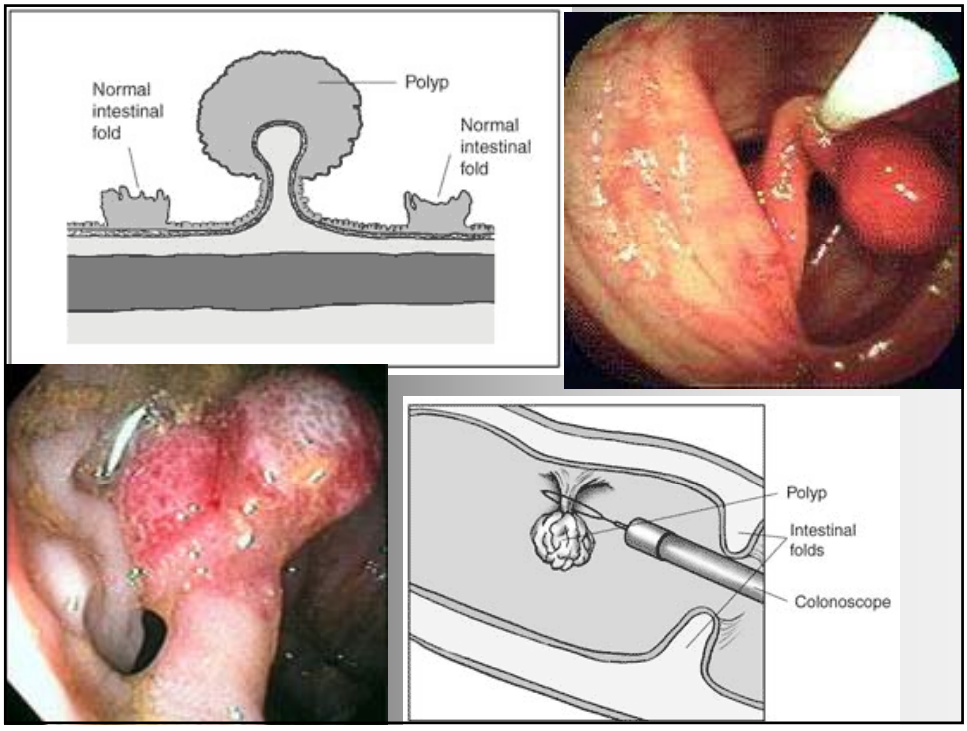
	Inher.	Malig.	Type
familial polyposis coli	dom	+	adenoma
Gardner syndrome	dom	+	"
Turcot syndrome	rec	CNS	"
Peutz-Jeghers syndrome	dom	(+)	hamartoma
Cowden syndrome	dom	?	"
juvenile polyposis coli	(?)	-	juvenile
Cronkhite-Canada syndrome	---	-	"

Familial Polyposis Coli

- autosomal dominant (33% sporadic)
- adenomas ==> colon Ca
  - absent at birth
  - adenomas develop in teenagers
- associations:
  - desmoid tumors
  - SB adhesions

Familial adenomatous polyposis

- etiological factor in 1% of colorectal cancers
- Its is an autosomal dominant
- Due to mutation on long arm of chromosome 5
- Mutation induces proliferation of mucosa throughout GI tract
- Develop colonic polyps in teens or early 20's
- Untreated progresses to cancer by 30's
- Screening – by rigid or flexible sigmoidoscopy
- Safe alternative to colonoscopy as rectal sparing rarely seen
- Start late teens and continue until 40 yrs and polyp free.
- Extra-colonic manifestations:
  - Osteomas. epidermoid cysts = Gardener's Syndrome
  - Gastroduodenal polyps
  - Desmoid tumours
  - Congenital hypertrophy of retinal pigmented epithelium
- Surgical options:
  - Panproctocolectomy and ileostomy
  - Restorative panproctocolectomy
  - Subtotal colectomy and ileorectal anastomosis
  - NB will require surveillance of rectal stump



Peutz-Jeghers syndrome

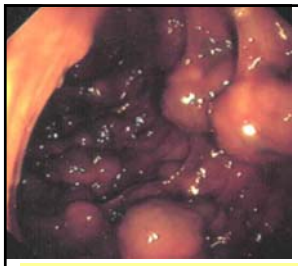
- autosomal dominant (50% sporadic)
- hamartomas
  - primarily of SB
  - also: stomach, colon
  - occasionally of urinary or respiratory tract
- mucocutaneous hyperpigmentation
- increased risk of GI + non-GI tumors! (NEJM 1988)

Cowden syndrome

"multiple hamartoma syndrome"
- •GI-tract hamartomas (incl. stomach and colon)
- •breast Ca
- •thyroid Ca
- •circumoral papillomatosis
- •nodular gingival hyperplasia

Cronkhite-Canada syndrome

- •not inherited; no pattern
- •inflammatory glandular dilatation of stomach, colon, SB (50%)
- •juvenile-type polyps
- •no malignant potential
- •protein and electrolyte loss
- •ectodermal abnormalities
  - •alopecia
  - •hyperpigmentation
  - •nail loss (onycholysis)
- •prognosis:
  - •males: remits
  - •females: die in 6-18 months d/t cachexia

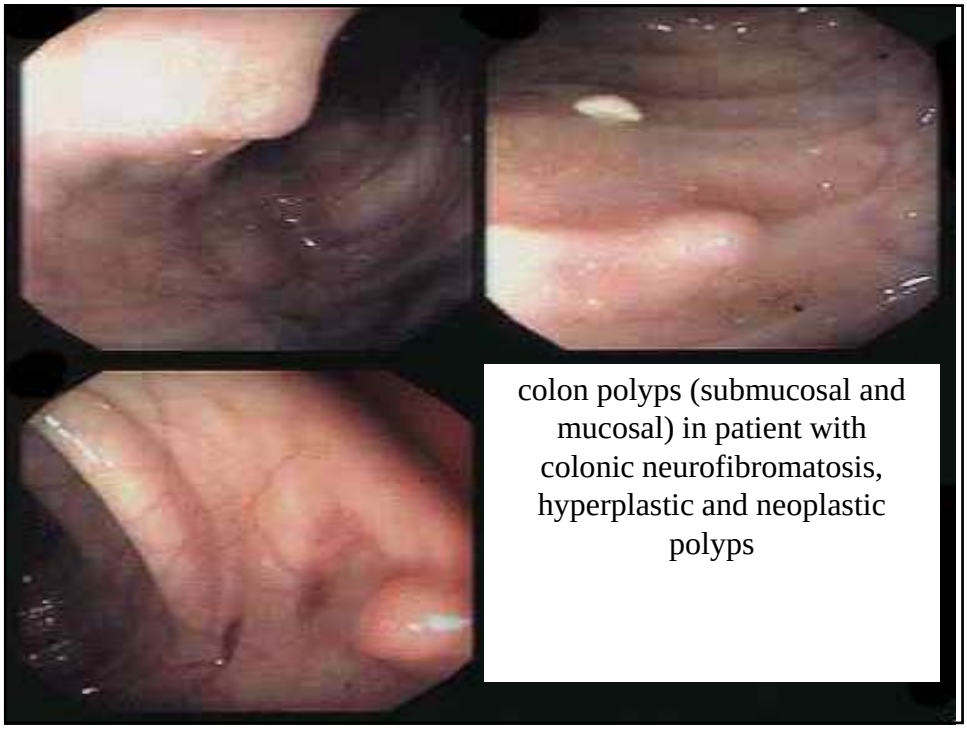


Juvenile Polyposis Coli

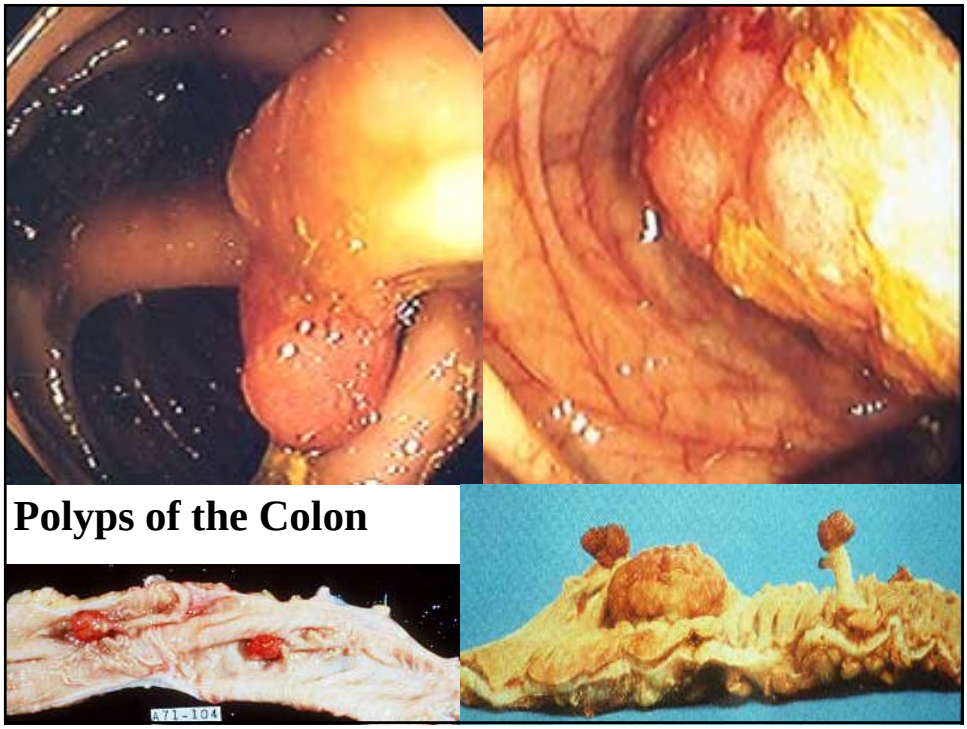
- benign polyposis
- inheritance uncertain
- inflammatory or retention polyps: round, smooth, soft, mucin-filled, non-neoplastic
- onset < 10 yrs
- polyps can prolapse thru anus
- a/w diarrhea, protein loss

Gardner syndrome

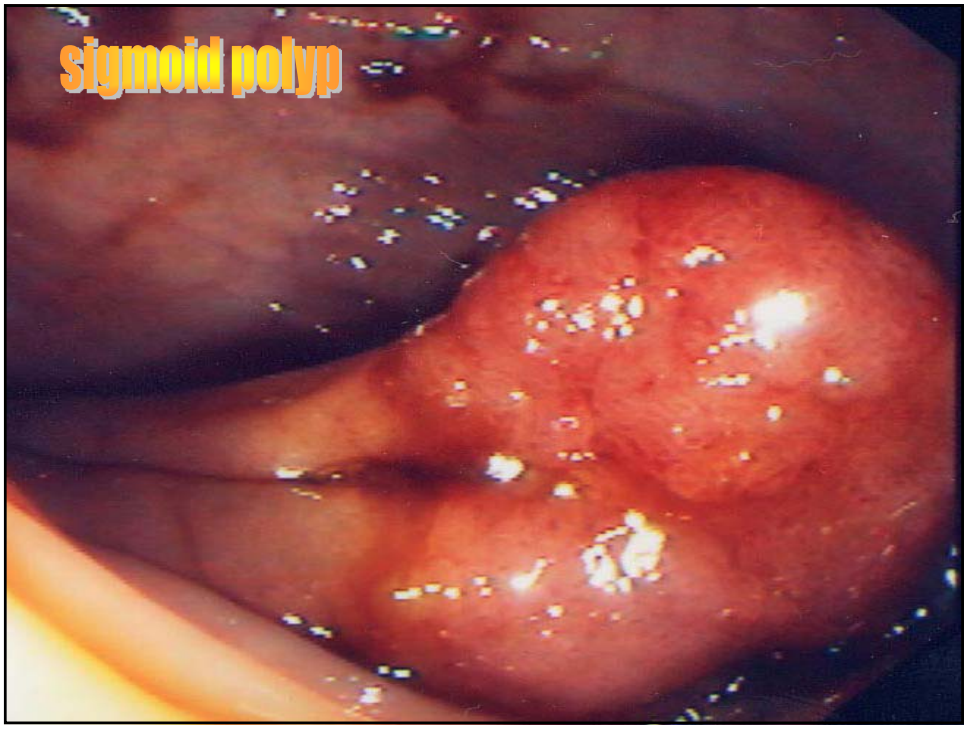
- autosomal dominant
- adenomas --> colon Ca (tx: colectomy)
- periampullary Ca
- soft-tissue lesions
  - sebaceous cysts
  - subQ fibromas, leiomyomas, lipomas
- bony lesions
  - osteomas (esp. in sinuses)
  - exostoses
  - cortical thickening
  - dental abnormalities (caries, extra teeth, odontomas

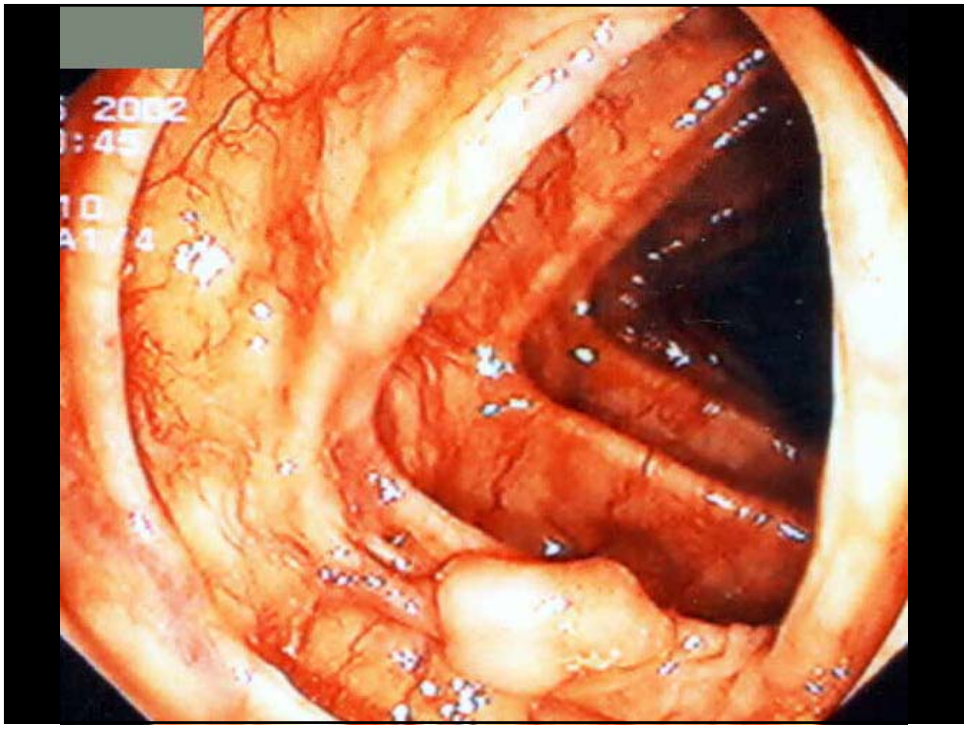


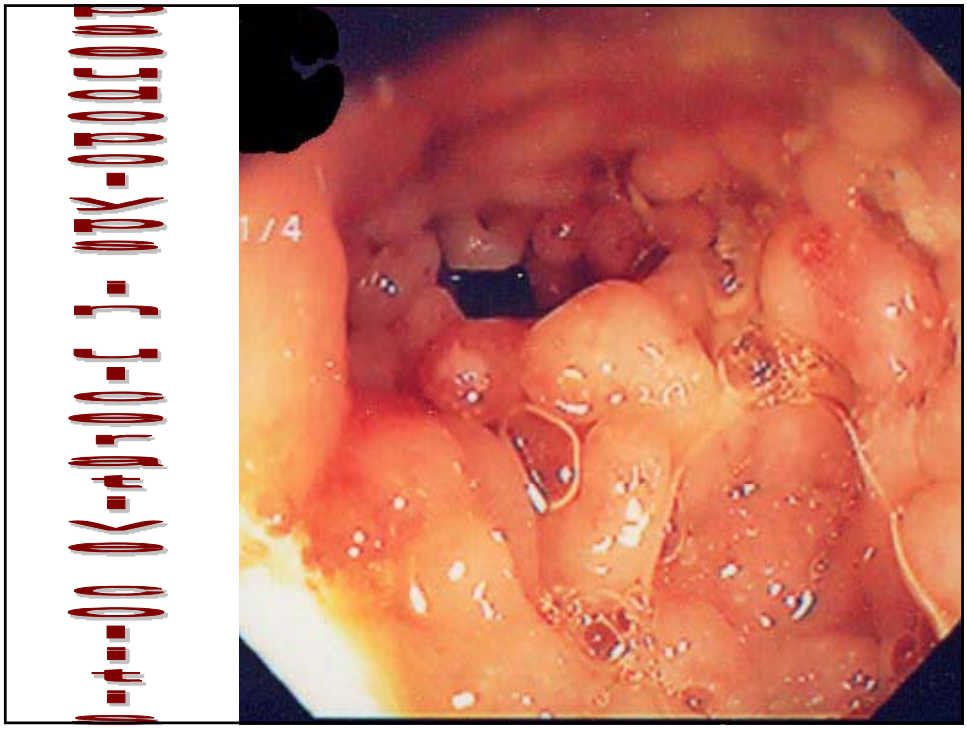
colon polyps (submucosal and mucosal) in patient with colonic neurofibromatosis, hyperplastic and neoplastic polyps

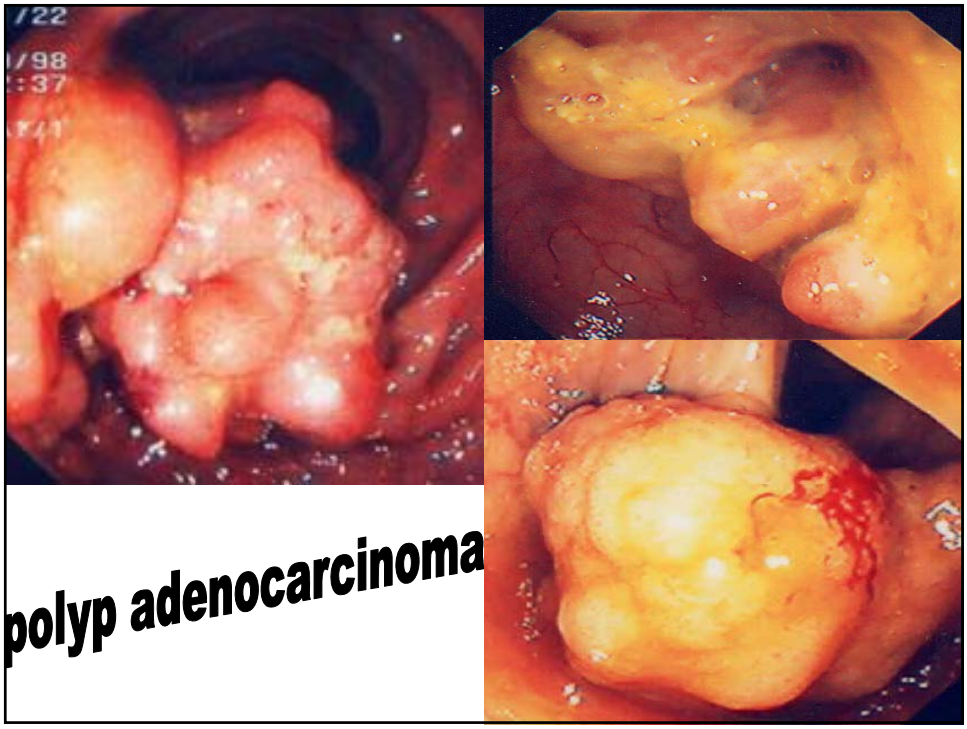


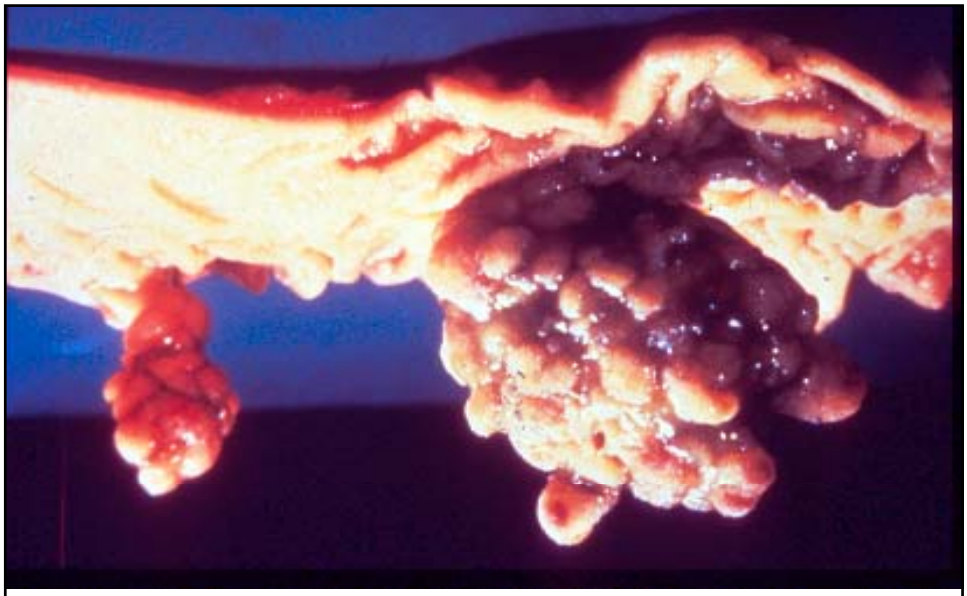
Polyps of the Colon


sigmoid polyp

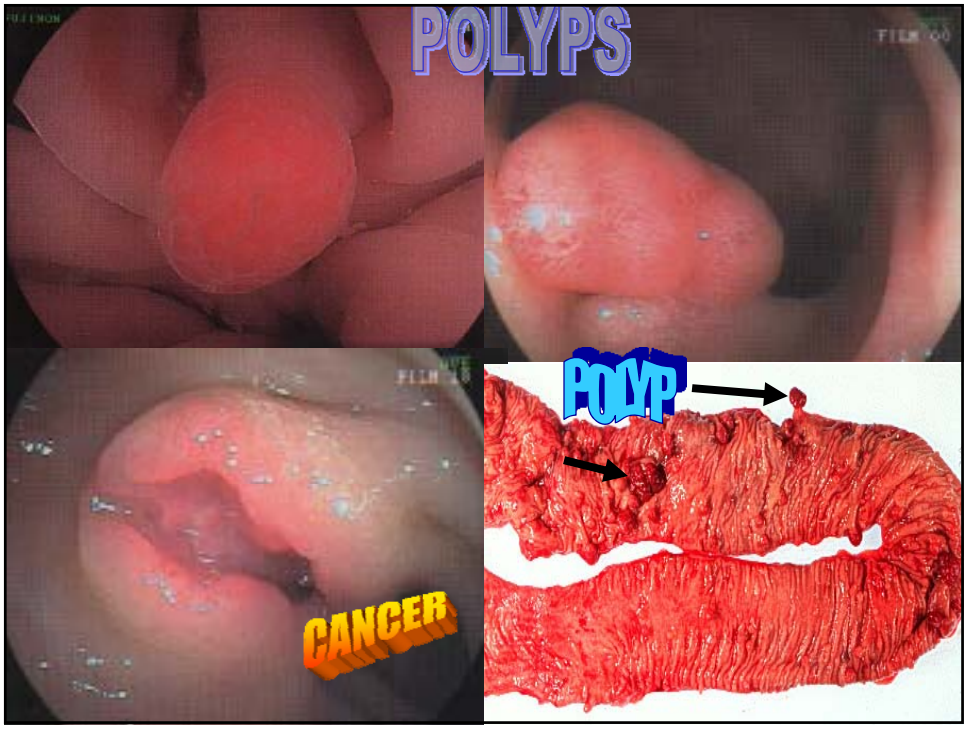




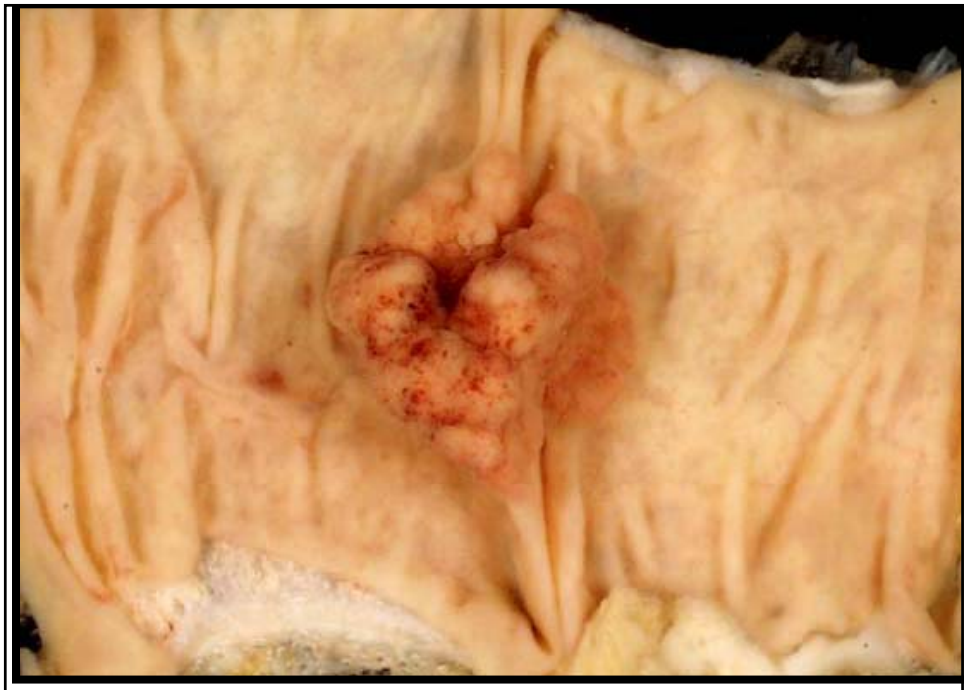

polyp adenocarcinoma



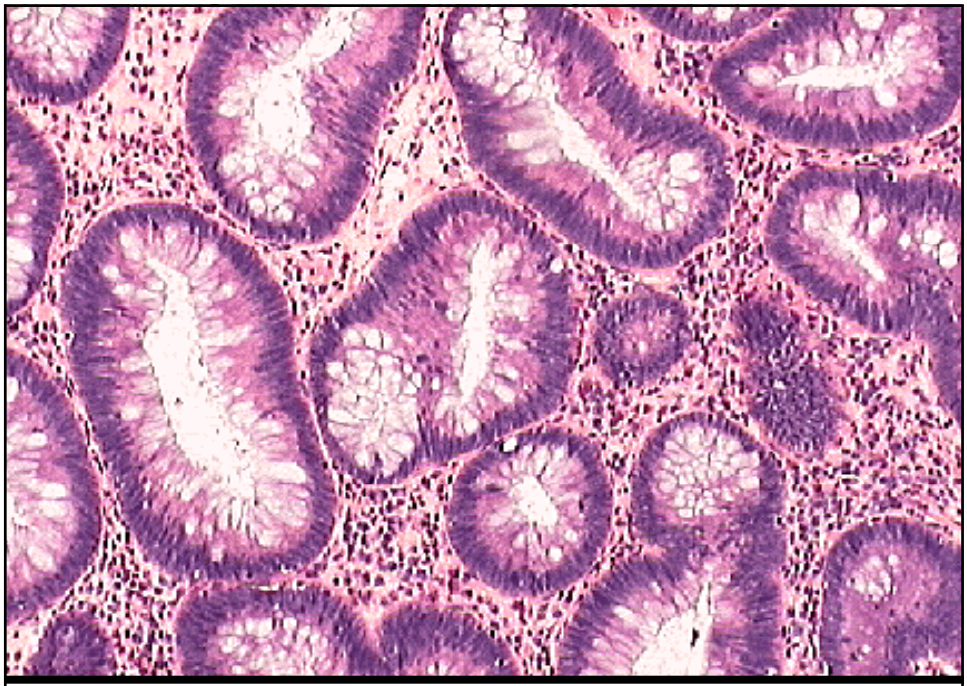
SUSPECT MULTIPLE POLYPS VILLOUS



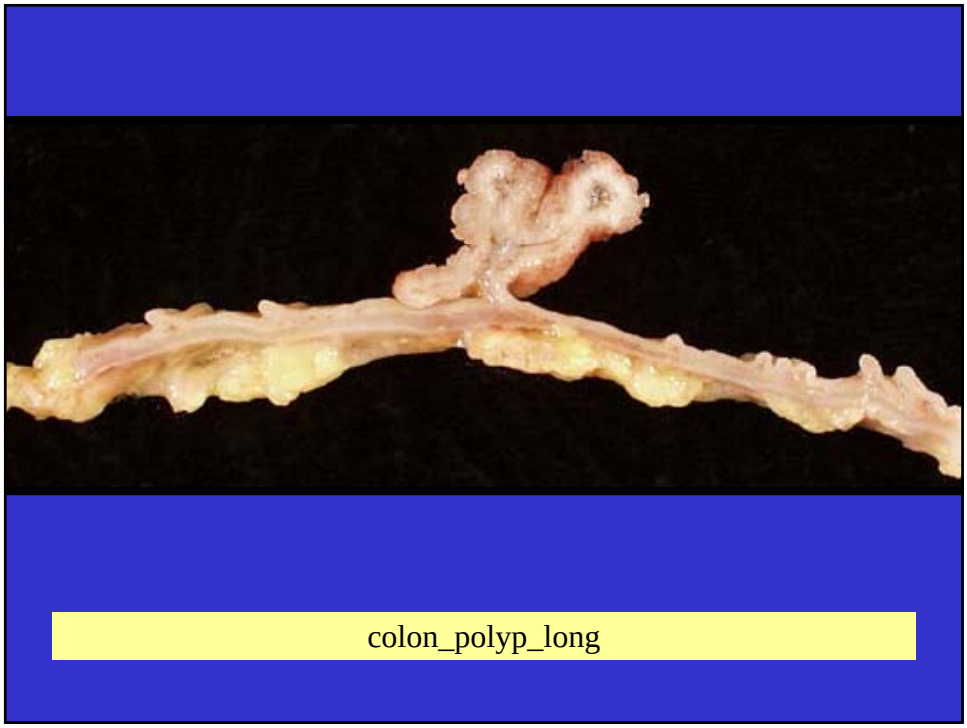
colon_polyp



colon_polyp_face

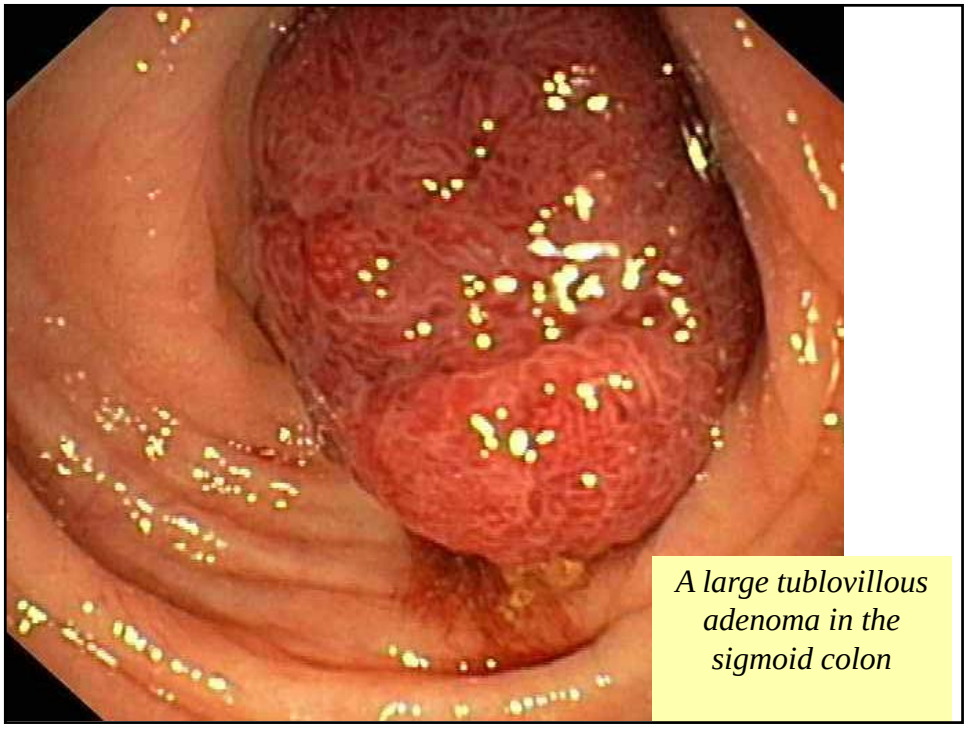


colon_polyp_histo

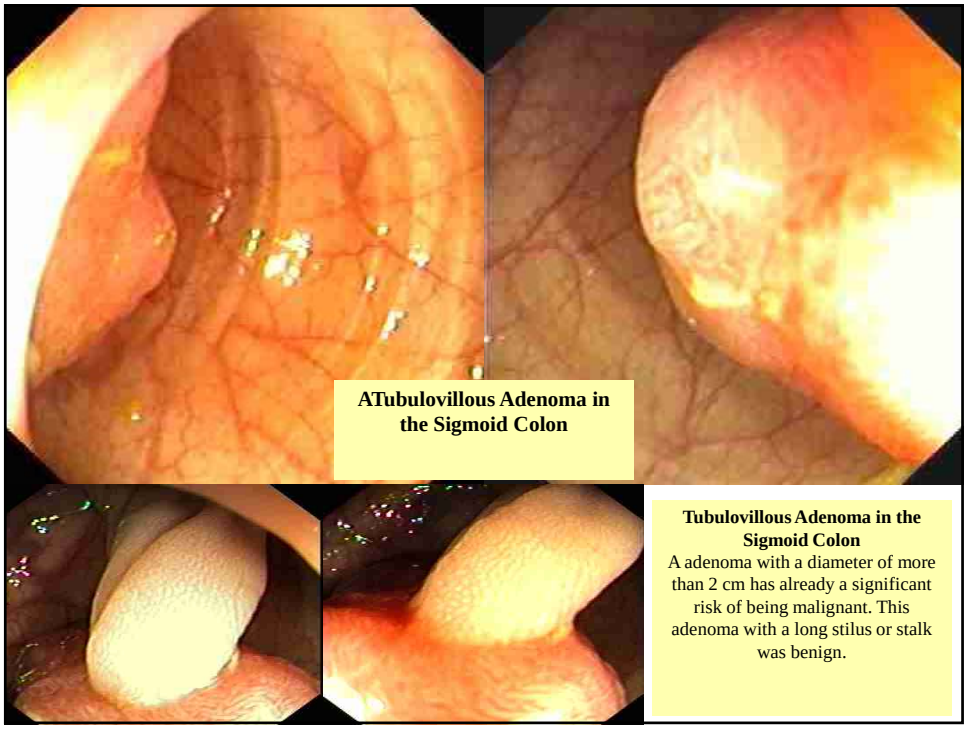


colon_polyp_long

colon_villous_polyp_1_gi2-1

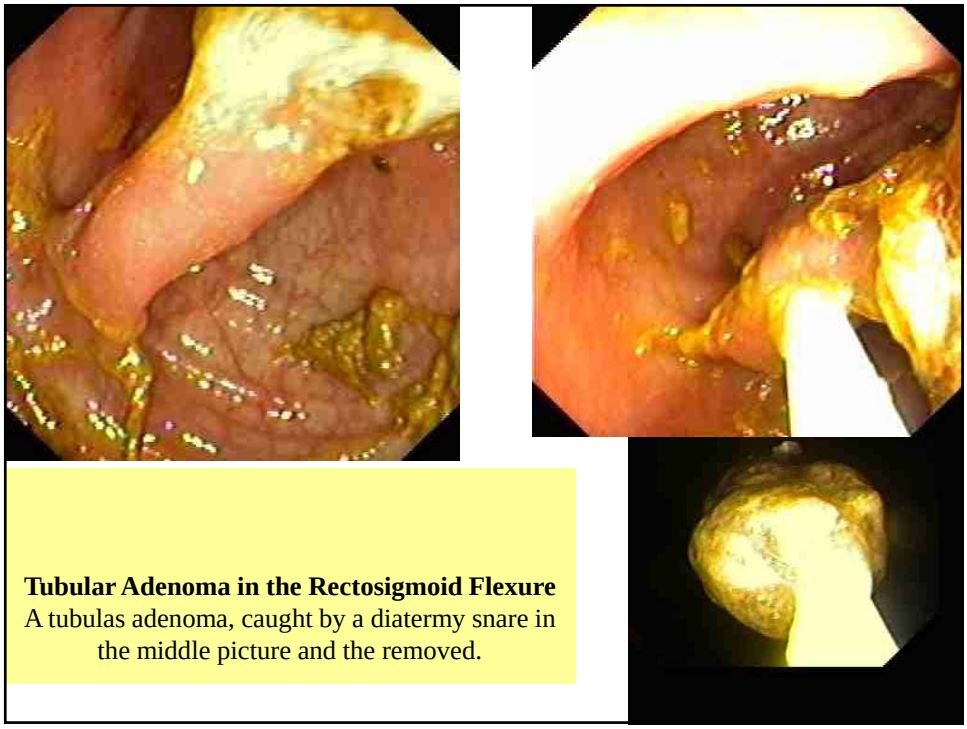


A large tublovillous adenoma in the sigmoid colon

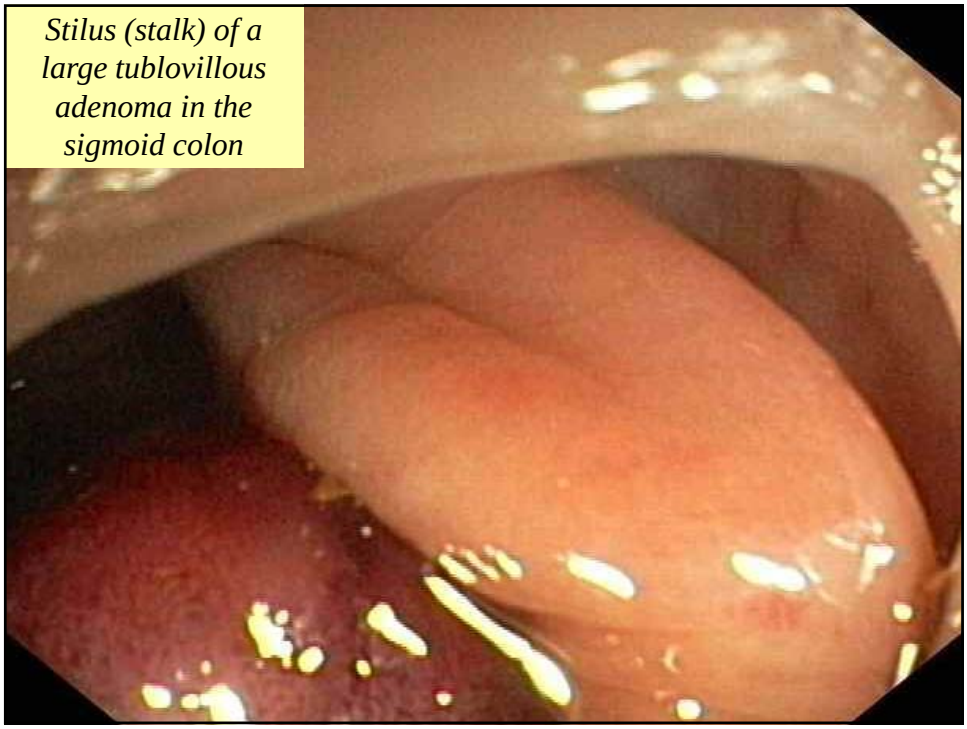


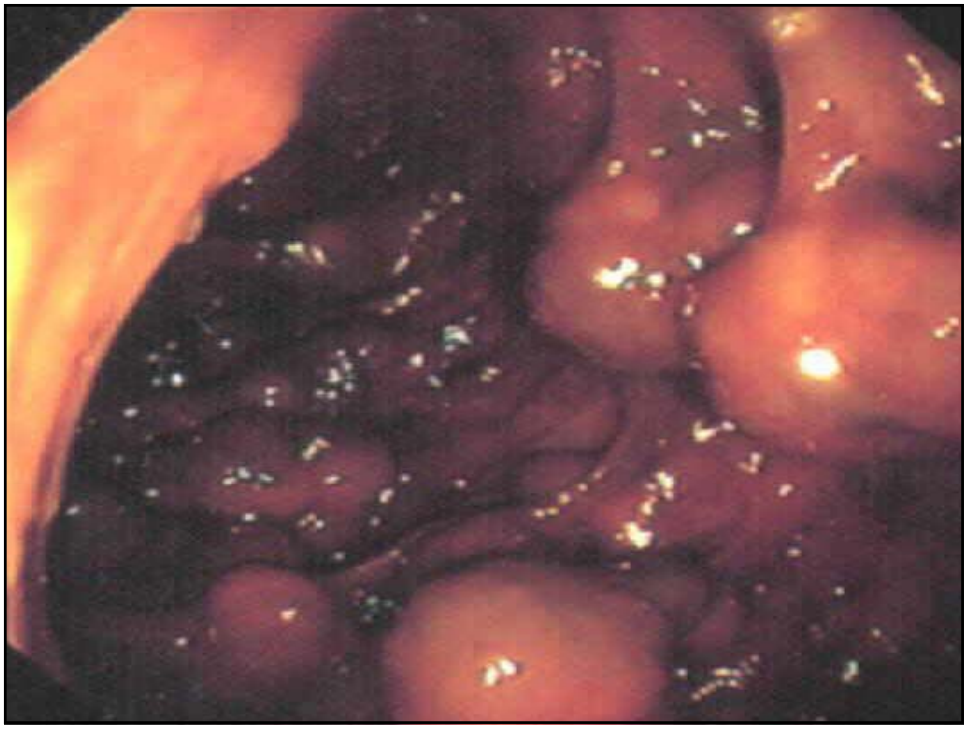
ATubulovillous Adenoma in the Sigmoid Colon

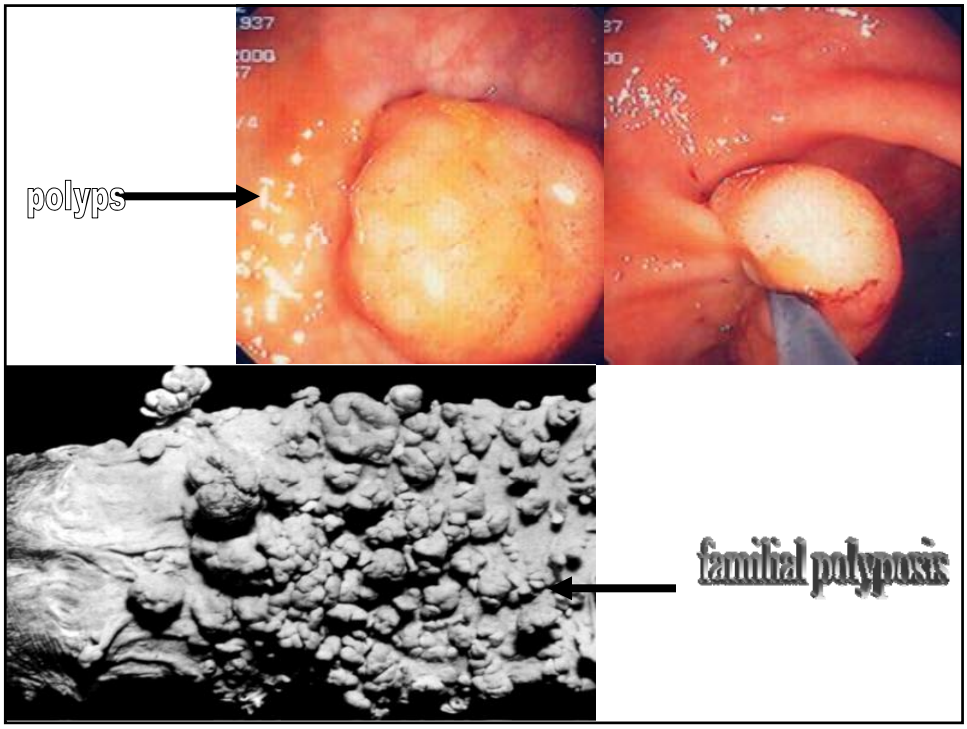
Tubulovillous Adenoma in the Sigmoid Colon
A adenoma with a diameter of more than 2 cm has already a significant risk of being malignant. This adenoma with a long stilus or stalk was benign.

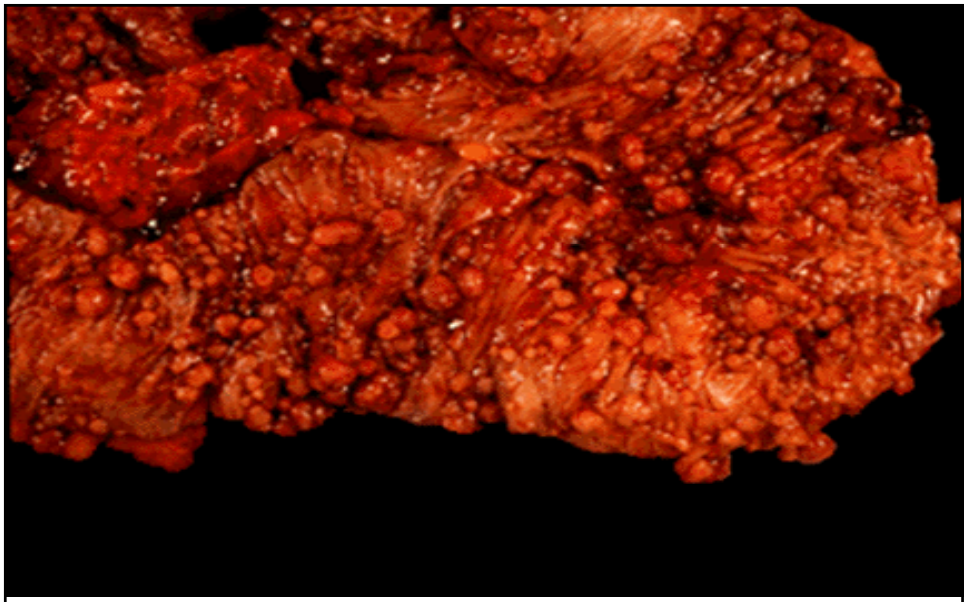


Tubular Adenoma in the Rectosigmoid Flexure
A tubulas adenoma, caught by a diatermy snare in the middle picture and the removed.


Stilus (stalk) of a large tublovillous adenoma in the sigmoid colon







Polyposis, Colon

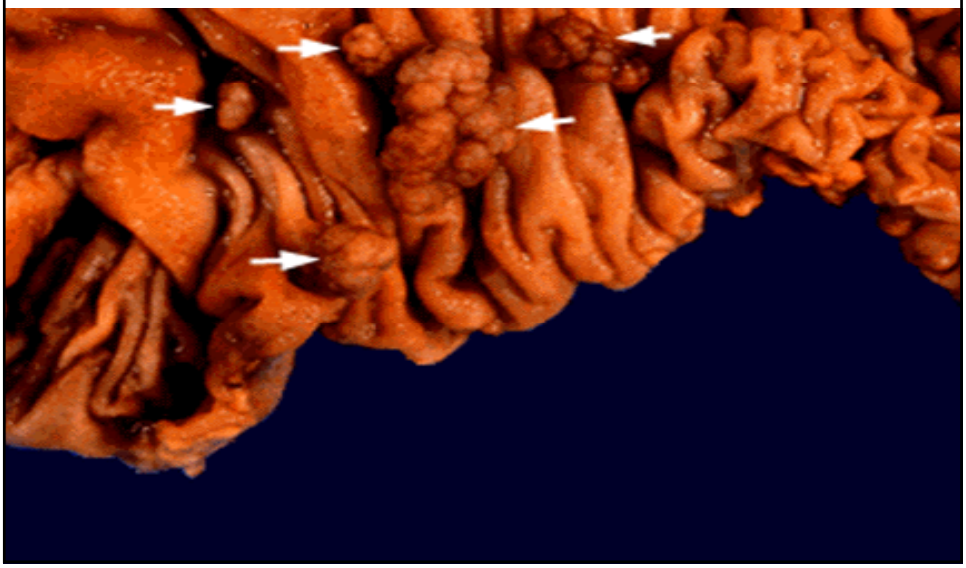
Polyposis Coli

•Opened colon displays a mucosal surface covered with numerous small to medium sized polyps
•The absence of a large mass lesion does not guarentee that carcinoma is not present

Polyposis, Colon

Multiple Polyposis of Colon

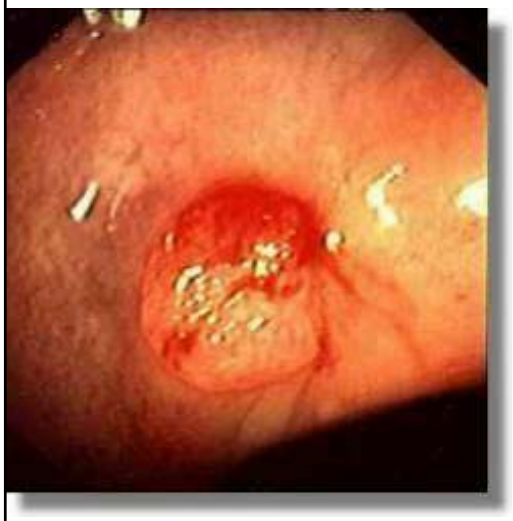
•Multiple adenomatous polyps (arrows) project from an otherwise normal appearing colonic mucosa.
•The number of polyps present in this case is at the low end of the number usually seen in APC.



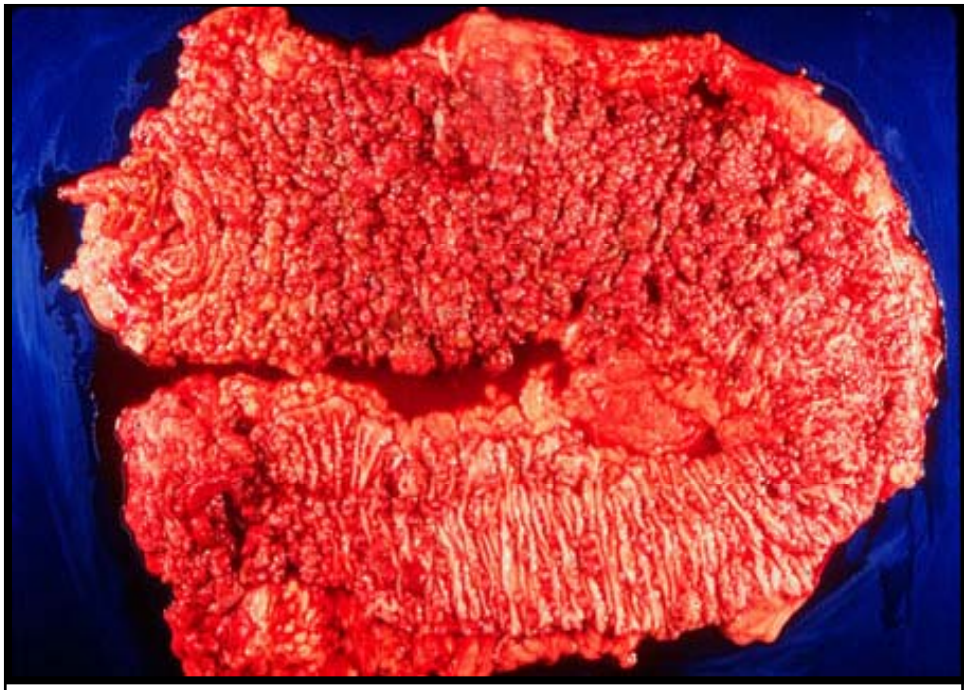
Large bowel mucosa with multiple adenomatous polyps

The patient has Familial Adenomatous Polyposis (FAP). It is inherited as an autosomal dominant condition with a high degree (>95%) of penetrance. It should be noted that 25% of those affected have no family history, the condition arising due to a new gene mutation. It is the commonest single gene disorder giving rise to a cancer. Those who possess the mutant gene invariably develop multiple colorectal polyps during their teens and untreated progress to colorectal cancer by their 30s or 40s. The polyps are usually tubular adenomas with over 1000 polyps often found in a typical patient.
The Adenomatous Polyposis Coli (APC) gene is found on the long arm of chromosome 5. FAP is associated with multiple osteomas and epidermoid cysts (Gardner's Syndrome). Other extra-intestinal manifestations include gastroduodenal and periampullary polyposis, desmoid tumours and pigmented retinal lesions (CHRPE = Congenital Hypertrophy of Retinal Pigment Epithelium). Probably all of these manifestations of FAP arise from the one genetic defect with variable phenotypic expression in different patients.

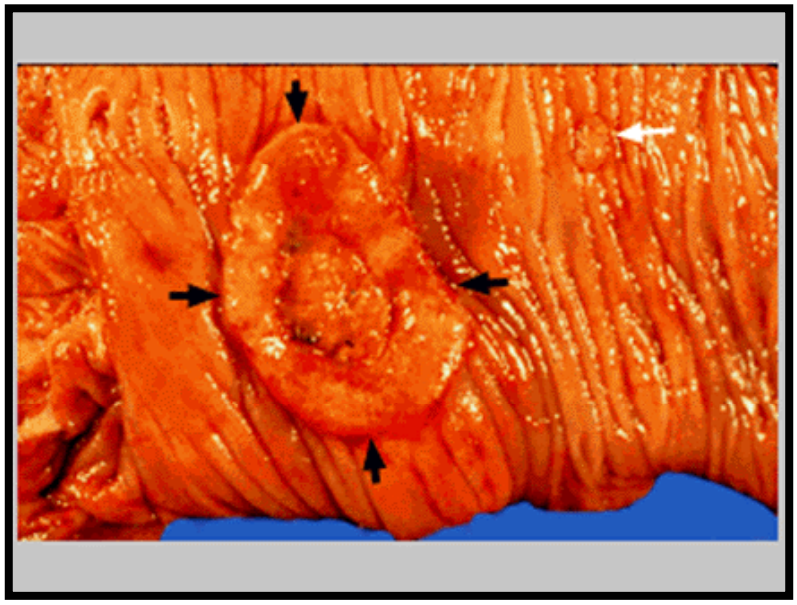
Tubulopapillary carcinoma (De-novo-carcinoma)



This is a de-novo-carcinoma with a diameter of 1,5 cm. Most carcinomas originate from adenomas (adenoma-carcinoma-sequence). The cause of the de-novo-carcinomas is unknown. Correlations to HNPCC, ulcerative Colitis and non tropical sprue are reported. On the other hand flat adenomas were identified as precursors, thus questioning the hypothesis of de-novo-carcinogenesis. HNPCC = Hereditary Non Polyposis Colorectal Carcinoma

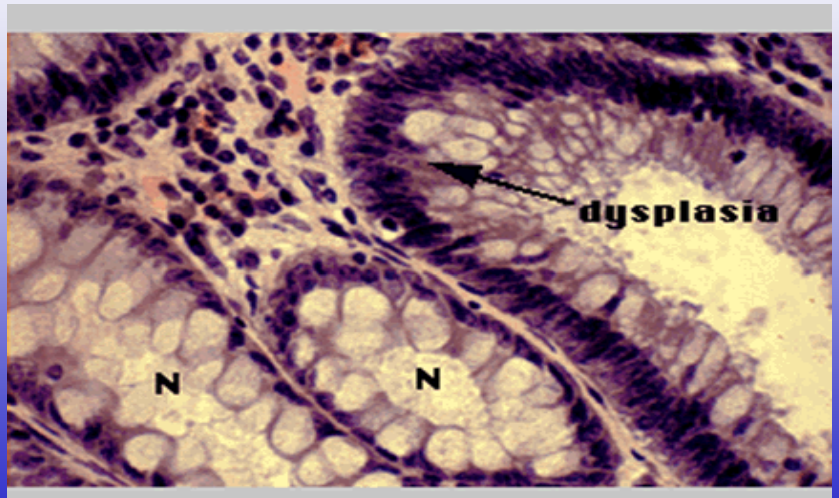


fam_polyposis



coloncagross

DYSPLASIA



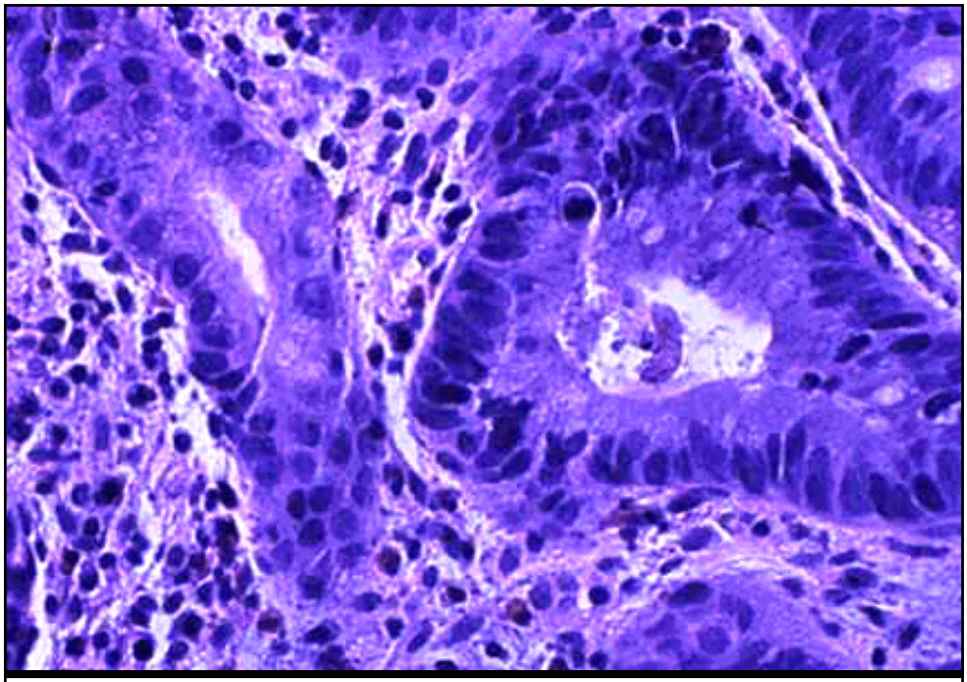
Dysplasia, Colon

Colon - low grade dysplasia in IBD

•N = normal glands with small basally placed nuclei and ample amounts of mucin

•Dysplastic cells have cigar shaped hyperchromatic nuclei.

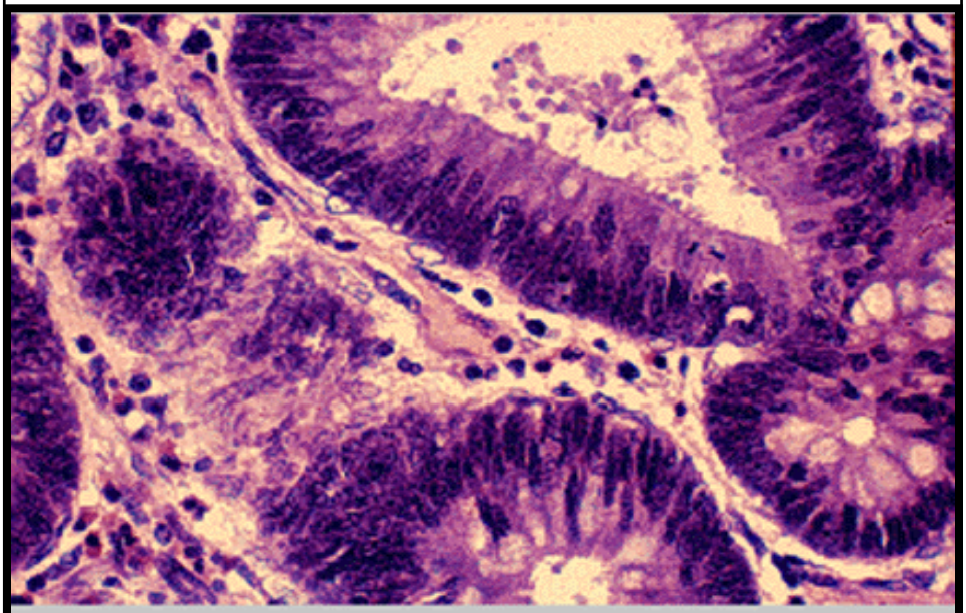
•Lack of severe architectural disarray(pseudopalisading) and continued mucin production distinguish this from high grade dysplasia.



dysplasia_in_UC2

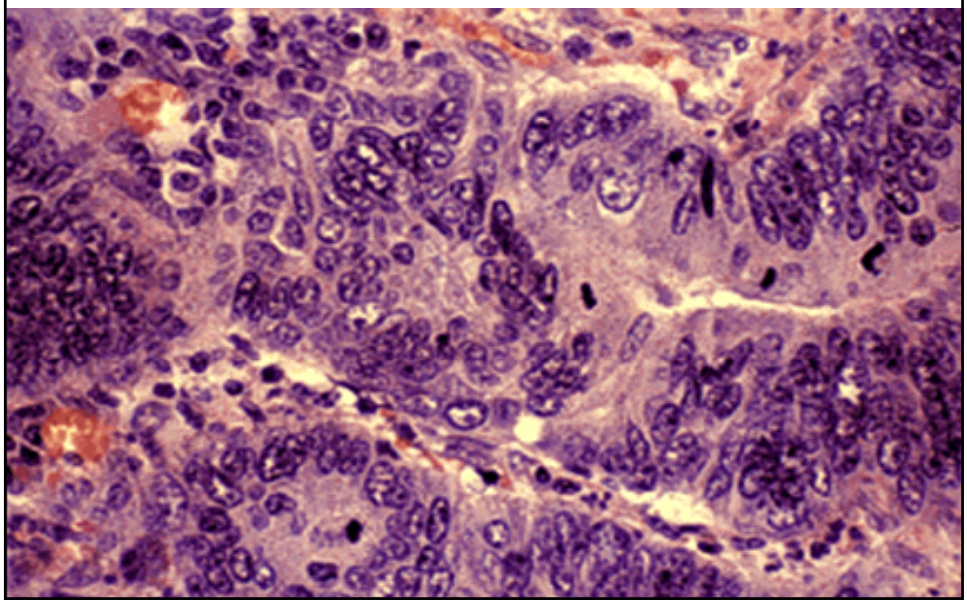
Colon - low grade dysplasia in IBD

- **Nuclear changes: hyperchromasia, cigar shaped nuclei.**
- **Functional changes: decrease in mucin production.**
- **Architectural changes: early loss of polarity with pseudopalisading**

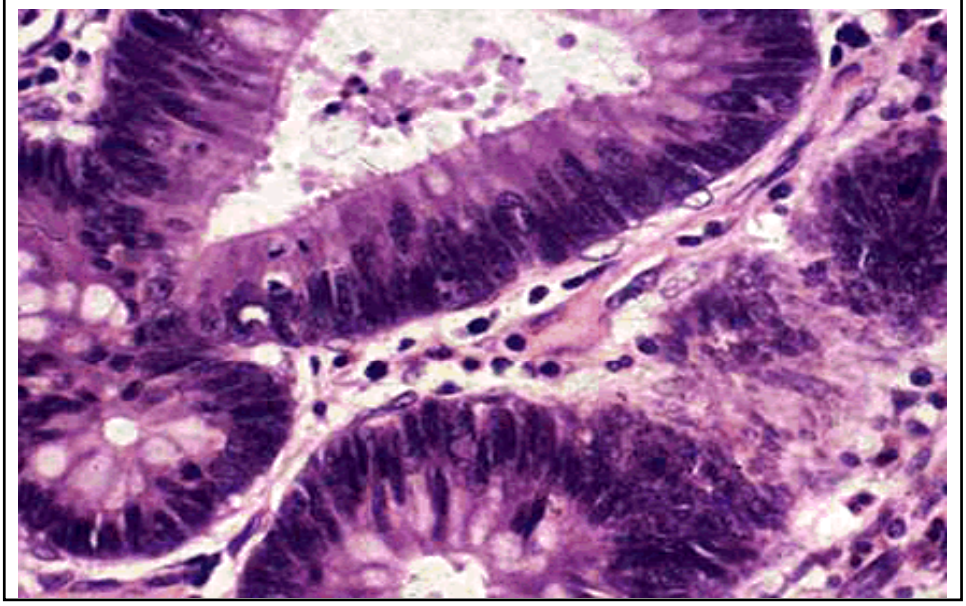


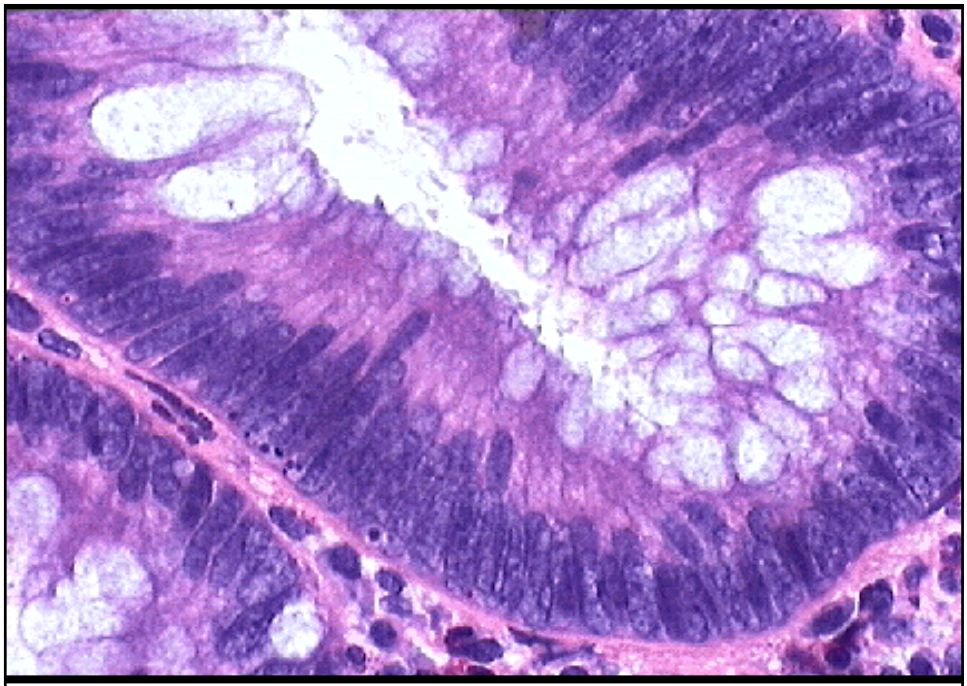
Dysplasia in IBD

HIGH GRADE DYSPLASIA IN CASE OF IBD



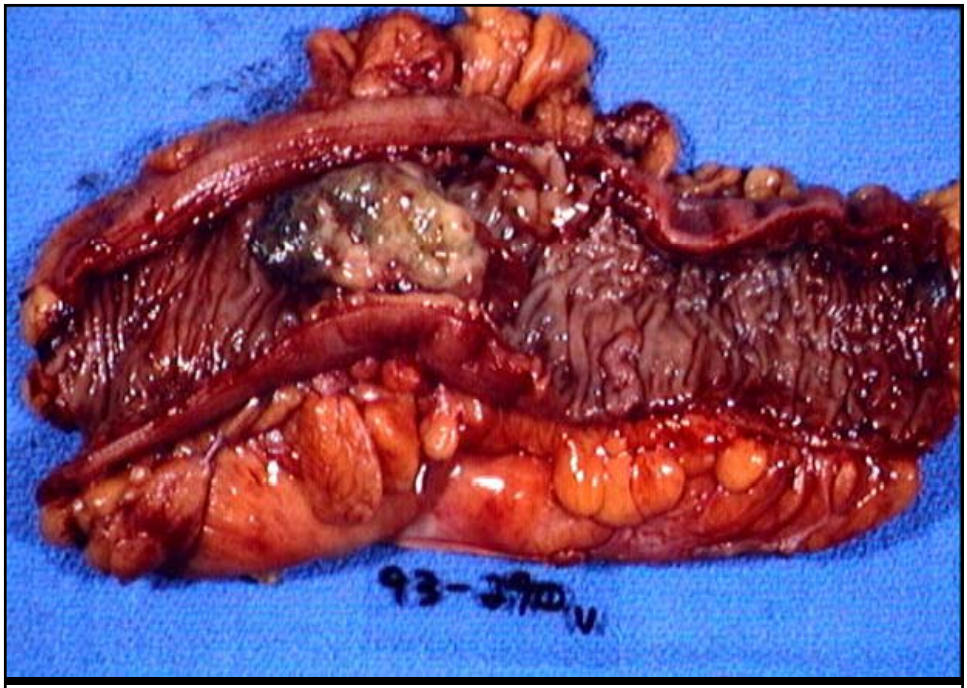
LOW GRADE DYSPLASIA



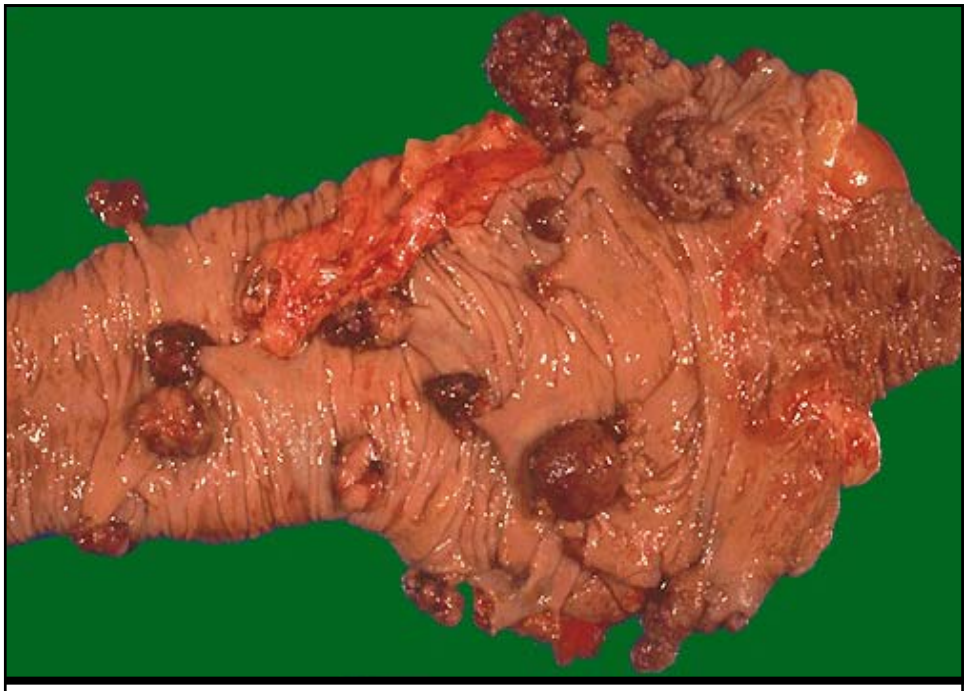


dysplastic_adenoma

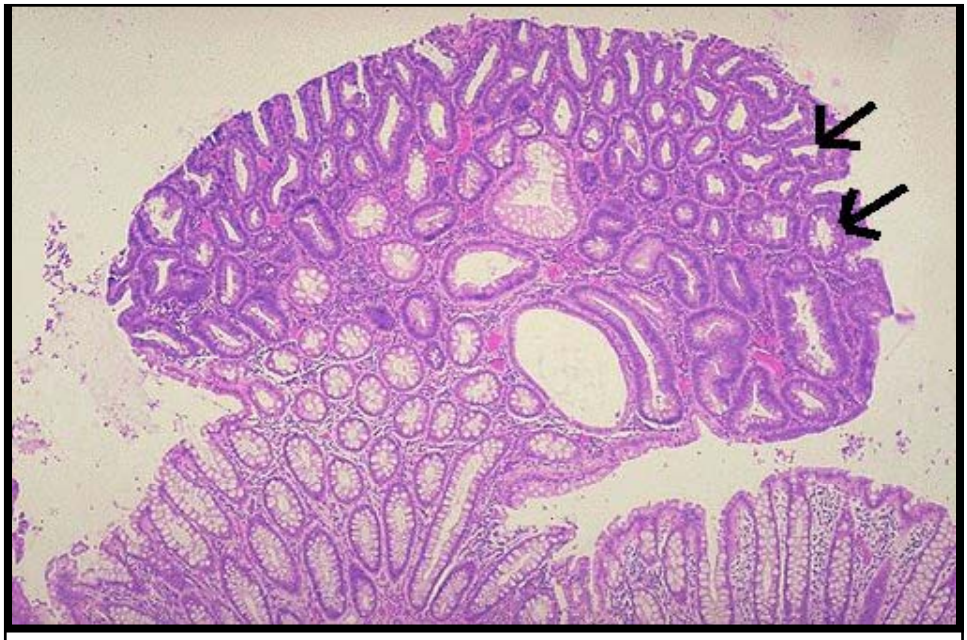
MORE VARIOUS PICTURES OF COLON TUMORS



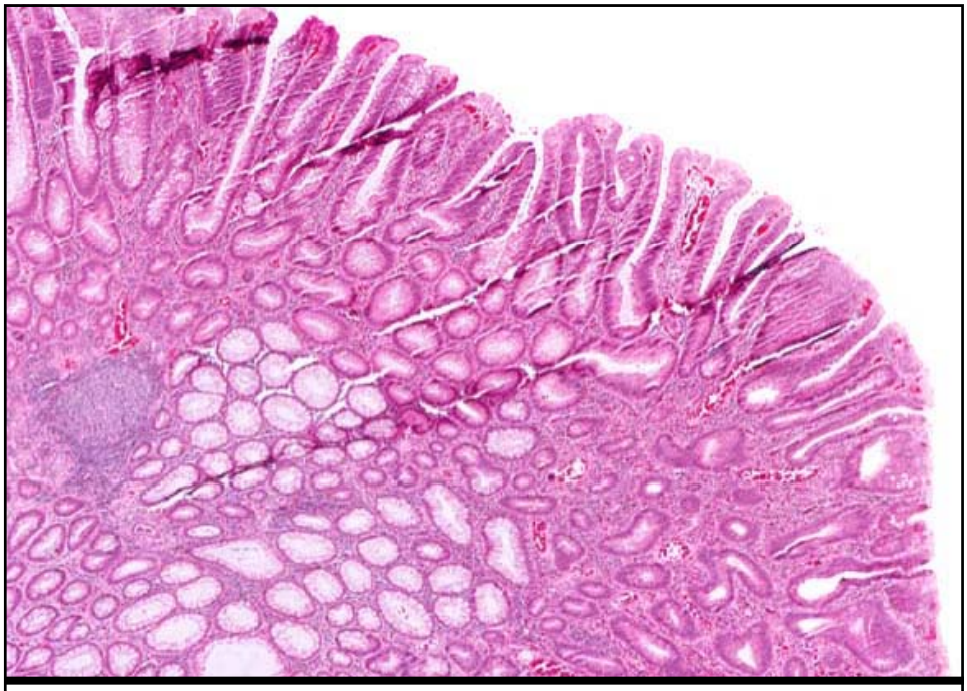
gcolon4



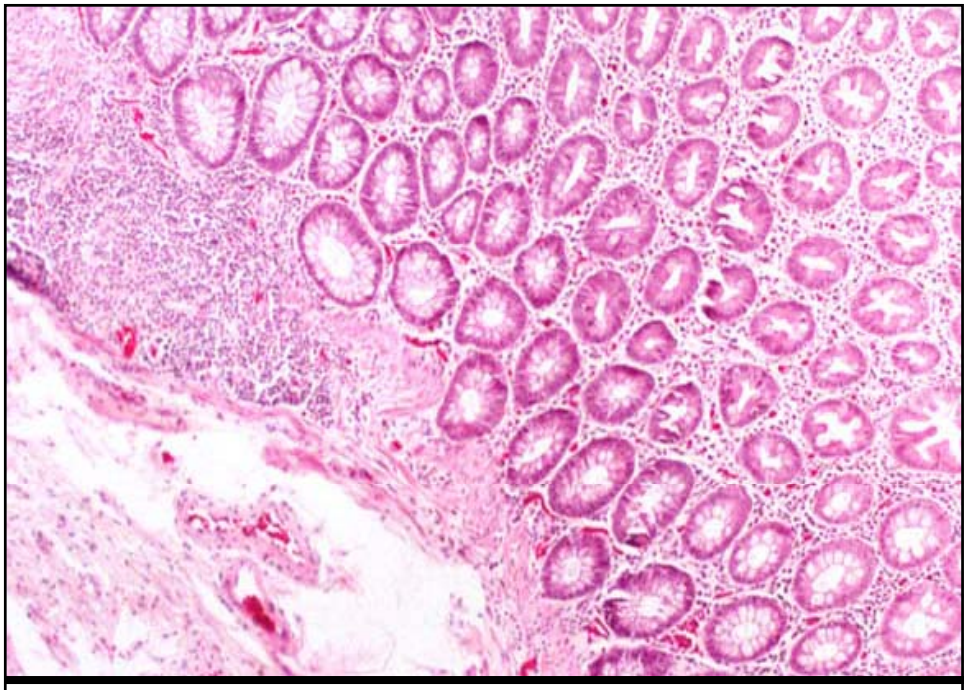
GI114



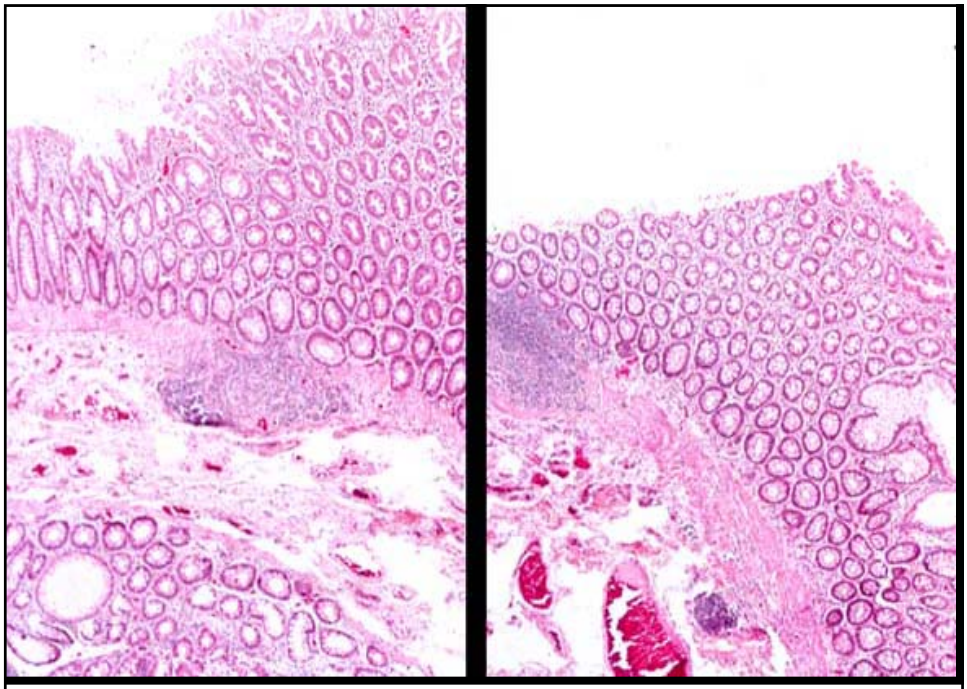
polyp8



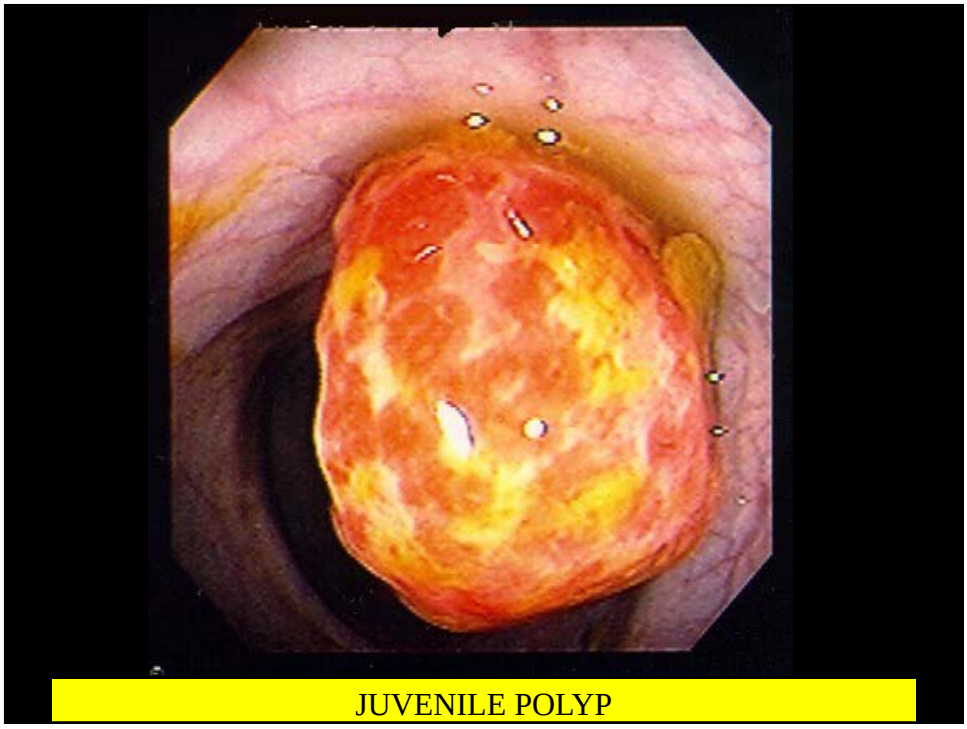
hyperplastic_polyp



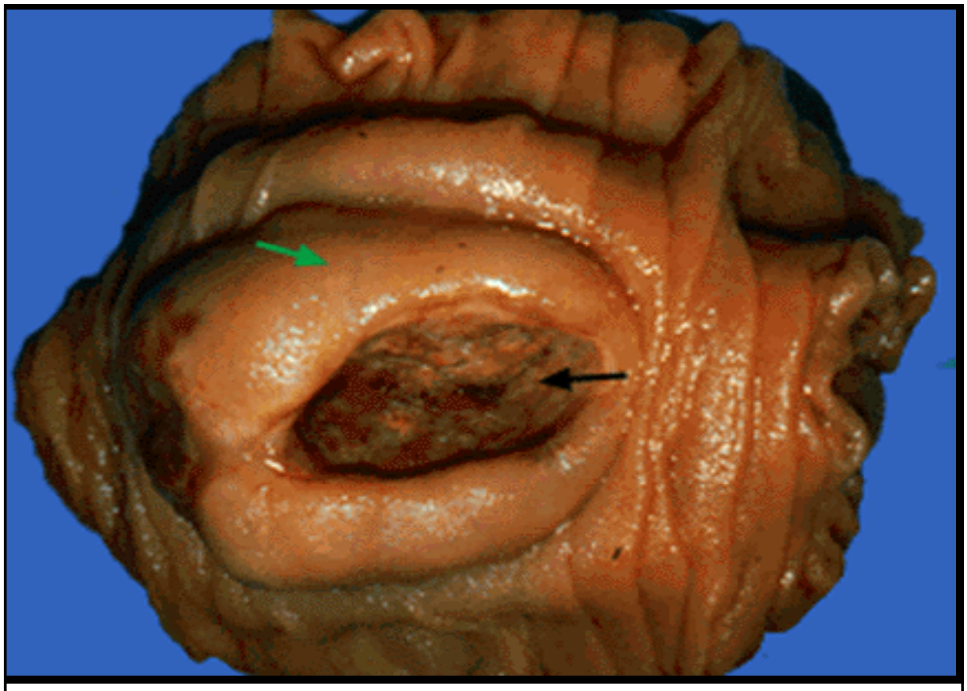
hyperplastic_polyp_2



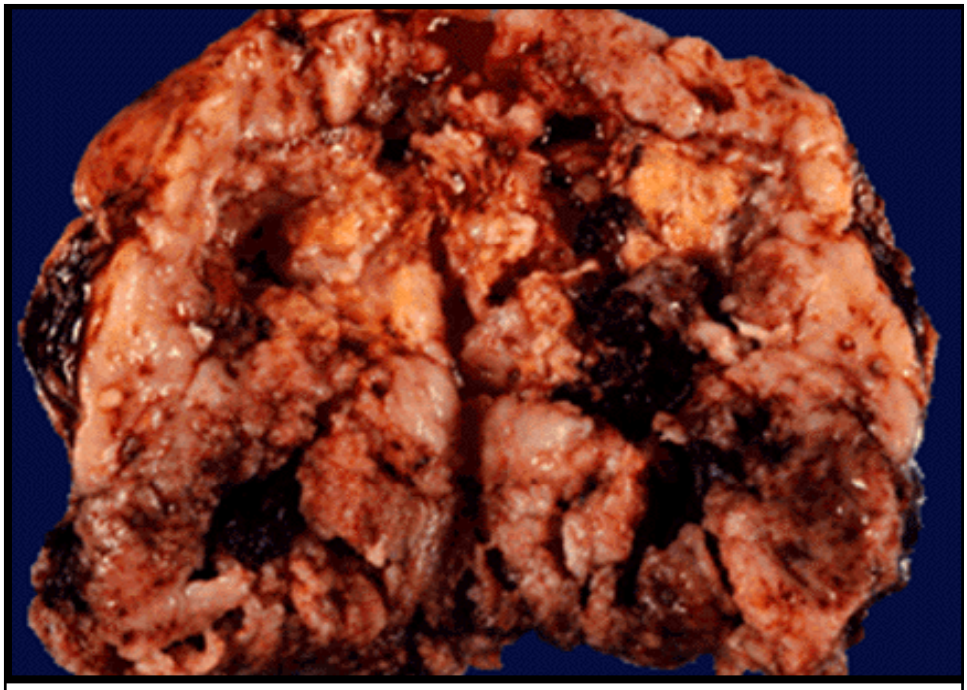
hyperplastic_polyp_vs_adenoma



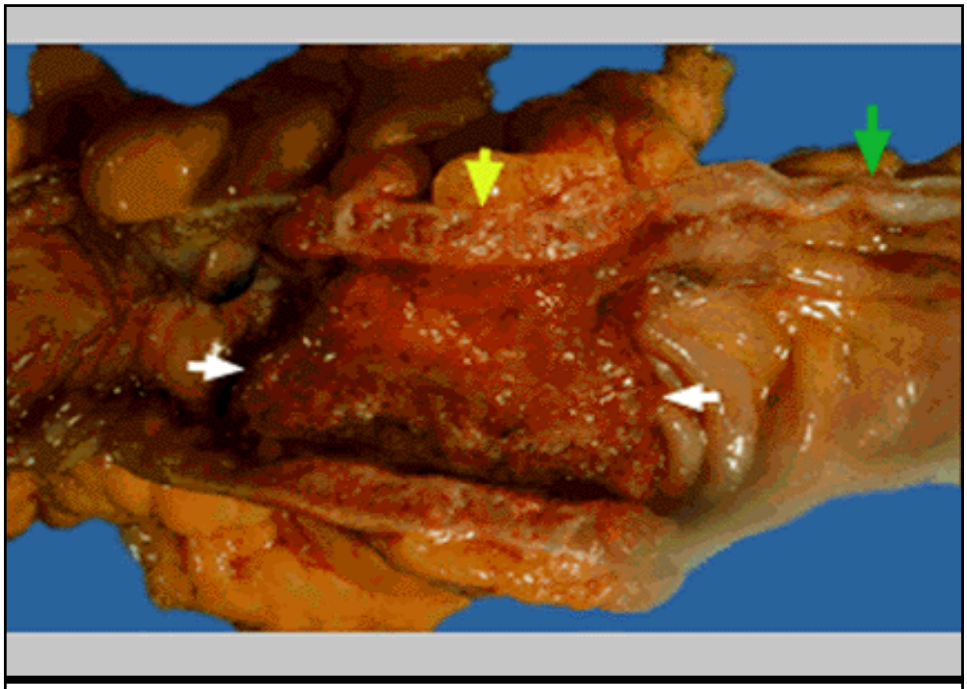
JUVENILE POLYP



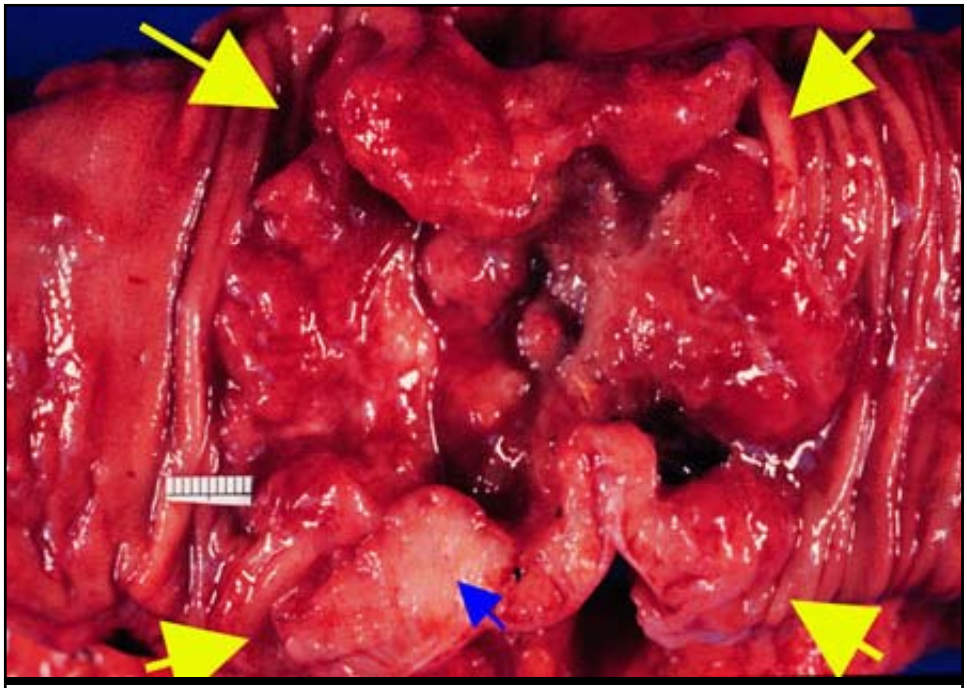
leiomyosarcoma



leiomyosarcoma2



napkinring



napkinringca

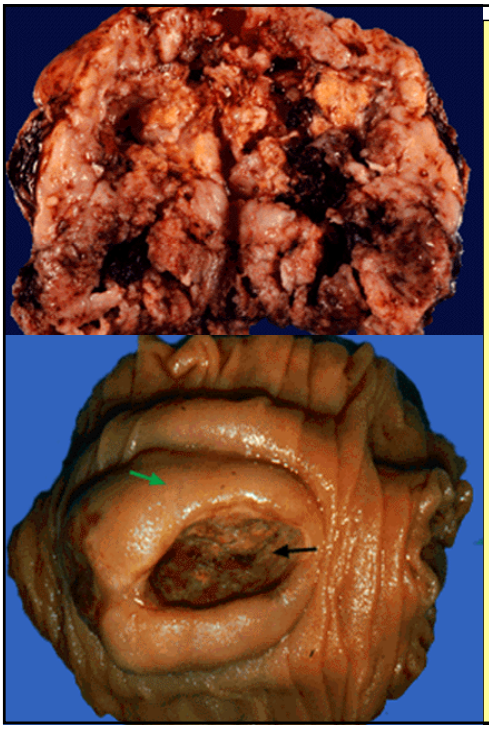
pedunculated

Metaplastic polyps

Small plaques approximately 2 mm in diameter

Pathogenesis unknown

Not pre-malignant



Leiomyosarcoma, Colon
Leiomyosarcoma of Colon
•The white rubbery cut surface is typical of a GIST tumor (Gastrointestinal Stromal Tumor)
•The large size of this tumor suggests malignant potential, but is not as accurate as mitotic counts as
an aid in classifying these tumors as benign or malignant •Necrosis and hemorrhage are found in malignant tumors, but in GIST tumors are related to the size

of the tumor and are not an independant predictor of malignant potential

Peutz-Jeghers syndrome

Peutz-Jegher's syndrome

Peutz-Jegher's syndrome is an autosomal dominant condition characterised by:

multiple hamartogenous polyps of the gastrointestinal tract - most often in the small bowel but may occur affect any portion of the GI tract
mucocutaneous pigmentation - mainly, of the lips, buccal mucosa, genitalia, hands and feet

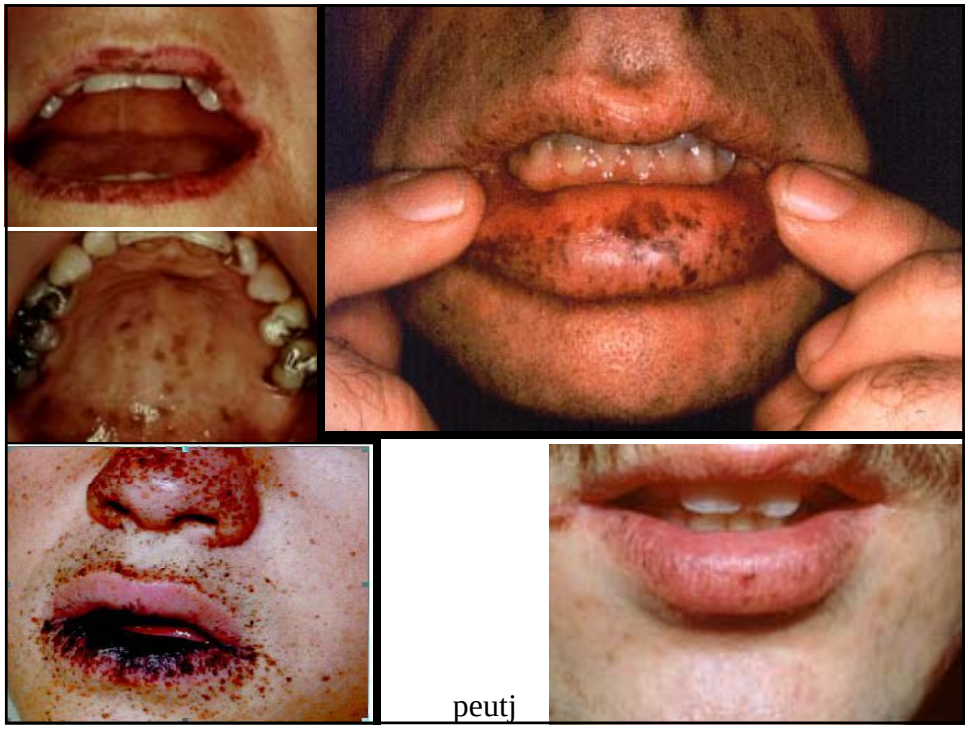
Patients often present with small bowel intussusception before the age of 10 years.

The polyps themselves have a very low malignant potential. About 10-20% of patients develop gastrointestinal carcinoma but this is thought to arise from coexistent adenomas.

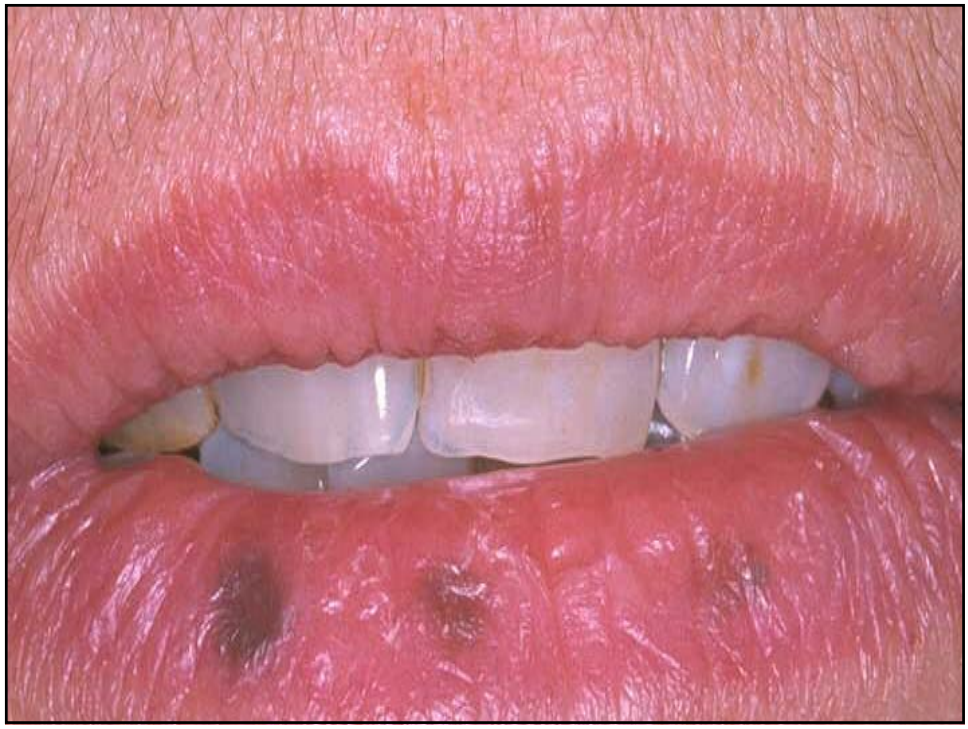
Patients have an increased risk of developing carcinomas of the pancreas, lung, ovary and breast.

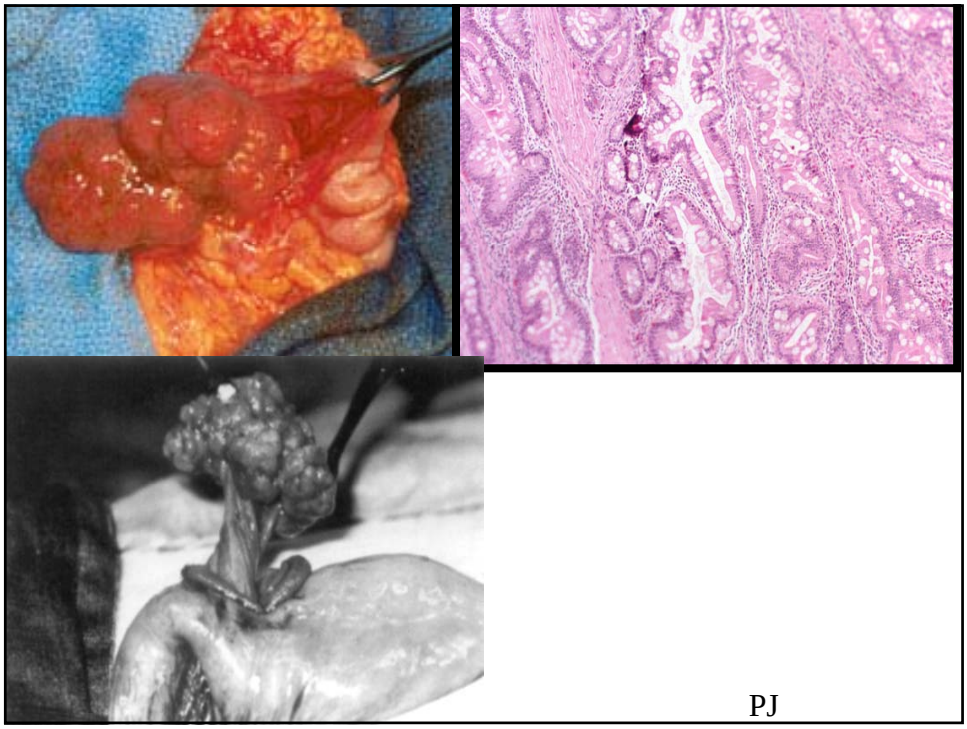
Peutz-Jeghers syndrome

Rare familial disorder
Circumoral pigmentation and intestinal polyps
Polyps found throughout gut but most common in the small intestine
Presents in childhood with bleeding, anemia or intussusception
Polyps can become malignant

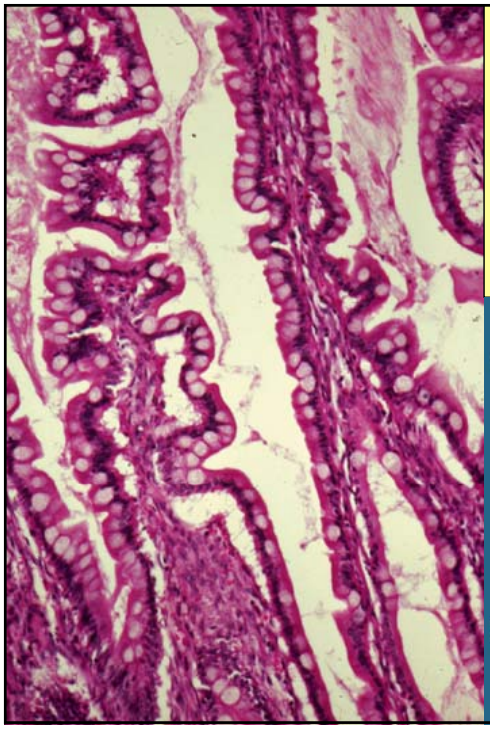


peutj



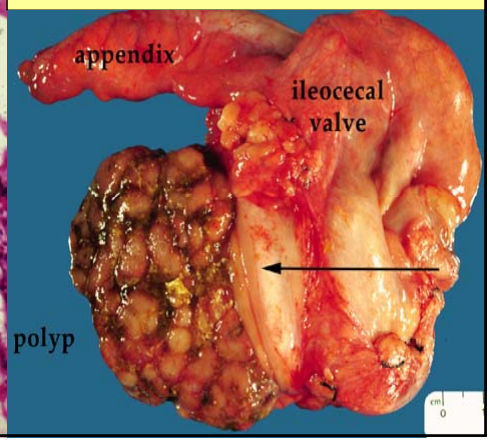


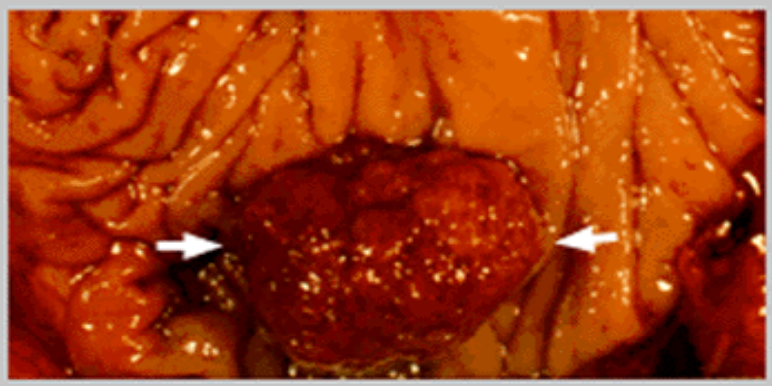
PJ



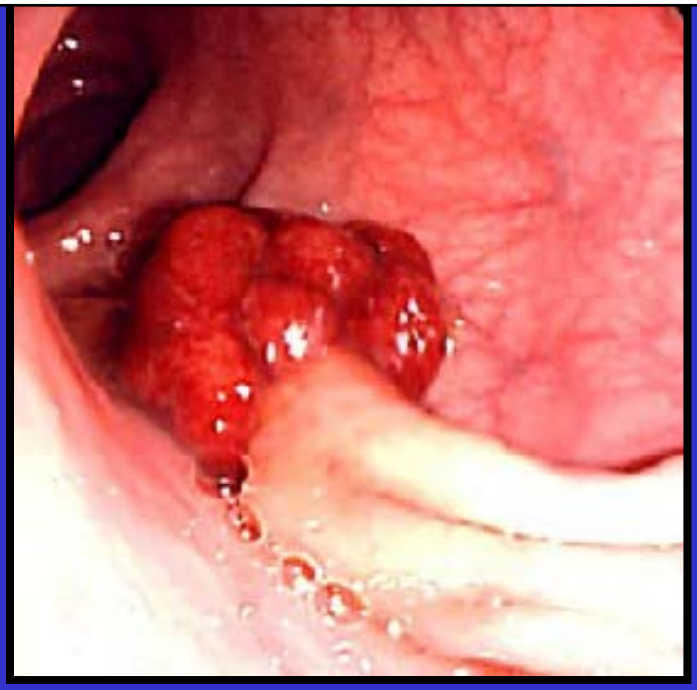
High-power photomicrograph of villous fronds in a Peutz-Jegher polyp

The Peutz-Jegher polyp is an overgrowth of normal tissue. Thus, the Peutz-Jegher polyp is a true hamartomatous polyp. The polyp shown here comes from small intestine, and so the epithelial compartment consists of a small-bowel type of mucosa with villous fronds and a mixture of goblet cells and absorptive cells over the villi.

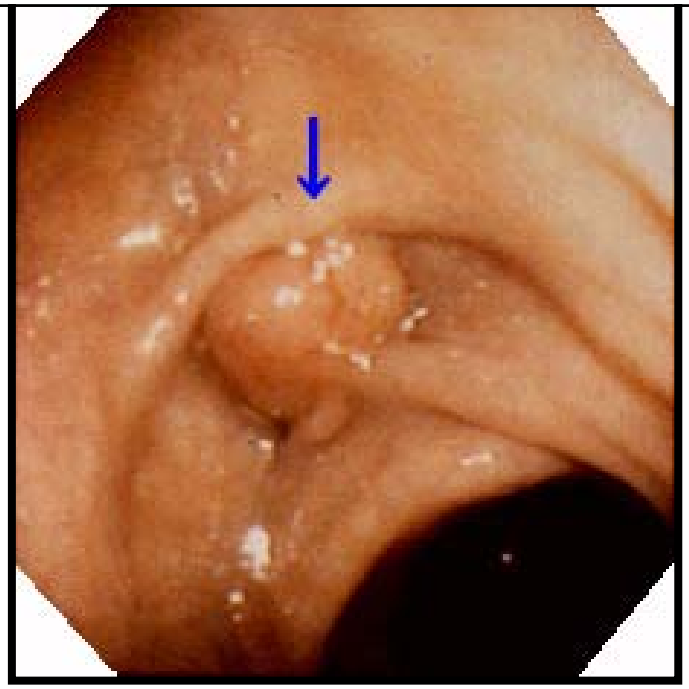




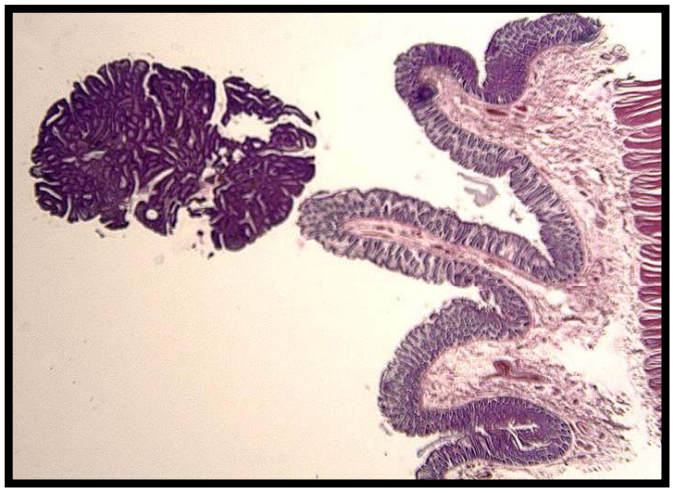
polyp_close



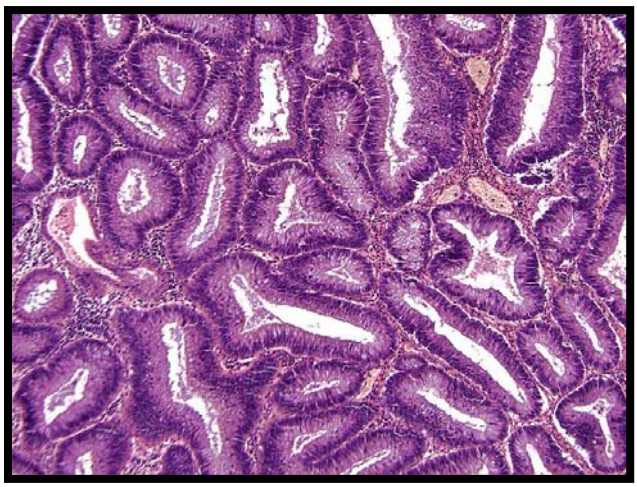
polyp4



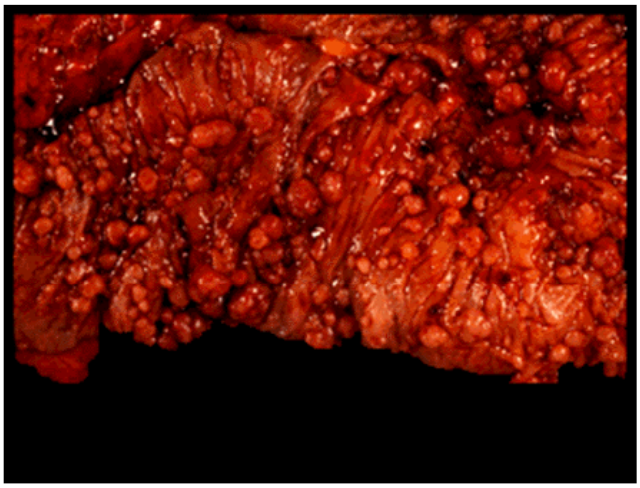
polyp6



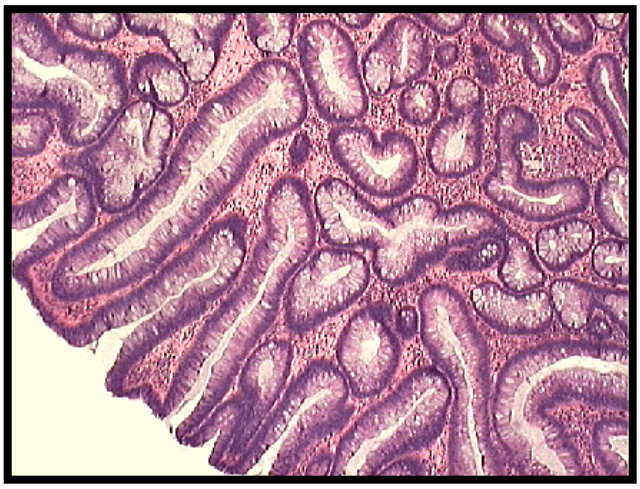
tubular_adenoma_colon



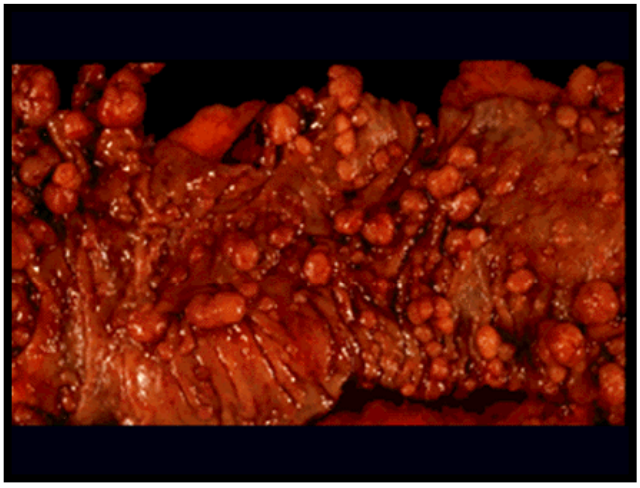
tubular_adenoma_colon_2



polyposis_coli



tubulovillous



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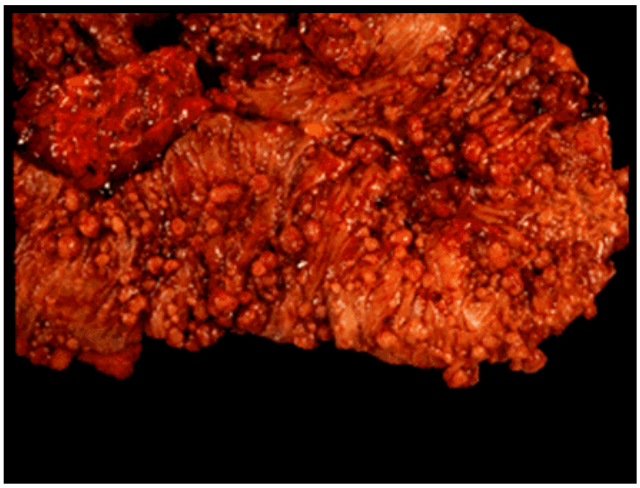

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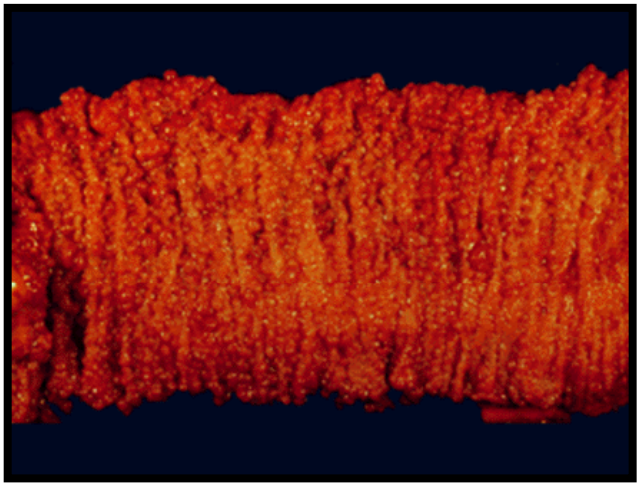

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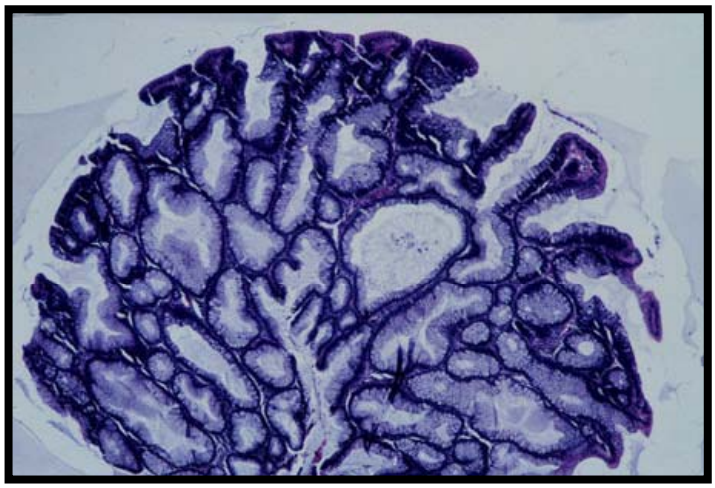
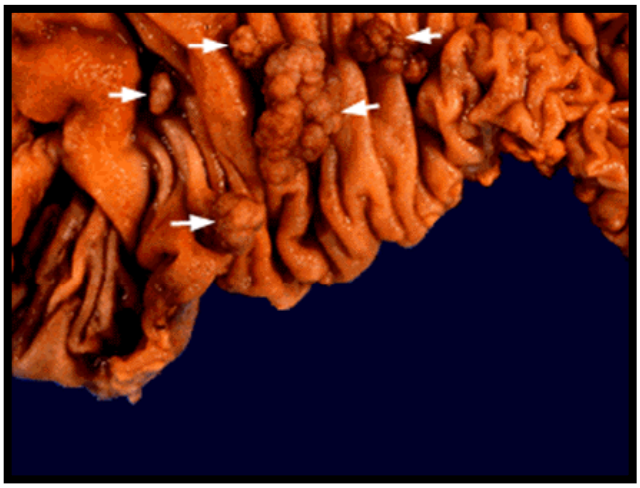
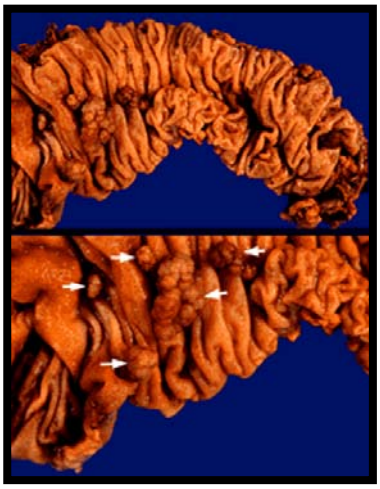


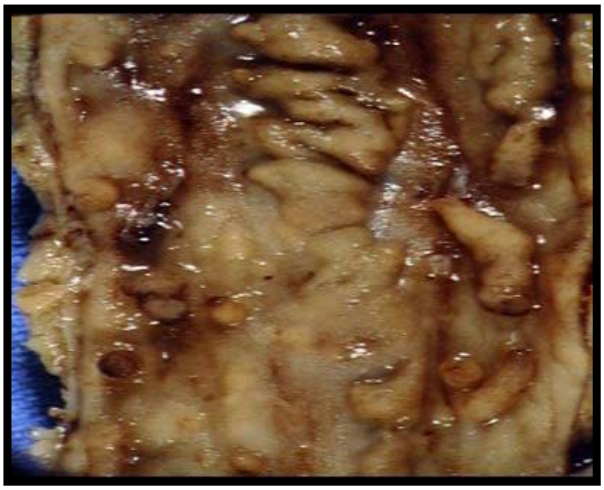
tubulovillous_indiana



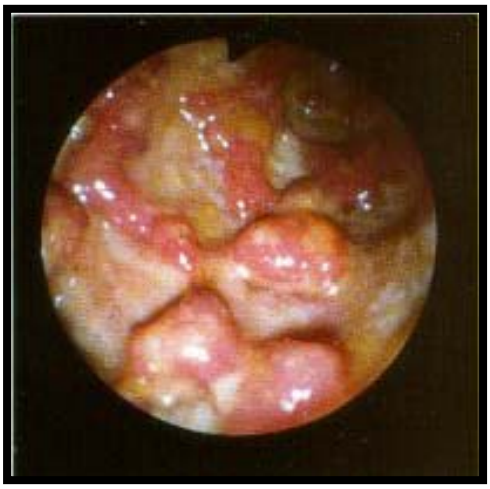
polyps



polyps2



pseudopolyps



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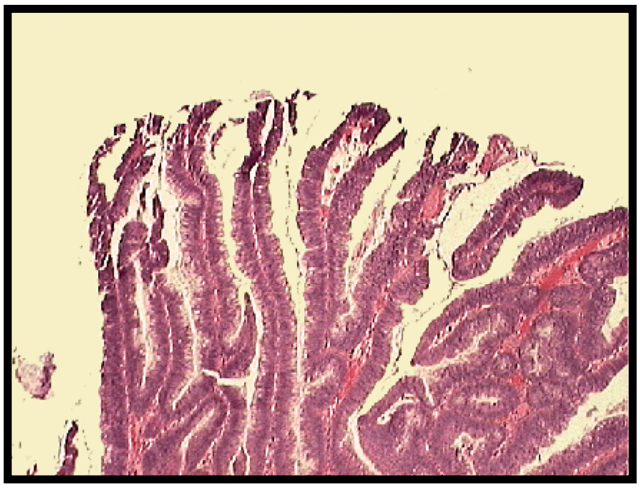
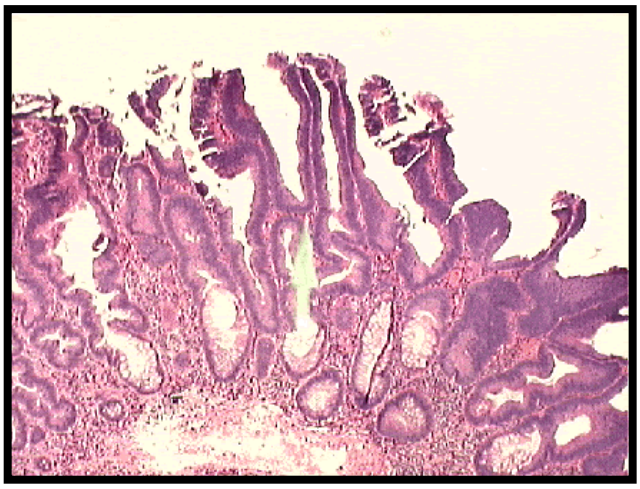
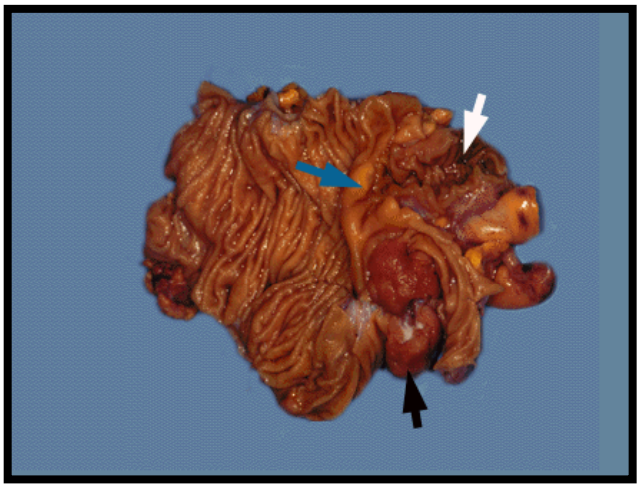


villous_adenoma6



villous_adenoma5



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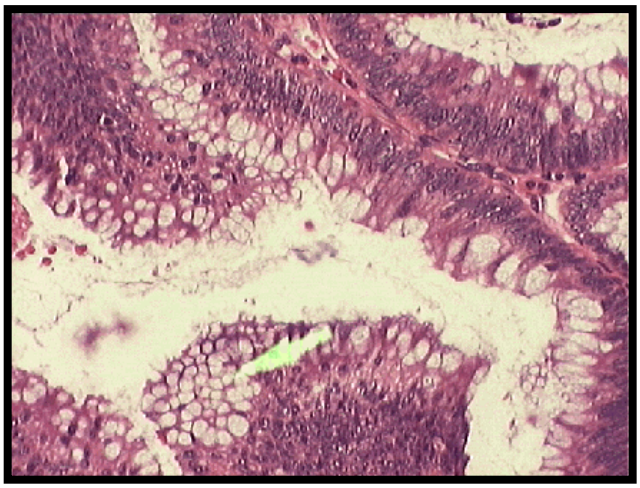

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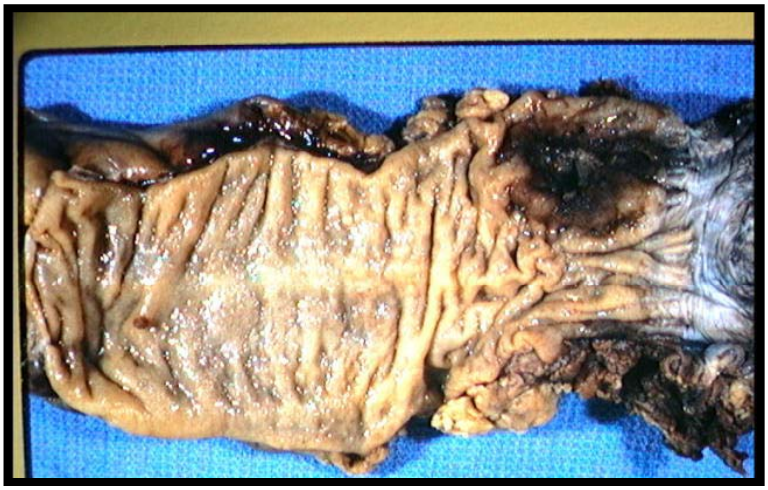
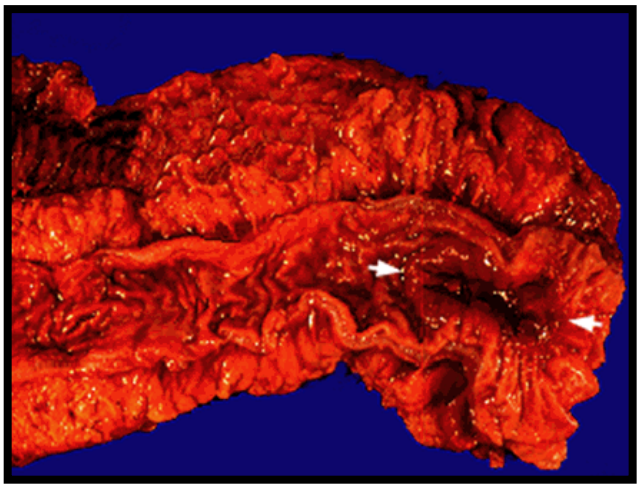


rectalca



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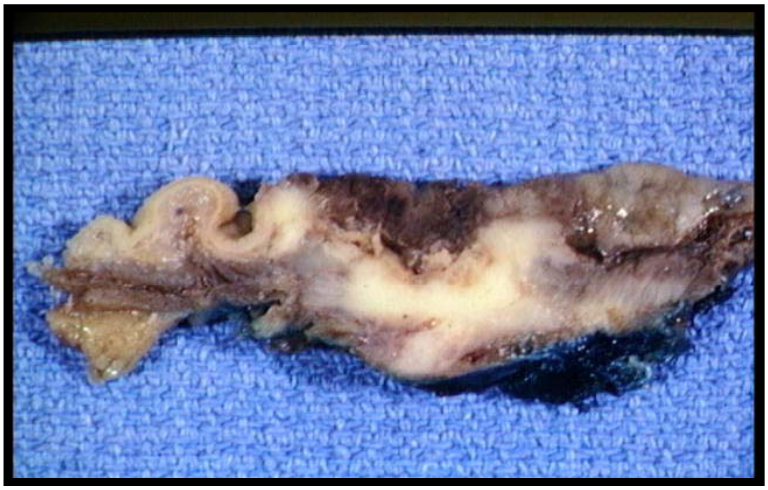

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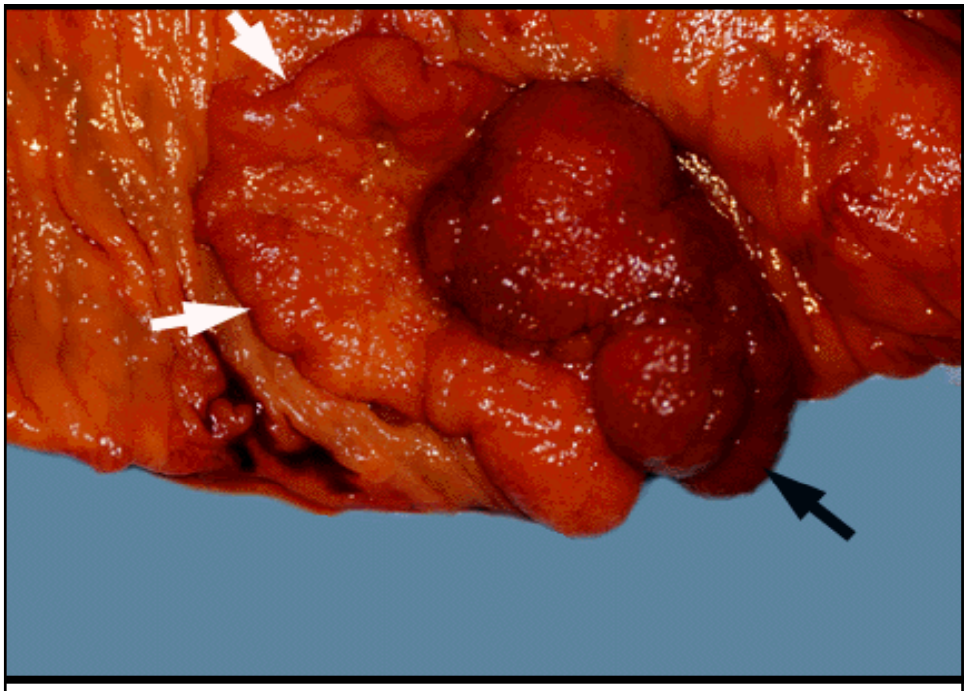
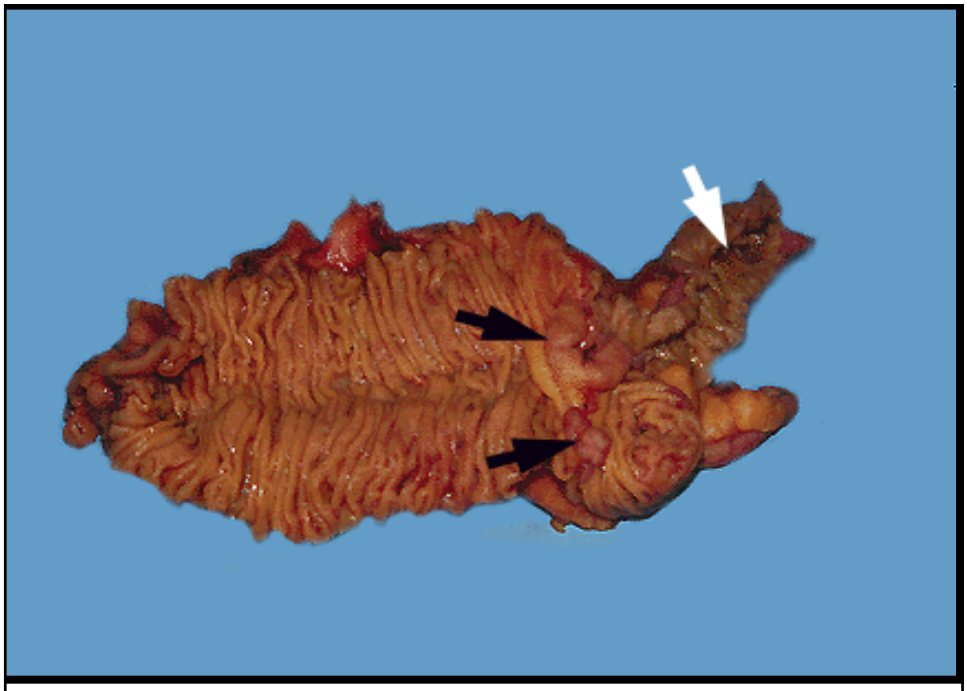
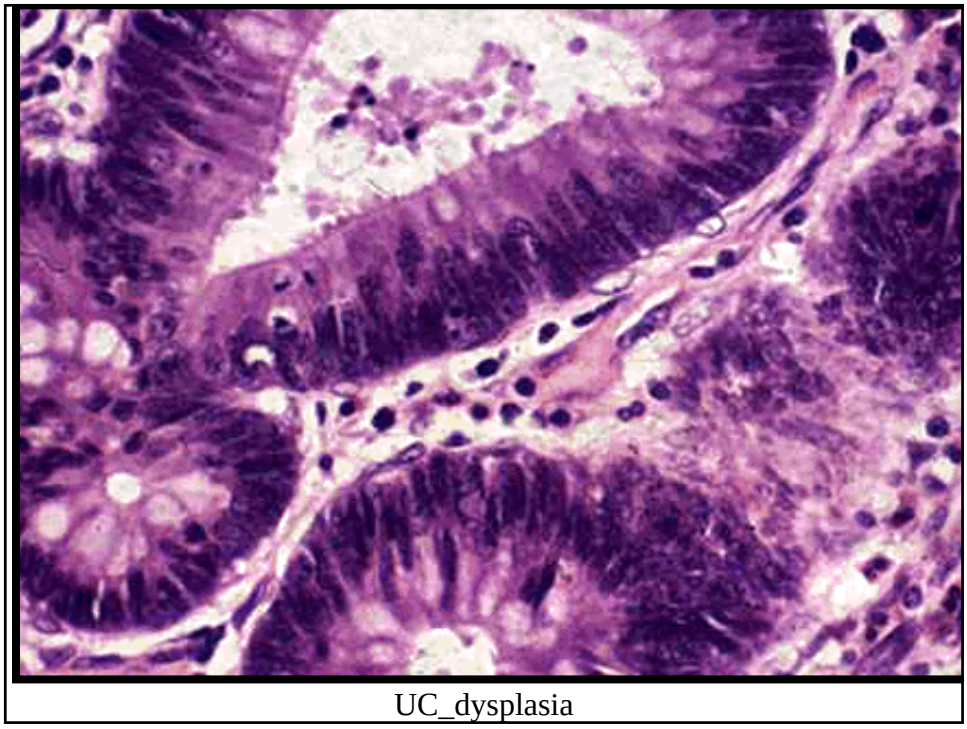


tubulovillous2

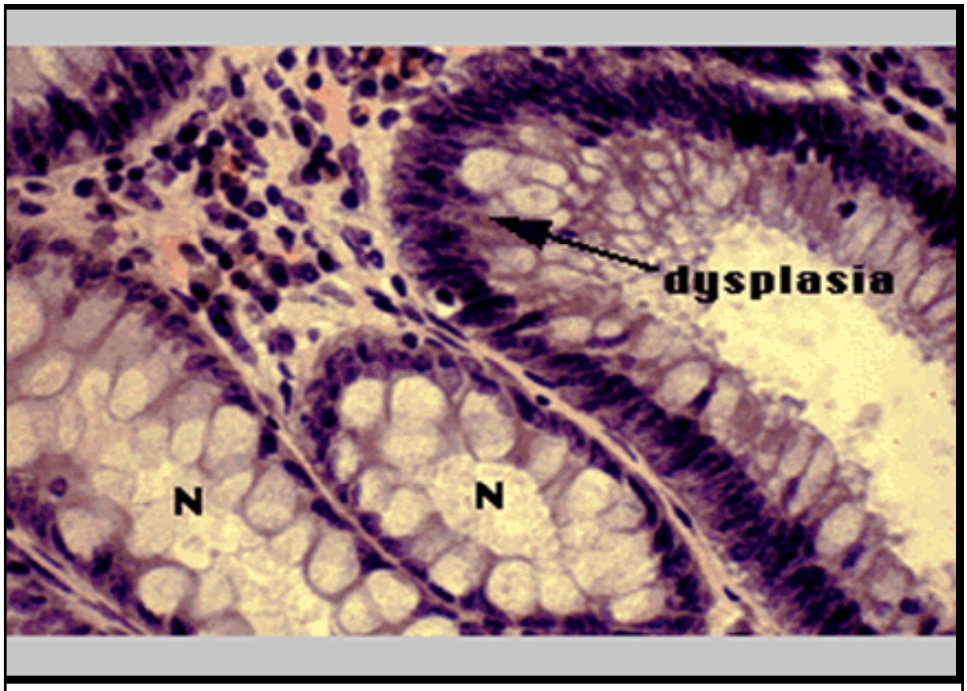
tvp_enface



two_polyps



UC_dysplasia



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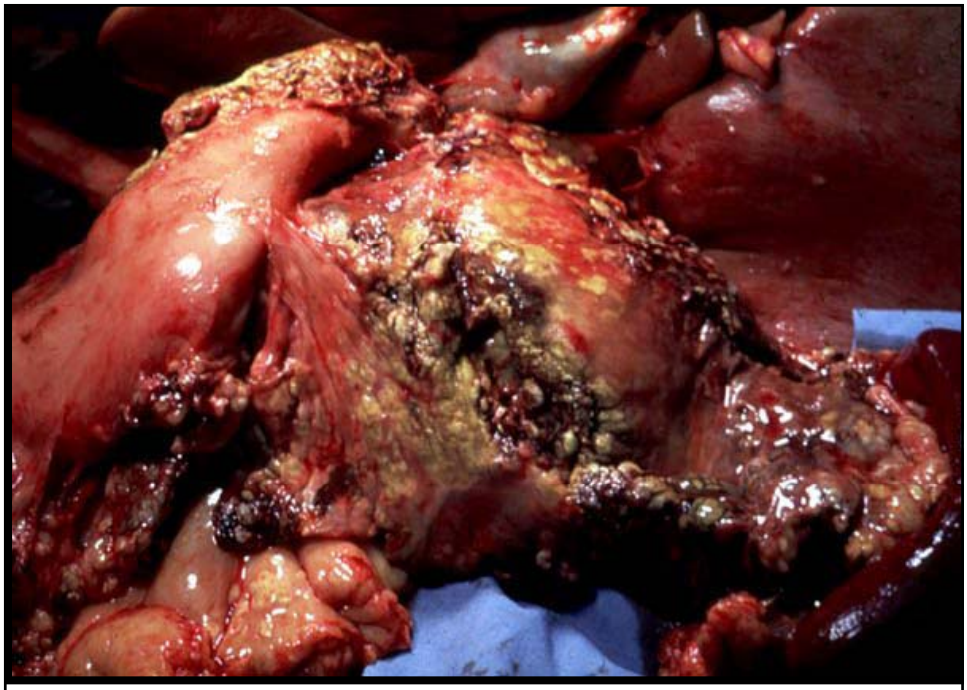
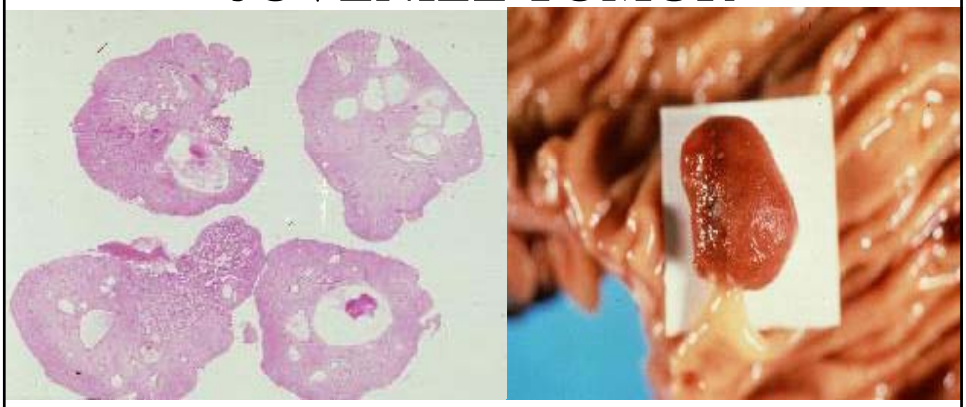


PERFORATED CARCINOMA_

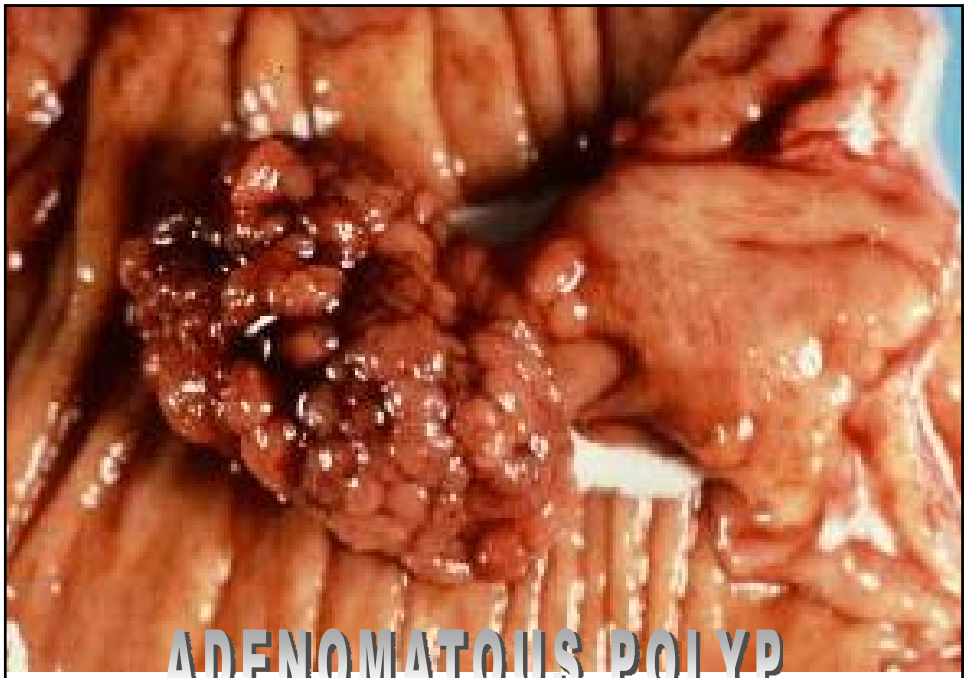
JUVENILE TUMOR



Juvenile polyps are usually non-neoplastic, often outgrow their blood supply, and autoamputate at puberty. Treatment is required only for uncontrollable bleeding or intussusception.
Hyperplastic polyps, also non-neoplastic, are common in the colon and rectum.
Inflammatory polyps and **pseudopolyps** occur in chronic ulcerative colitis and in Crohn's disease of the colon

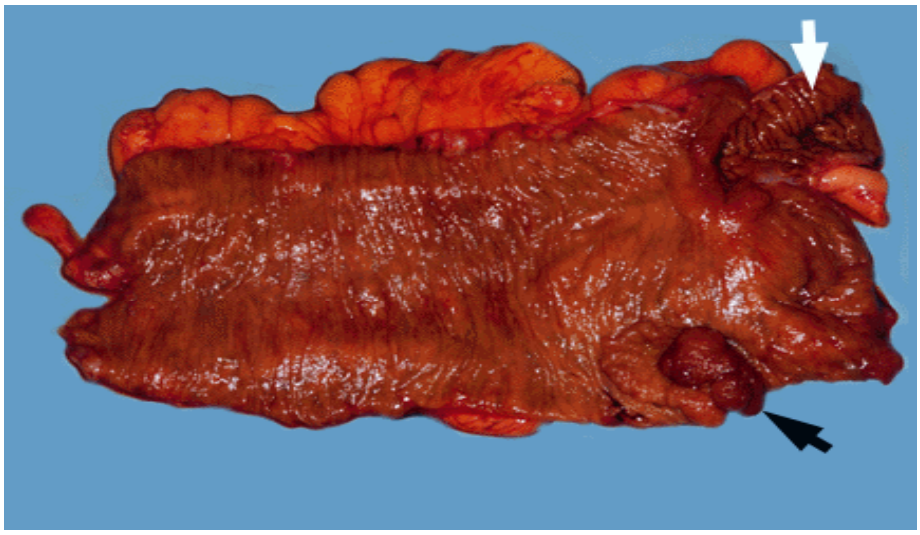
Juvenile polyps

Commonest form of polyp in children
Can occur throughout large bowel but are most common in the rectum
Usually present before 12 years
Present with Prolapsing lump or rectal bleeding
Not pre-malignant
Treated by local endoscopic resection



Adenoma, Tubulovillous, Colon

Tubulovillous Adenoma of Colon



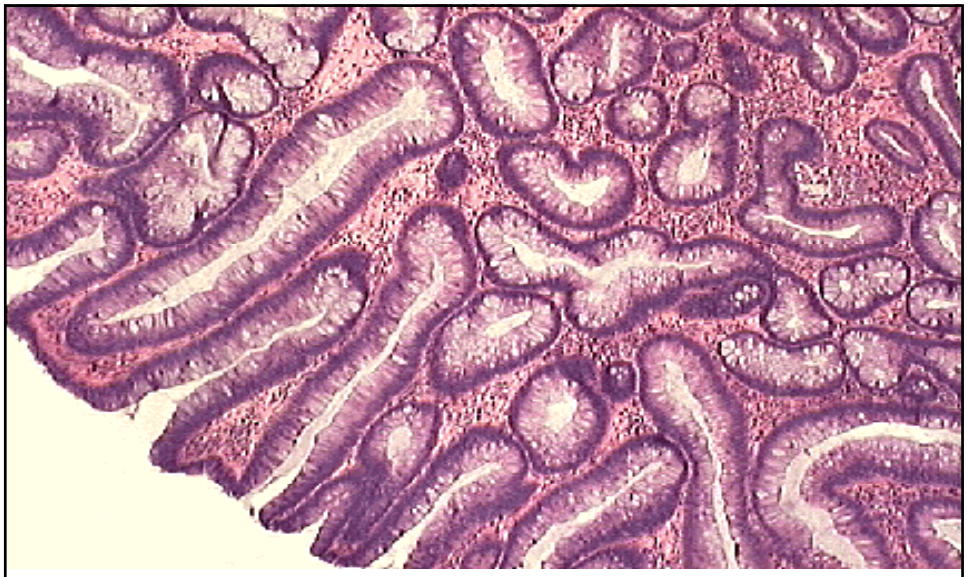
Adenoma, Tubulovillous (Colon)	
Etiology	•The dysplasia-adenoma-carcinoma sequence occurs in the setting of increasing loss of heterozygosity in genes involved in: DNA replication accuracy(mismatch repair)-Chromosomes 2 and 3; tumor suppression-Chromosomes 5,18, and 17; and oncogene activation-chromosomes 5,17,and 18 •A hereditary predisposition to cancer is found in 1% of colorectal carcinoma patients with the Adenoma Polyposis Coli Syndrome involving Chr.5, and in 5-10% of patients with Hereditary Non-Polyposis(Lynch Syndromes) gene changes on Chr 2 and 3 •For each patient loss of heterogosity must occur in multiple genes
Pathogenesis	•Two pathways are commonly hypothesized to account for the known environmental, dietary and genetic predispositions to colorectal carcinoma. Both eventuate in loss of gene heterozygosity •The first of these postulate mucosal damage either through dietary induction of increased bile acid production or the direct affect of dietary and environmental carcinogens. This leads to increased mucosal cellular proliferative activity and an increase risk for gene match failure •The second postulates a direct genotoxic affect possibly mediated through production of oxygen free radicals •As increased numbers of defective gene growth regulators are formed, increased abnormal cellular activity eventuates in carcinoma,
Epidemiology	•Tubulovillous adenomas have the same epidemiology as colorectal carcinoma but occur at an earlier age
General Gross Description	•Tubulovillous adenomas are polyps and may be pedunculated or have a broad base •They are generally not as sessile as true villous adenomas •Their surface may have a fissured appearance similar to tubular adenomas, or they may appear more granular •The surface is red brown and benignancy can not be predicted from their gross appearance
General Microscopic Description	
Clinical Correlation	•Tubulovillous polyps are defined as adenomatous polyps with a villous component of 25-50% •Their epithelium is identical to other adenomatous polyps, and the diagnosis is purely a pattern diagnosis

Adenoma, Tubulovillous, Colon

Tubulovillous Adenoma

The black arrow marks a partly sessile polypoid lesion which on gross examination can not be distinguished from a carcinoma

•The white arrows mark the ileocecal valve



Adenoma, Tubulovillous, Colon

Tubulovillus Adenoma (4X)

•Since the lesion has glands lined by dysplastic cells it is an adenoma
•The surface has papillary features and the remainder is tubular
•When between 25-50% of the epithelium is papillary and the rest tubular, these lesions are called tubulovillous adenomas

RECTAL TUMORS

Adenocarcinoma (Colon)	
Etiology	•The dysplasia-adenoma-carcinoma sequence occurs in the setting of increasing loss of heterozygosity in genes involved in: DNA replication accuracy (mismatch repair)-Chromosomes 2 and 3; tumor suppression-Chromosomes 5,18, and 17; and oncogene activation-chromosomes 5,17,and 18 •A hereditary predisposition to cancer is found in 1% of colorectal carcinoma patients with the Adenoma Polyposis Coli Syndrome involving Chr.5, and in 5-10% of patients with Hereditary Non-Polyposis (Lynch Syndromes) gene changes on Chr 2 and 3 •For each patient loss of heterogosity must occur in multiple genes
Pathogenesis	•Two pathways are commonly hypothesized to account for the known environmental, dietary and genetic predispositions to colorectal carcinoma. Both eventuate in loss of gene heterozygosity •The first of these postulate mucosal damage either through dietary induction of increased bile acid production or the direct affect of dietary and environmental carcinogens. This leads to increased mucosal cellular proliferative activity and an increase risk for gene match failure •The second postulates a direct genotoxic affect possibly mediated through production of oxygen free radicals •As increased numbers of defective gene growth regulators are formed, increased abnormal cellular activity eventuates in carcinoma,
Epidemiology	•Colorectal carcinoma is a disease of the older population except for people with hereditary non-polyposis and polyposis syndromes or chronic inflammatory bowel disease •The male/female ratio for rectal carcinoma is 2/1 while the male/female ratio of right sided lesions is 1/1 •The remarkably higher incidence in more affluent countries and the change in incidence in migrants to the area of migration suggests a strong environmental affect which most studies relate to high dietary fat, low fiber and high refined carbohydrates

General Gross Description	•The gross appearance is dependent on the stage of the tumor •Early invasive carcinoma may maintain the appearance of the original adenoma either polypoid or sessile •More commonly, the tumor has obliterated evidence of the underlying adenoma and when first seen is a firm, white, flat, well demarcated mucosal lesion with raised rolled margins, often with central ulceration •Over time, the lesion spreads circumferentially through circular lymphatics to produce a constricting napkin-ring lesion in the mucosa •As mucosal spread occurs, tumor also invades the full thickness of the muscular wall, and only at this stage is tumor seen grossly involving the perirectal fat, or mesentery • Cecal lesions often have a different appearance because of the large volume of space in which they can grow before producing symptoms. Cecal carcinomas often cover large areas of the cecum with fungating sessile or bulky lesions which can be extensively necrotic •Because of their long growth time before discovery cecal lesions often are seen as large deeply invasive tumors with attachment to the adjacent peritoneal wall
General Microscopic Description	•Most colorectal carcinomas have a characteristic appearance which facilitates their identification in metastatic as well as primary lesions •Most lesions are at least moderately differentiated with gland formation and a lining of tall columnar cells with palisading large oval or pencil shaped nuclei with increased coarse chromatin, easily found mitoses and some mucin production •The epithelium bears a clear resemblance to the dysplastic epithelium which covers adenomas •Mucin lakes are found in 10-15% of tumors and these patients are often those with hereditary non-polyposis syndromes or ulcerative colitis •Neuroendocrine differentiation can be identified in 10% of cases but true neuroendocrine carcinoma is extremely uncommon
Clinical Correlation	•Colorectal carcinoma develops over a long period of time with an estimated doubling time of almost two years •Presenting symptoms are related to chronic blood loss with the signs and symptoms of iron deficiency anemia, increasing luminal obstruction with change in bowel habits, diameter of stool, variable constipation, diarrhea, and vague abdominal discomfort or pain. A palpable mass lesion is a late finding •Right sided tumors are most associated with blood loss and rarely with obstruction while left sided lesions present with signs of obstruction •Treatment is surgical and prognosis is related to stage at presentation •About 25% of patients will present with metastatic disease, primarily lymph nodes and liver. The overall survival is 50%

Adenocarcinoma of the rectum is a major cause of mortality and morbidity in North America and western Europe.

Rectal cancers are, after colon cancers, the second most common GI carcinoma and have the best prognosis.

The 5-year survival rate is approximately 50%.

Screening for and removing adenomatous polyps may improve survival rates.

Almost all rectal cancers are primary adenocarcinomas

Adenocarcinoma of the rectum arises as an intramucosal epithelial lesion, usually in an adenomatous polyp or gland.

As cancers grow, they invade the muscularis mucosa and lymphatic and vascular structures to involve regional lymph nodes, adjacent structures, and distant sites, especially the liver.

Several factors increase the risk for rectal cancer, including the following:

- •High-fat, low-fiber diet
- •Patient older than 50 years
- •Personal history of colorectal adenoma or carcinoma (3-fold risk)
- •First-degree relative with colorectal cancer (3-fold risk)
- •Familial polyposis coli, Gardner syndrome, and Turcot syndrome (in which all patients without a colectomy develop colorectal carcinoma)
- •Juvenile polyposis syndrome, Peutz-Jeghers syndrome, and Muir syndrome (risk increased slightly)
- •Hereditary nonpolyposis colorectal cancer (as many as 50% of patients are affected)
- •Inflammatory bowel disease
  - •Ulcerative colitis (risk is 30% after 25 y)
  - •Crohn disease (4- to 10-fold risk)

Rectal cancers tend to be symptomatic earlier than colonic tumors. Overt rectal bleeding is more common in rectal than colonic tumors.
A change in bowel habit or symptoms of large bowel obstruction, such as pain and abdominal distension, may be the presenting features in patients with a rectosigmoid or upper rectal tumor.

The primary tumor may be palpable by digital examination of the rectum. Weight loss, jaundice, and ascites are associated with advanced metastatic disease.

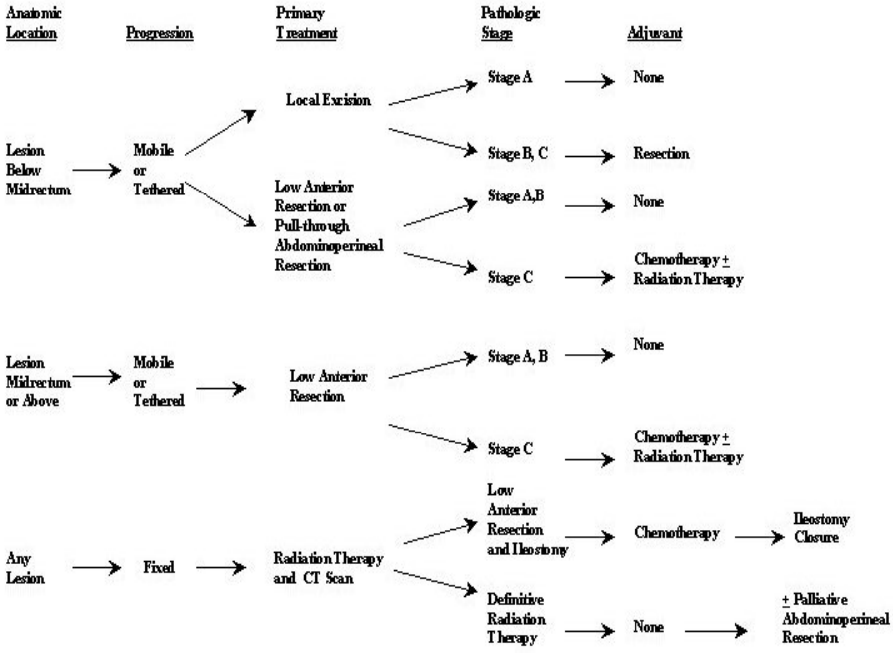
Perforation is rare but may occur as a result of distension proximal to the tumor (usually in the cecum) or locally at the site of the tumor.

Pneumaturia and feculent vaginal discharge may occur as a result of fistula formation into the bladder or vagina.

- •Possibly asymptomatic
- •Palpable mass on digital rectal examination
- •Overt rectal bleeding
- •Microcytic anemia with fatigue, shortness of breath, and angina
- •Vague abdominal discomfort
- •Change in bowel habit
- •Large bowel obstruction
- •Pneumaturia
- •Feculent vaginal discharge
- •Perforation (rare)
- •Weight loss
- •Jaundice
- •Ascites

Evaluation begins with a history and physical examination, including a digital rectal examination.

- •Inspect the stool and test for occult blood.
- •Order blood tests, ie, complete blood count, liver function tests, and carcinoembryonic antigen levels.
- •Perform either sigmoidoscopy (rigid or flexible) or a double-contrast barium enema.
- •Perform CT studies to stage the tumor prior to treatment to choose the most appropriate treatment. Although MRI is slightly more accurate than CT in staging primary rectal tumors, CT is much more widely available. Most institutions and departments have more extensive experience using CT than MRI and continue to use CT for staging rectal tumors. This may change in the future.



•**Sigmoidoscopy:** The 60-cm flexible sigmoidoscope has an increased range over the rigid sigmoidoscope, which at best reaches only to the rectosigmoid junction (20 cm). The sigmoidoscope also is more accurate in the rectum. Sigmoidoscopy detects smaller adenomatous polyps than barium enema; polyps may be excised by this method.

•**Double-contrast barium enema:** Detects most colorectal tumors (80-95%) but should be preceded by flexible sigmoidoscopy. It has a low perforation rate (1/25,000).

•**CT and MRI cannot be used to assess the exact degree of mural invasion of the primary rectal tumor**. These techniques cannot distinguish enlarged lymph nodes resulting from tumor from those resulting from inflammation. Normal-sized nodes containing tumor cannot be detected by either technique.

Local complications of the primary tumor

- •A large bowel obstruction usually results from an annular carcinoma in the upper rectum or rectosigmoid junction.
- •A localized perforation resulting from tumor necrosis may result in a pararectal abscess that simulates an inflammatory process.
- •Perforation also may occur proximal to an obstructing tumor, usually in the cecum.
- •Local invasion of adjacent organs (bladder, uterus, vagina) and fistula formation are late manifestations.

Synchronous lesions

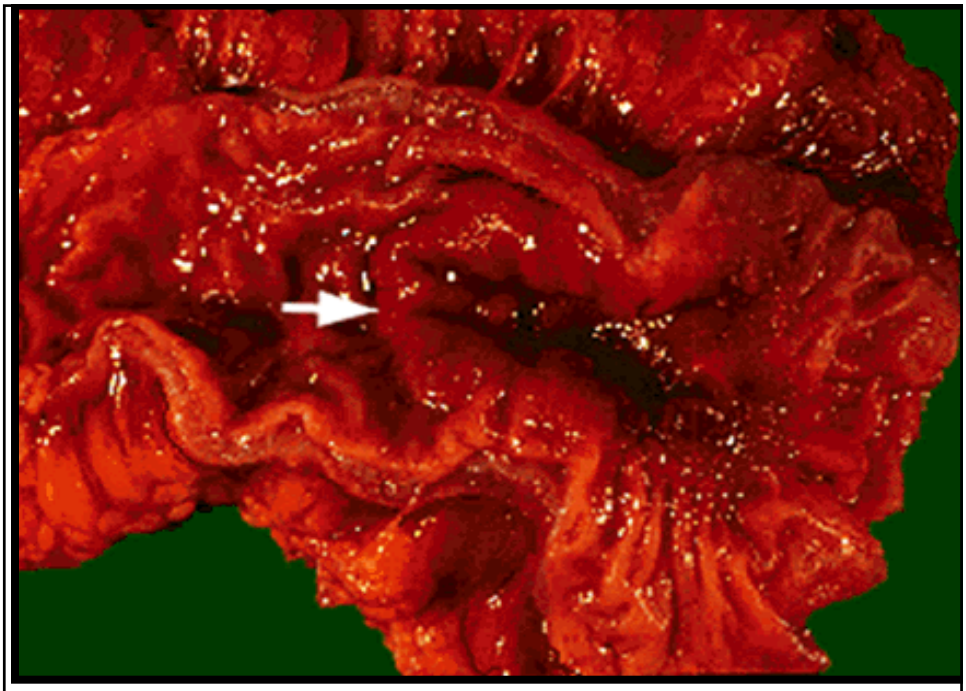
- •Approximately 5% of colorectal cancers demonstrate multiple lesions at diagnosis.
- •An adenomatous polyp is present elsewhere in the colon or rectum in 35% of patients diagnosed with a primary colorectal carcinoma.
- •Second tumors are more likely to be overlooked (“satisfaction of search error”).

Plain abdominal radiographs

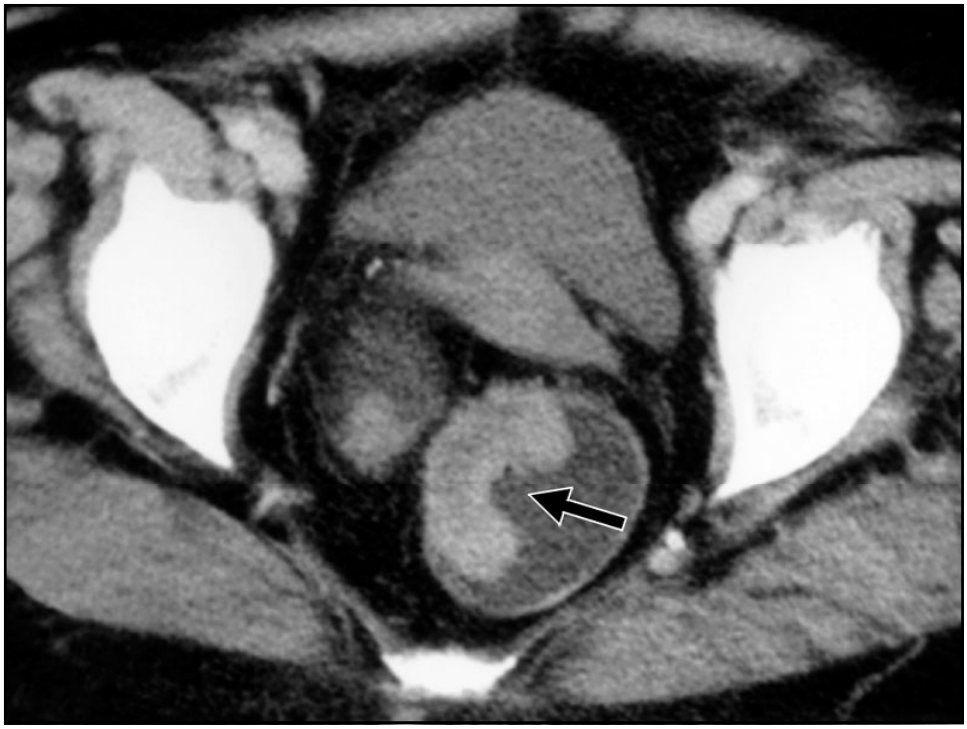
- •These are useful in patients presenting with large bowel obstruction or perforation.
- •Free gas under the diaphragm is detected best by a plain erect chest radiograph.
- •Rarely, mucin-producing colonic cancers demonstrate calcification in the primary tumor and in hepatic and peritoneal secondary deposits

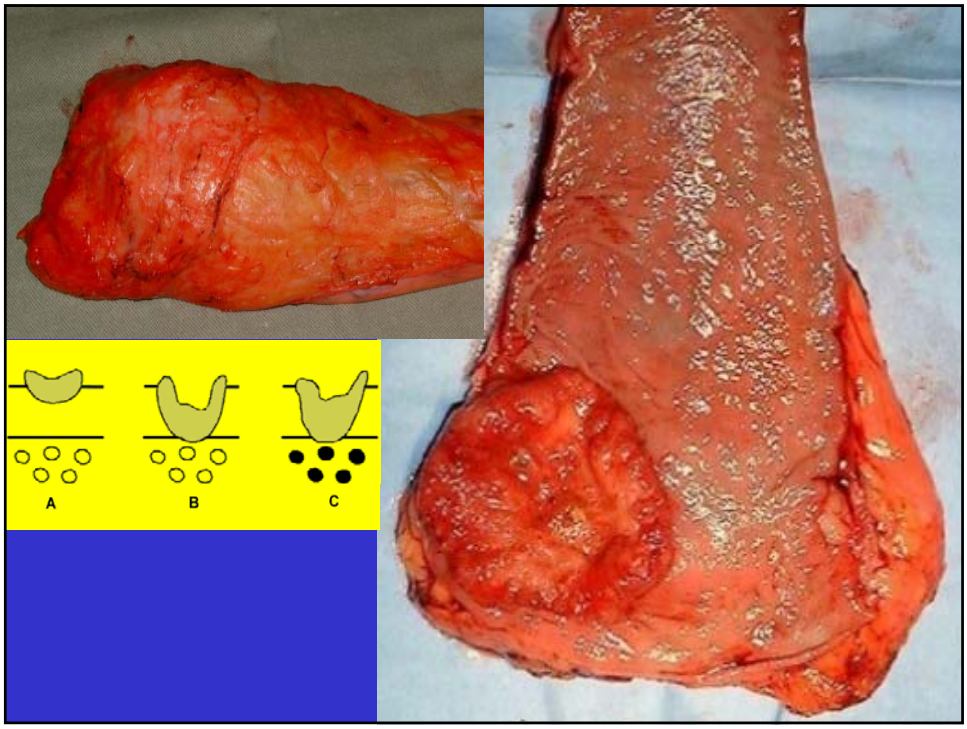
CT Staging System For Rectal Cancer*

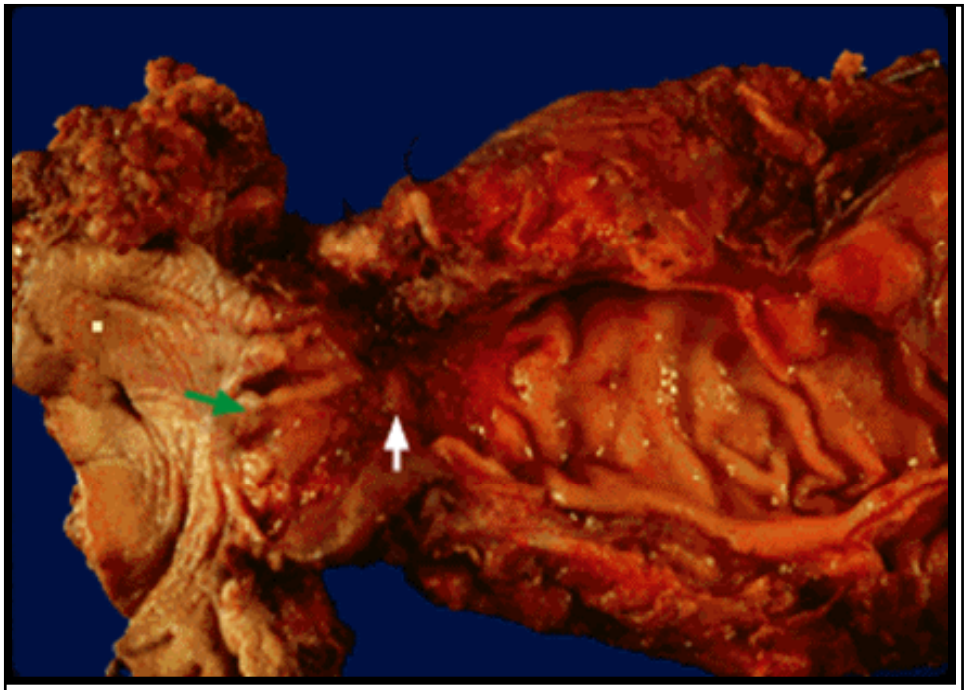
Stage	Description
T1	Intraluminal polypoid mass; no thickening of bowel wall
T2	Thickened rectal wall >6 mm; no perirectal extension
T3a	Thickened rectal wall plus invasion of adjacent muscle or organs
T3b	Thickened rectal wall plus invasion of pelvic side wall or abdominal wall
T4	Distant metastases, usually liver or adrenal



rectalcarcinoma







Rectal_adenocarcinoma

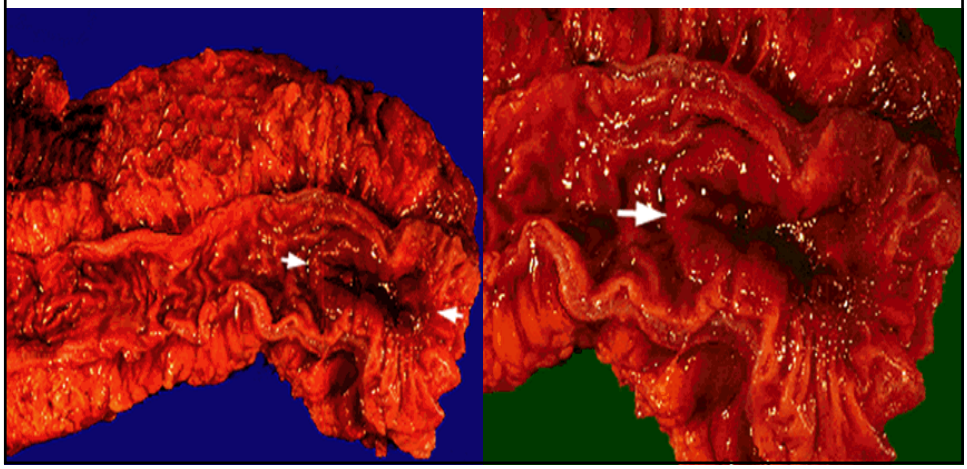
Adenocarcinoma, Colon

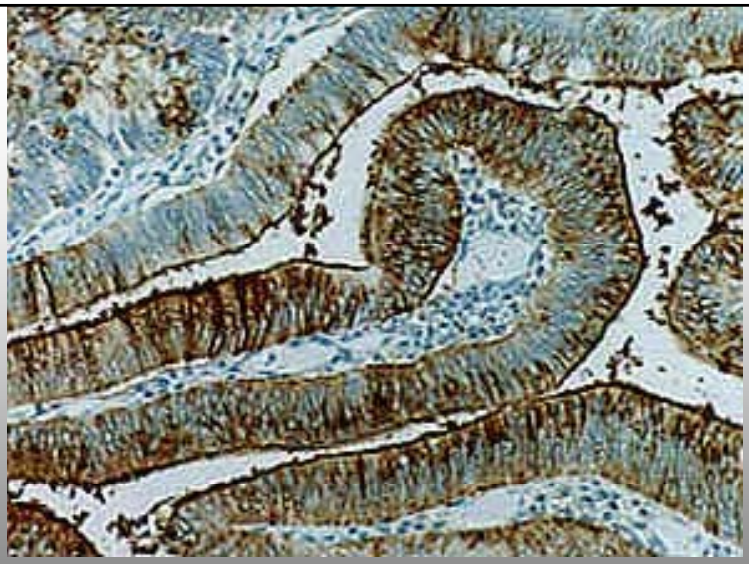
Adenocarcinoma of rectum

•This colon has been opened longitudinally.

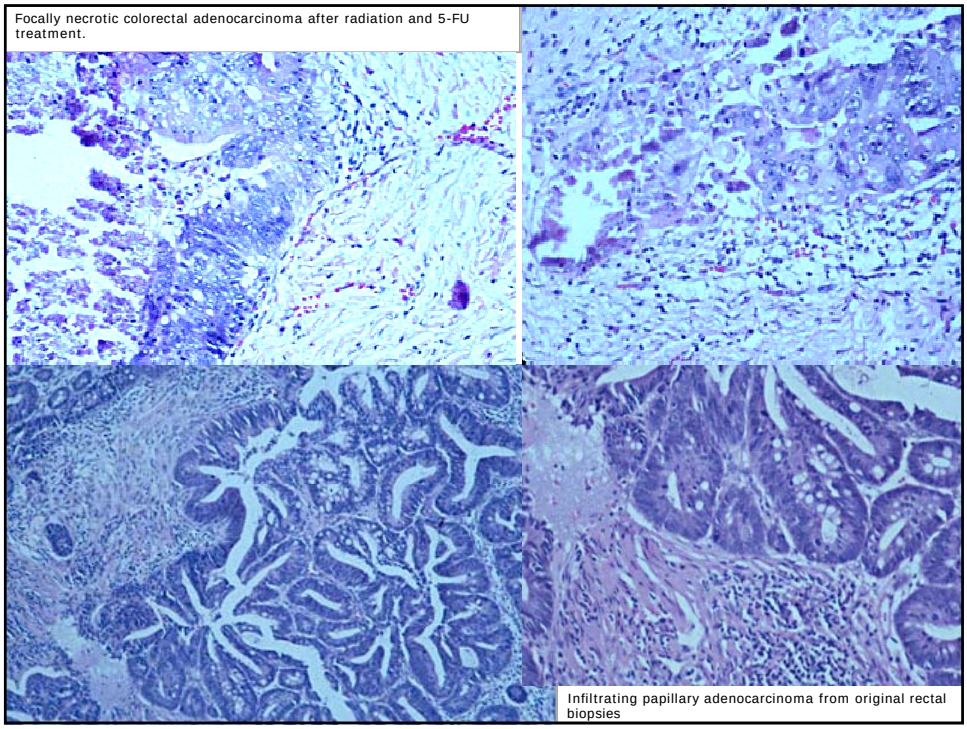
•Note ulcerated mass with raised rolled borders(arrows).

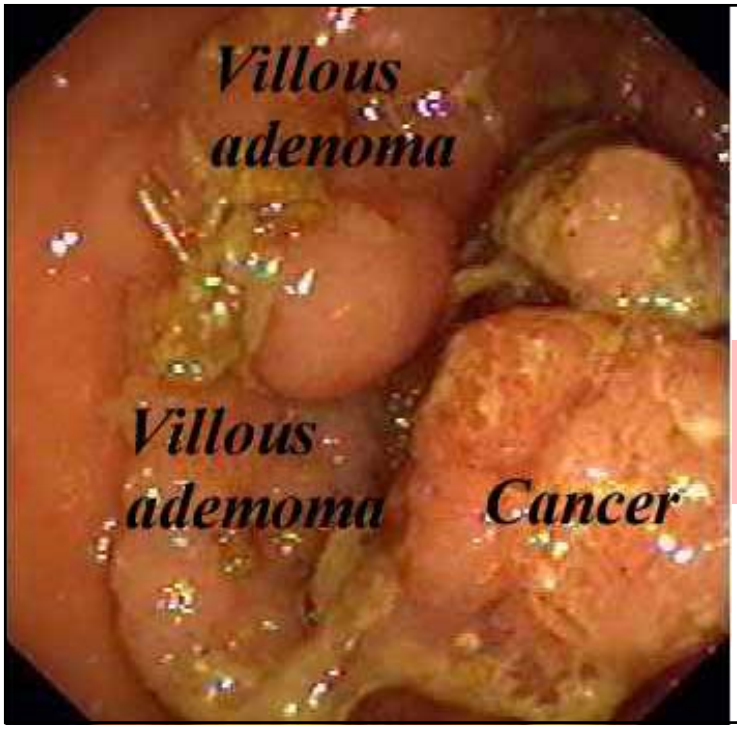
•The remaining mucosa is tan with normal folds.



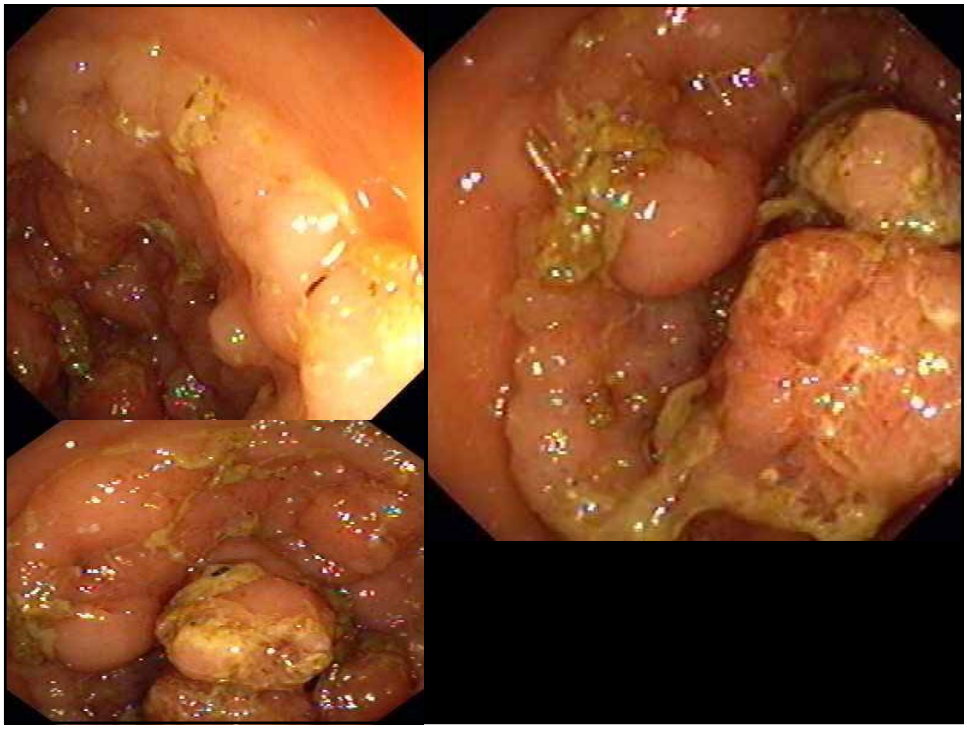


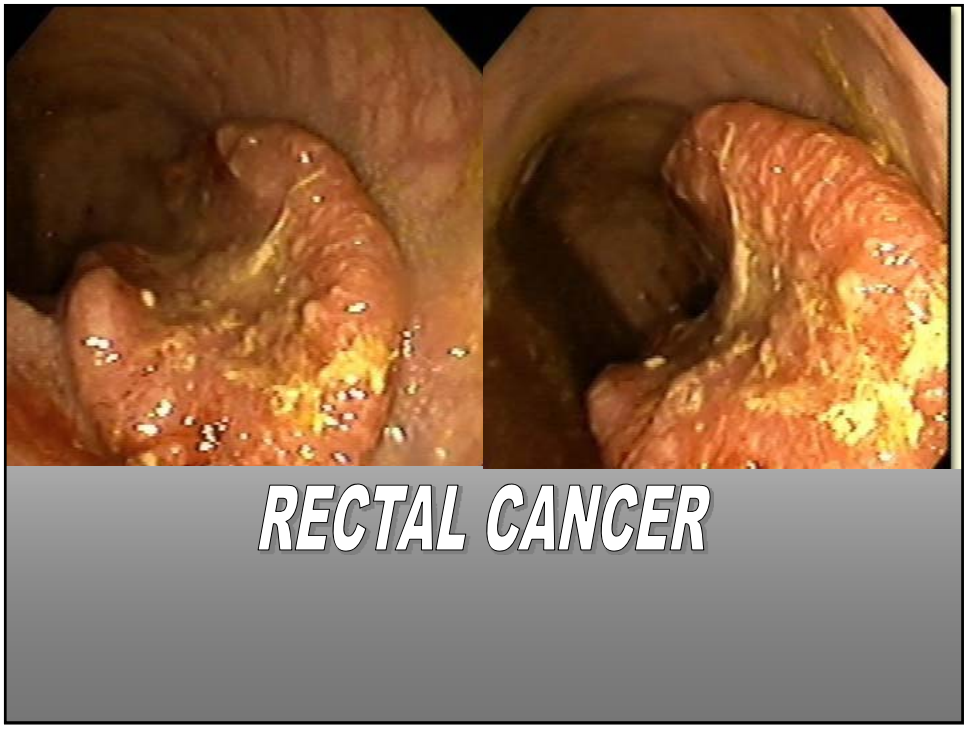
rectal adenocarcinoma: immunohistochemical staining for carcinoma associated mucin antigen using NCL-CA50.
Note cytoplasmic staining of tumour cells and extracellular mucins. Paraffin section.



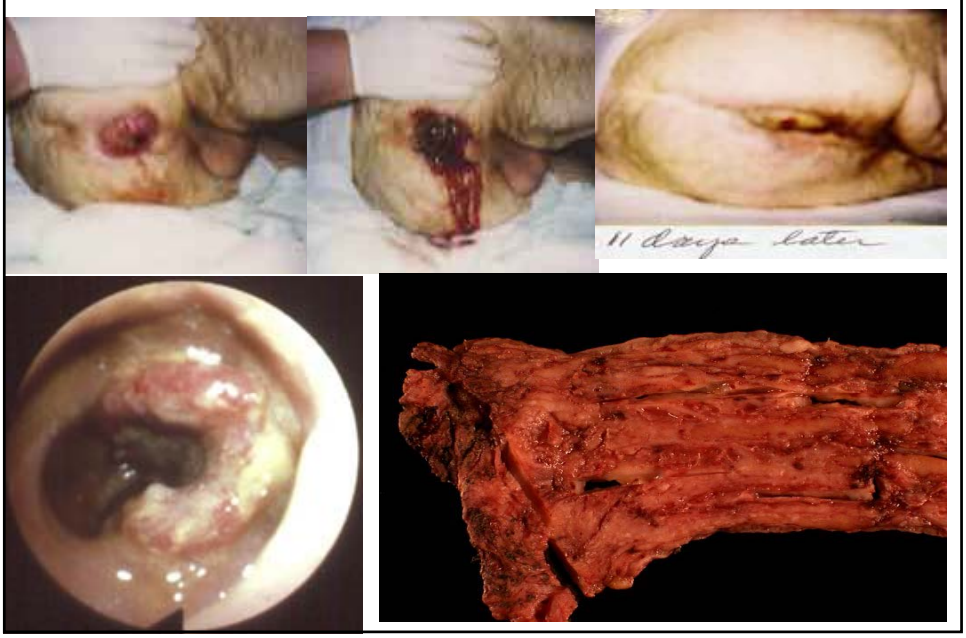


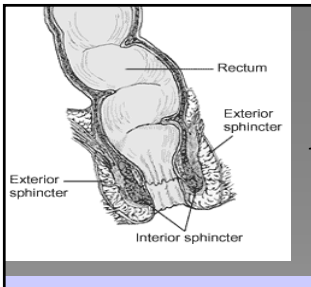
A rectal adenocarcinoma arising from a villous adenoma




RECTAL CANCER

Rectal Cancer[NOT VERY GOOD PICTURES BUT NOYICE THE HEMORRHAGE AND THE MASS]





ANAL TUMORS

Anal cancer, an uncommon cancer, is a disease in which cancer (malignant) cells are found in the anus.

The anus is the opening at the end of the rectum (the end part of the large intestine) through which body waste passes.

Cancer in the outer part of the anus is more likely to occur in men; cancer of the inner part of the rectum (anal canal) is more likely to occur in women.

If your anus is often red, swollen, and sore, you have a greater chance of getting anal cancer.

Tumors found in the area of skin with hair on it just outside the anus are skin tumors, not anal cancer.

Types
Anal cancers are skin cancers.

The majority of anal cancers are squamous cell carcinomas (in situ or epidermoid), which originate in the first layer of anal tissue and may spread to deeper layers.

This type is associated with HPV. About 15% of anal cancers originate in the glands near the anus; this is called adenocarcinoma, or Paget's disease.

The remaining anal cancers are basal cell carcinoma and malignant melanoma.

Melanoma in the anus is difficult to see and is often discovered at a late stage, after the cancer has spread through layers of tissue.

- very rare tumour — 300 new cases per year
- approximately 4% of large bowel tumours
- tumours of the anal canal more common in women
- tumours of the anal margin are more common in men
- high percentage of homosexuals have anal intra-epithelial neoplasia (AIN) – comparable to CIN
- slight female predominance overall
- most tumours occur between the ages of *55* and *65*

CAUSES

possible AIDs related malignancy

- homosexual activity - ano-receptive intercourse
- linked with human papilloma virus (HPV)
- previous history of sexually transmitted disease
- may be in association with carcinoma of the vulva or cervix in women

Histological Variants

in descending order of incidence

- mostly squamous cell carcinoma - approx. 90% (anal margin)
- adenocarcinomas - most of the remaining 10%

other histological types can occur

- basal cell carcinoma + mucoepidermoid
- melanoma
- cloacogenic carcinoma - lower metastatic potential than squamous carcinoma, is histological variant of anal margin tumours

Squamous cell carcinomas Most anal canal malignancies are squamous cell carcinomas. These have been classified as cloacogenic carcinomas, basaloid carcinomas, transitional cell carcinomas, or mucoepidermoid carcinomas. However, there is little difference in the natural history of these various types.
About 75% of carcinomas of the anal canal
•large-cell, keratinising subtype
•large-cell, non-keratinising subtype
•basaloid subtype

Unusual tumors arising in the anal canal include small-cell carcinomas, anal melanomas, and lymphomas.

Small-cell carcinomas of the anal canal are aggressive neoplasms similar in natural history to bronchogenic small-cell carcinomas. If such a histology is identified, the clinician should be alerted to the possibility of early distant metastases, and treatment should include chemotherapeutic regimens used in bronchogenic small-cell carcinomas.

Anal melanomas Although advanced anal melanomas generally are associated with a dismal survival, prognosis may be related to depth of penetration. Early anal melanomas < 2.0 mm in depth can be cured with wide excision. Abdominoperineal resection is indicated only rarely in the management of anal melanoma.

Adenocarcinomas are uncommon cancers associated with a poor prognosis. Treatment should be aggressive and based on a multimodality approach. The rarity of this tumor precludes the development of specific clinical trials.
About 25% of carcinomas of the anal canal
•of the rectal type
•of the anal glands
•in anorectal fistulas

According to the definition of the UICC, the anal margin extends from the dentate line up to 5 cm distal from it.

Generally spoken, carcinomas of the anal margin include those of the skin, e.g. basal cell carcinoma (basalioma) and squamous cell carcinoma.

The classification is detailed below. The frequency of carcinoma of the anal margin is 4 times higher in men than in women.

The basaloid subtype of squamous cell carcinoma of the anal canal should not be confused with the basal cell carcinoma (basalioma) of the skin of the anal margin.

This is important in terms of tumour biology and is therefore significant for therapy.

The basaloid type of squamous cell carcinoma of the anal canal is a metastatic tumour, whereas basal cell carcinomas (basaliomas) of the skin of the anal margin may demonstrate local infiltration but do not metastasise and can therefore be primarily treated with local surgical excision.

On the other hand, the basaloid subtype of squamous cell carcinoma of the anal canal is usually treated with radiotherapy or chemotherapy.

Presenting signs and symptoms

Anal discomfort
Bleeding
Tenesmus
Nodular or ulcerating lesions sometimes mistaken for haemarroids.
Enlarged inguinal nodes

Spread

Direct – rectum, perineum, peri-anal structures.
Lymph – inguinal nodes + femoral → external iliac
Blood – usually late

Staging

T1	tumour 2cm or less
T2	tumour less than 5cm
T3	tumour greater than 5cm
T4	extension beyond anus to involve adjacent structures (bladder, vagina)
N1	involvement of nodes (usually +ve on presentation)

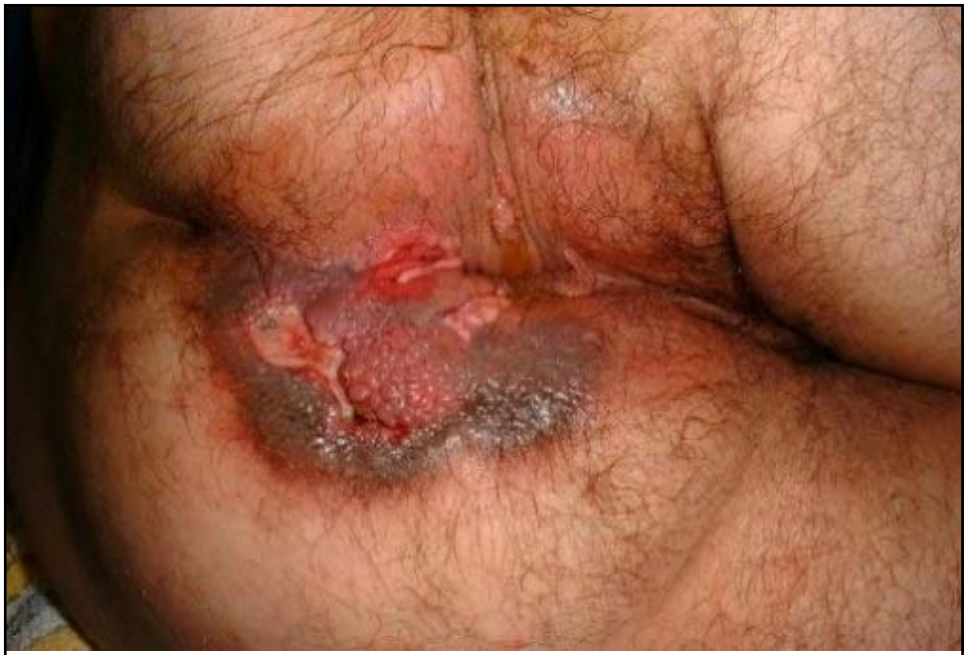
Squamous Carcinoma of Anus (Colon)	
Etiology	•Squamous carcinoma of the anus has been associated with a variety of sexually transmitted diseases including: HPV, Lymphogranuloma venereum, Herpes simplex, Chlamydia and Gonorrhea. •Many cases do not have evidence of a sexually transmitted disease and the etiology in these cases is unknown.
Pathogenesis	•Development of carcinoma in HPV+ patients has been linked to the development of anal condyloma and intraepithelial dysplasia associated with HPV 16 and 18, similar to the develoment of cervical neoplasia in HPV+ patients. •The mode of pathogenesis in HPV- patients is unknown.,
Epidemiology	~~•Squamous carcinoma of the anus is most common during the 5th and 6th decades.~~ •Females are affected 2-3X more than males and whites more than blacks. •Single males have a six-fold higher incidence than married males, and this appears due to the high prevalence of HPV infection associated with anal intercourse. •There is a recent significant rise in the incidence of anal squamous carcinoma associated with the rise in HPV infection. •An increased incidence has also been noted in renal transplant patients with immunosuppression.
General Gross Description	•The tumors present as mass lesions which are exophytic with a smooth surface which may be ulcerated in larger lesions. •They are present above and below the Dentate Line. •Cut surface is firm and white. •Some tumors may show evidence of contiguous condyloma in the HPV+ patients.
General Microscopic Description	•The microscopic appearance is variable as several variants exist. •Most commonly their appearance is similar to non-keratinizing cervical squamous carcinoma. •The most commonly seen variants demonstrate basaloid, adenoid cystic or transitional (cloacogenic) patterns. •The two most common symptoms are anal bleeding (50%) and pain (30%).
Clinical Correlation	•Regardless of histologic variant, squamous carcinomas of the anus show similar behavior with direct extension into the sphincter, vagina, perianal tissue and prostate. •Lymphangitic spread through the perirectal and groin nodes are both seen. •Primary treatment is surgical with local resection limited to Stage 1 and small Stage 2 lesions. Abdominal-perineal resection is no longer in favor. •More advanced lesions or larger lesions are treated with radiation therapy often combined with chemotherapy. •Prognosis is determined by site (above or below the dentate line), Size (Tumors <2cm are more favorable), and degree of cellular differentiation. •Overall survival at 5yrs is greater than 70%

Abdominoperineal resection (extensive surgical resection from anus extending upward)

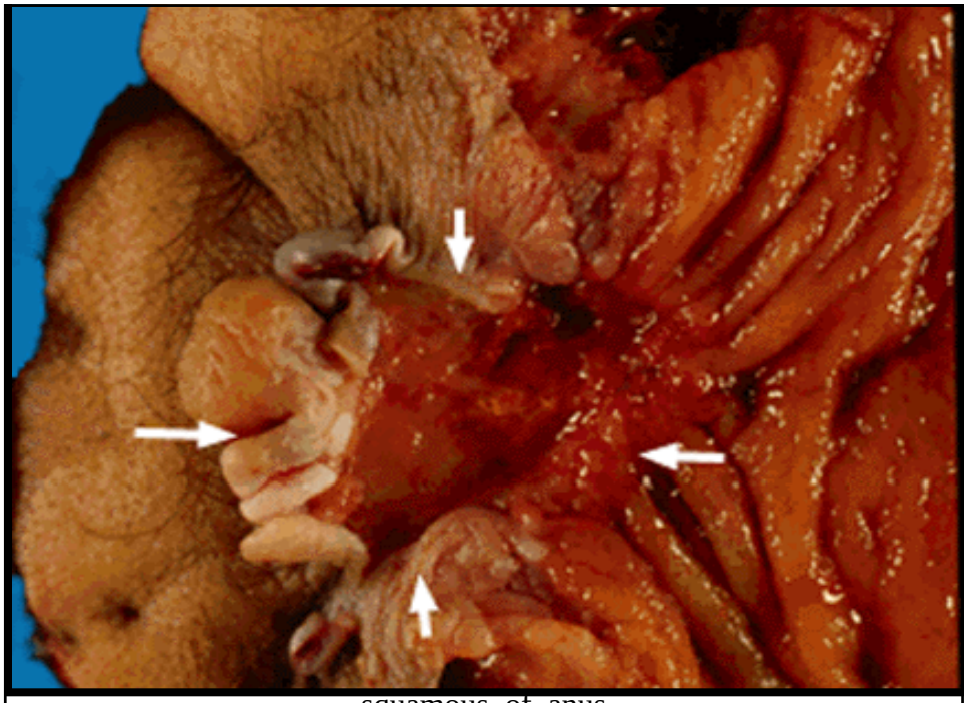
Very small tumors may only need surgical removal.

Radiation treatment for inoperable disease (disease has spread too much)

If metastatic, spreads to lymph nodes (glands) in the groin and these glands may be resected.



ULCERATIVE ANAL SQUAMOUS CELL CARCINOMA



squamous_of_anus