

Tumors of small & large intestine

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Peutz-Jeghers syndrome

This is characterized by;

- ∞ **Familial intestinal hamartomatous polyposis** in jejunum,
 - Which may results in hemorrhage, &
 - often intussusception.
- ∞ **Melanosis** of the oral mucosa & lips.



Treatment

- ∞ **Resection** in cases of serious bleeding or intussusception.
- ∞ Large single polyps can be removed by enterotomy or short lengths of heavily involved intestine can be resected.

Carcinoid tumor

Pathology

- ∞ These tumors occur thru-out GIT, mostly in the **appendix, ileum & rectum** in decreasing order of frequency (AIR).
- ∞ Arise from **neuroendocrine cells** at the base of intestinal crypts.
- ∞ Primary is usually small but when they **metastasize**, the liver is usually involved with numerous secondaries, which are larger & more yellow than the primary.
- ∞ Produce vasoactive peptides, esp. 5-hydroxytryptamine (**serotonin**);
 - appear as 5-hydroxyindoleacetic acid in the urine during attacks.

Clinical features

- ☞ Reddish-blue **cyanosis**.
- ☞ **Flushing** attacks, classically induced by alcohol.
- ☞ **Diarrhea** & borborygmi.
- ☞ **Asthmatic** attacks.
- ☞ Sometimes pulmonary & tricuspid stenosis.

Treatment

∞ Medical treatment

- **Octreotide** (a somatostatin analogue) reduces both flushing & diarrhea.

∞ Surgical treatment

- **Resection** is usually sufficient.
- If found incidentally at appendicectomy nothing further is required.
- In patients with metastatic disease, multiple enucleations of hepatic metastases or even partial hepatectomy can be carried out.

Familial adenomatous polyposis

- ∞ FAP mainly affect **large bowel**, but polyps can occur in the stomach, duodenum & small intestine.
- ∞ Main risk is **large bowel cancer**, but duodenal & ampullary tumors have been reported.
 - Ca colon occurs 10-20 years after the onset of polyposis.
- ∞ It is inherited as a Mendelian dominant, & the responsible **APC gene** is identified on the short arm of chromosome 5.
- ∞ It can be associated with benign **mesodermal tumors** eg desmoid tumors & osteomas;
 - epidermoid cysts can also occur (Gardner' s syndrome).



Clinical Features

☞ Symptomatic cases

These are either new cases or those from an affected family who have not been screened;

- Loose stools, with passage of blood & mucus.
- Lower abdominal pain.
- Weight loss.

☞ Asymptomatic cases

- These are usually members of affected families, attending for screening.
- If there are no adenomas by the age of 30, FAP is unlikely.

Diagnostic Investigations

∞ Sigmoidoscopy

- Polyps are usually visible by the age of **15** years, & will almost always be visible by the age of 30.
- If in doubt colonoscopy is performed with biopsies.
- If over **100** adenomas are present the diagnosis is confirmed.

∞ Double-contrast barium enema

- Number & distribution of polyps can be shown.

Screening policy

- ☞ All members of the family should have sigmoidoscopy at the age of **10-12** years, & repeated every 1-2 years.
 - Most of those who are going to get polyps will have them at 20, & these require operation.
 - If there are no polyps at 20, continue with 5-yearly examination until age 50; if there are still no polyps there is probably no inherited gene.
- ☞ **Pigmented spots in the retina (CHIRPES) & DNA tests for the FAP gene can make screening more reliable.**

Treatment

- ∞ If the diagnosis is made during adolescence, operation is deferred to the age of 17 or 18.
- ∞ **Colectomy** with ileorectal anastomosis;
 - rectum is subsequently cleared of polyps by snaring or fulguration.
 - Followed by flexible sigmoidoscopy at 6-monthly intervals thereafter.
- ∞ **Restorative proctocolectomy** with an ileoanal anastomosis.
 - indicated in cases of serious rectal involvement, those who can't attend for follow-up & those with established cancer of the rectum or sigmoid.

Carcinoma of colon

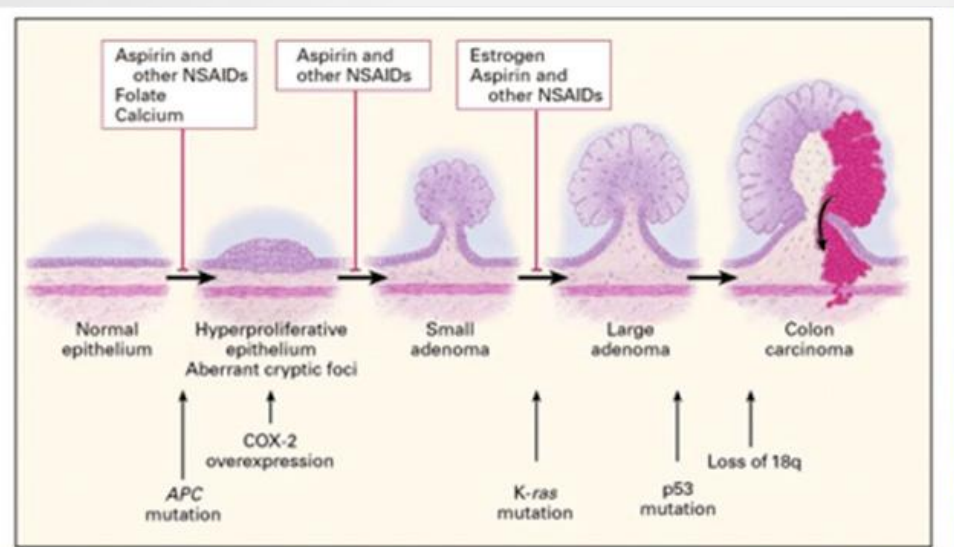


Etiology

∞ Exact cause is unknown.

∞ Risk factors

- Genetic → Family cancer syndrome
- Low-fibre, high-fat diet
- Smoking & alcohol
- Inflammatory bowel disease
- Polyposis syndromes (FAP, HNPCC)
- Cholecystectomy (with increase bile acid secretion)

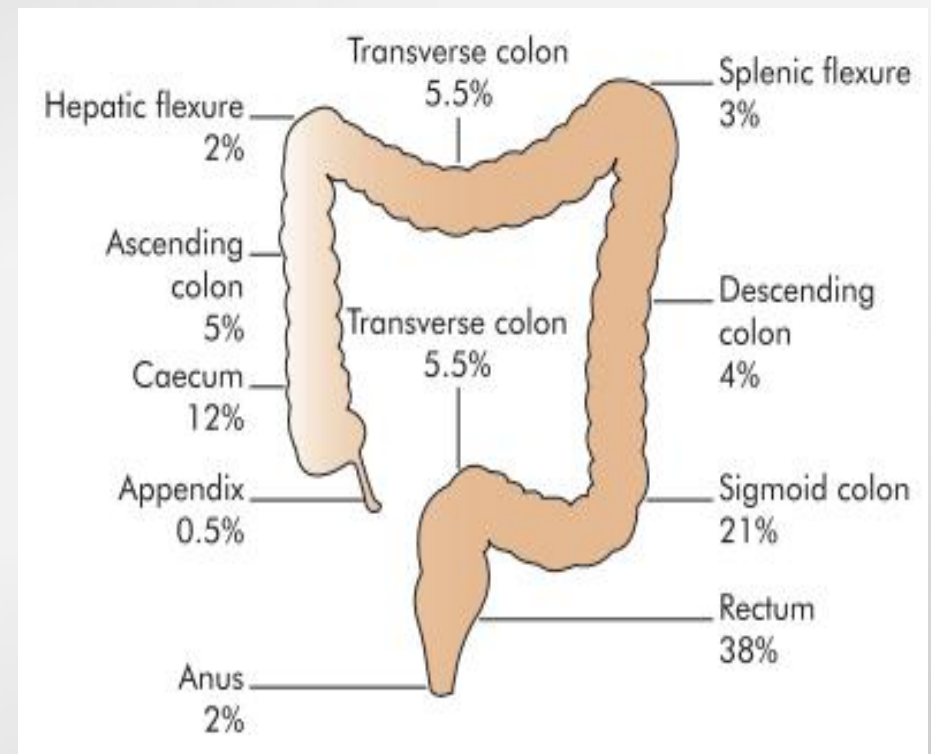


Colon cancers result from a series of pathologic changes that transform normal epithelium into invasive carcinoma. Specific genetic events, shown by vertical arrows, accompany this multistep process.

Pathology

Site

1. Anus (2%)
2. Rectum (38%)
3. Rectosigmoid junction (7%)
4. Sigmoid colon (21%)
5. Descending colon (4%)
6. Splenic flexure (3%)
7. Transverse colon (5.5%)
8. Hepatic flexure (2%)
9. Ascending colon (5%)
10. Cecum (12%)
11. Appendix (0.5%)



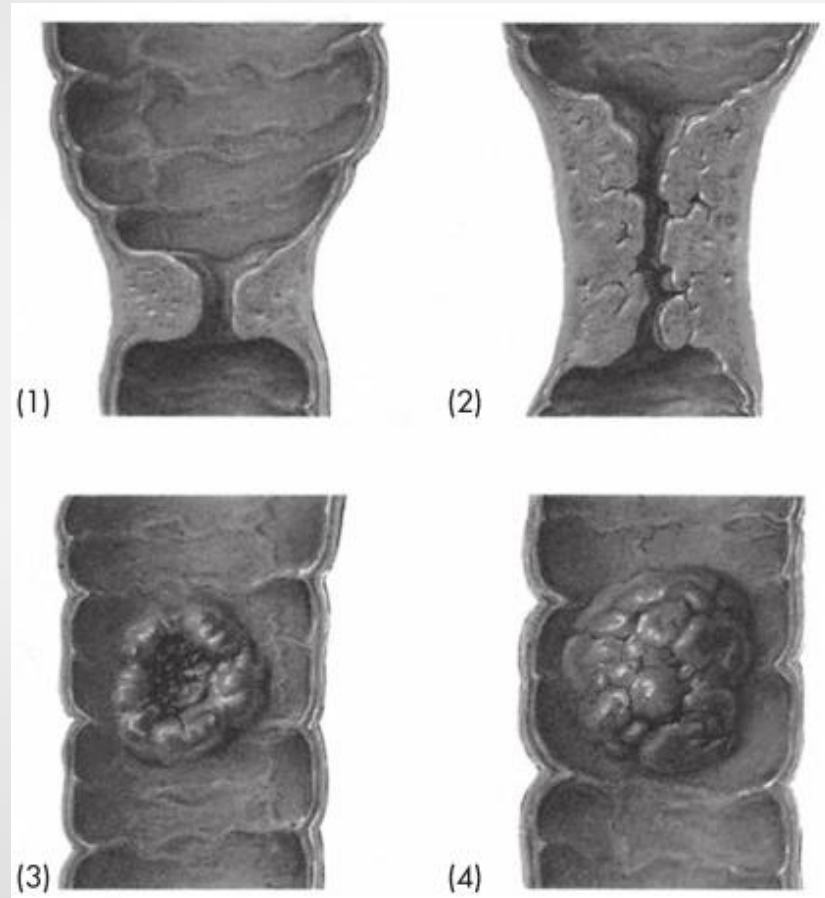
Pathology

Macroscopic Features

1. Annular
2. Tubular
3. Ulcerative
4. Cauliflower

Microscopic Features

- ∞ Columnar – cell carcinoma originating in epithelial cells that line the colon, or in crypts of Lieberkuhn.



Pathology

Spread

∞ *Local spread*

- Growth is limited to bowel for a considerable time.
- It spreads round the intestinal wall, & to a certain extent longitudinally.
- Penetration of serous coat occur esp. in ulcerative variety, & according to the segment involved, adjacent structure then become invaded by growth.

∞ *Lymphatic spread*

- Epicolic lymph nodes (N1) → Paracolic lymph nodes (N2) →
- Superior & inferior mesenteric lymph nodes (N3).

∞ *Bloodstream spread*

- Liver.

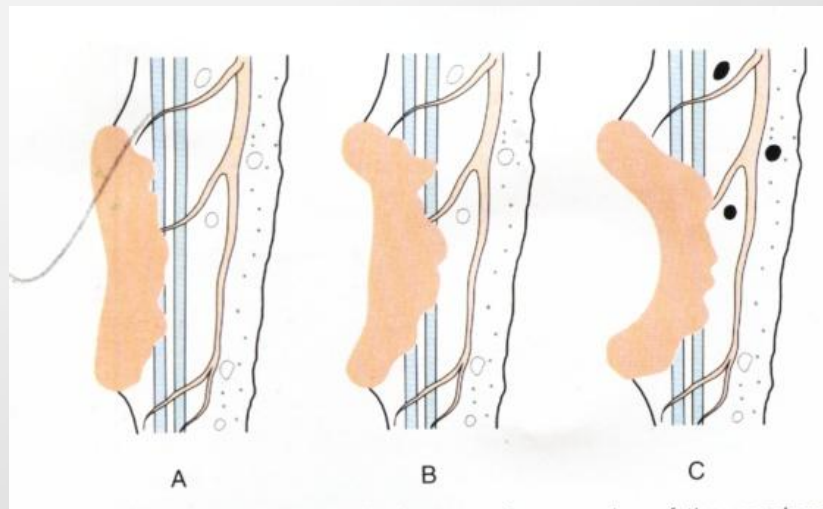
∞ *Transcelomic spread*

- to other structures within the peritoneal cavity.

Staging

Dukes' classification

- A. Stage A → Confined to bowel wall.**
- B. Stage B → Thru the bowel wall but not involving the free peritoneal serosal surface.**
- C. Stage C → Lymph nodes involved.**
- D. Stage D → Advanced local disease or metastases to the liver.**



Staging

TNM classification

∞ Tumor stage (T)

- Into submucosa (T1) → Into muscularis propria (T2) → Into pericolic fat but-not breaching serosa (T3) → Breaches serosa or directly involving another organ (T4).

∞ Nodal stage (N)

- No nodes involved (N0) → 1-2 nodes involved (N1) → 3 or more nodes (N2).

∞ Metastases (M)

- No metastases (M0) → Metastases (M1).

∞ Venous invasion (V)

- No vessel invasion (V0) → Vessels invaded (V1).

∞ Residual tumor (R)

- No residual tumor (R0) → Margins involved (R1).

Clinical features

- ∞ **Age** → Usually, over 50 years.
- ∞ **Sex** → Men to women ratio is 3 : 2.

Carcinoma Of Left Side Of Colon

Neoplasms are usually of tubular or annular variety.

Symptoms

∞ Abdominal pain

- Usually mild lower abdominal **colic** which, becomes persistent in left lower abdomen, with severe colicky exacerbations.

∞ Alteration of bowel habit

- Initially **constipation**, ie infrequent passage of hard feces. →
- Suddenly, often following an episode of colic, patient passes a number of loose stools ie **diarrhea**. →
- **Constipation** then returns.

∞ Abdominal distension

- Relieved by passing flatus or feces.

∞ Feeling of **lump** in abdomen

∞ **Loss of weight**

∞ **Vomiting**

Signs

1. **Weight loss.**
2. **Pallor (if chronic blood loss occur).**
3. **Left supraclavicular glands** may be enlarged.
4. **A palpable mass on left side**, usually in left iliac fossa (due to the presence of hard feces above tumor).
5. **Liver** may be palpable.
6. **Hyperactive bowel sounds** in cases of chronic obstruction.

Carcinoma Of Sigmoid Colon

- ∞ Symptoms & signs are similar to that mentioned above, with following differences:
- **Pain** → Usually colicky.
 - **Tenesmus** → Feeling of the need for evacuation, esp. in early morning.
 - **Bleeding per rectum**
 - **Bladder symptoms** → Herald colovesical fistula.

Carcinoma Of Transverse Colon

- ⌘ **Mimics carcinoma of stomach, because of its position, & anemia & lassitude that it cause.**

Carcinoma Of Cecum & Ascending Colon

Symptoms

1. Abdominal **pain** → A dull ache in right iliac fossa.
2. **Alteration of bowel habit** → Not as prominent as in left-sided growth.
3. Anorexia, & loss of weight.
4. Vomiting.
5. Lump in right iliac fossa.

Signs

1. Weight loss.
2. Pallor.
3. Supraclavicular lymph nodes may be palpable.
4. A firm, irregular **mass** may be palpable in right iliac fossa, which may be fixed or freely mobile.
5. Right iliac fossa is often tender, with some overlying guarding.
6. Liver may be palpable.
7. Bowel sounds may be hyperactive.

Diagnostic investigations

∞ *Sigmoido - & colonoscopy*

- Demonstrates the macroscopic variety of neoplasm, & biopsy can be taken to confirm histologic diagnosis.

∞ *X-ray with barium enema*

- Shows a constant, irregular filling defect.

∞ *Exfoliative cytology*

- Valuable, if endoscopy is not available.

∞ *Ultrasonography*

- Shows liver metastases.

∞ *CT scanning*

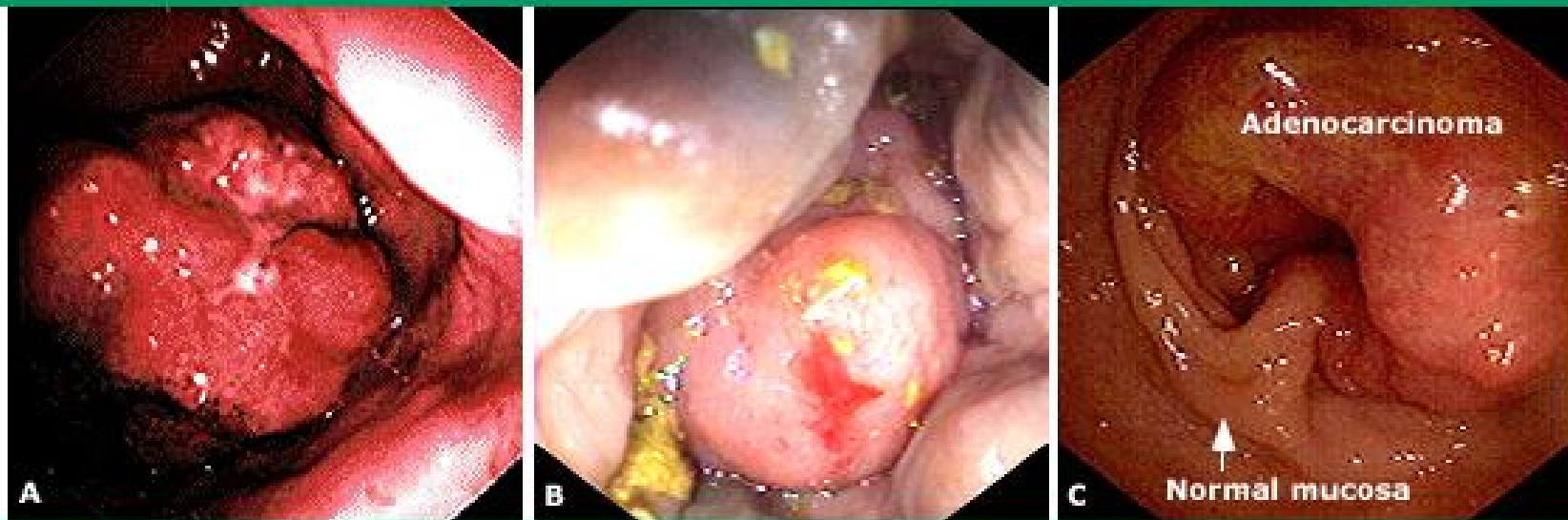
- Used in patients with large palpable abdominal masses to determine local invasion.

Normal sigmoid colon

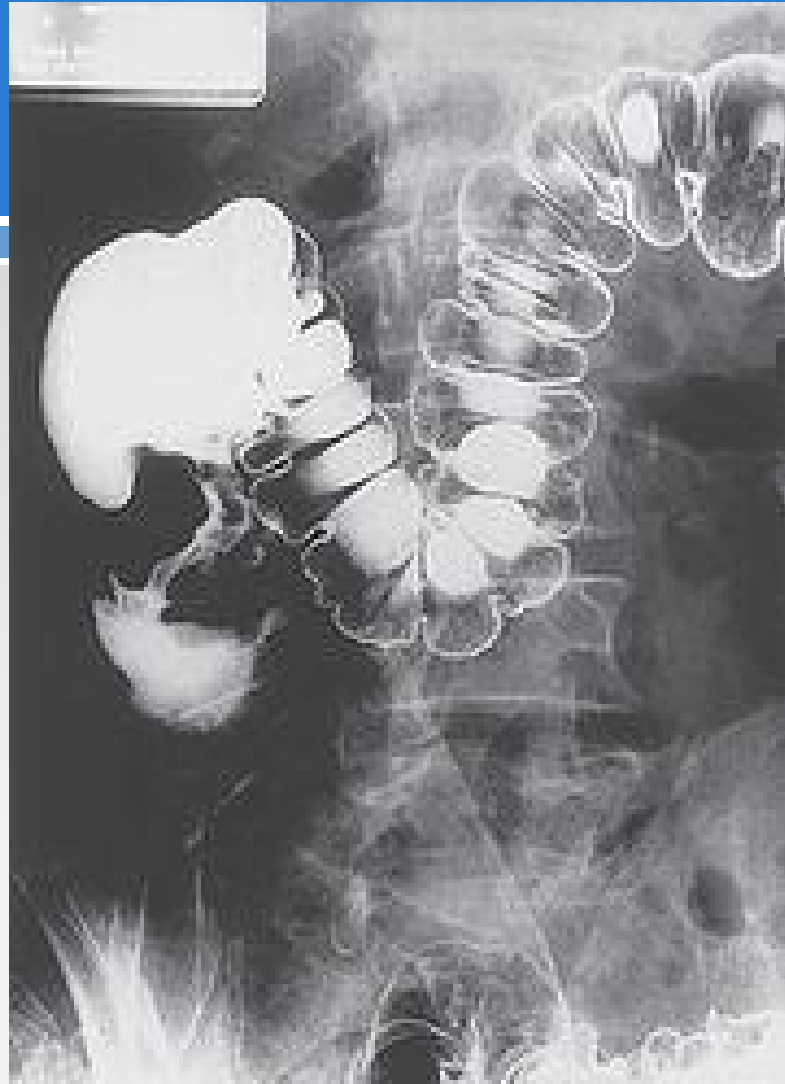


Endoscopic appearance of the normal sigmoid colonic mucosa. The fine vasculature is easily visible, and the surface is shiny and smooth. The folds are of normal thickness.

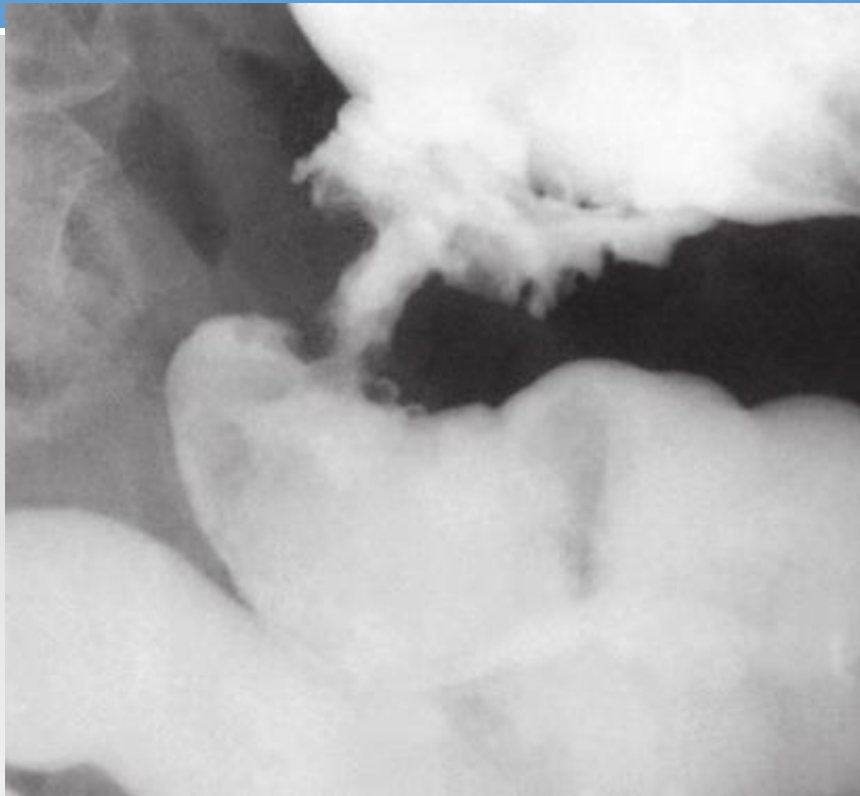
Adenocarcinoma of the colon



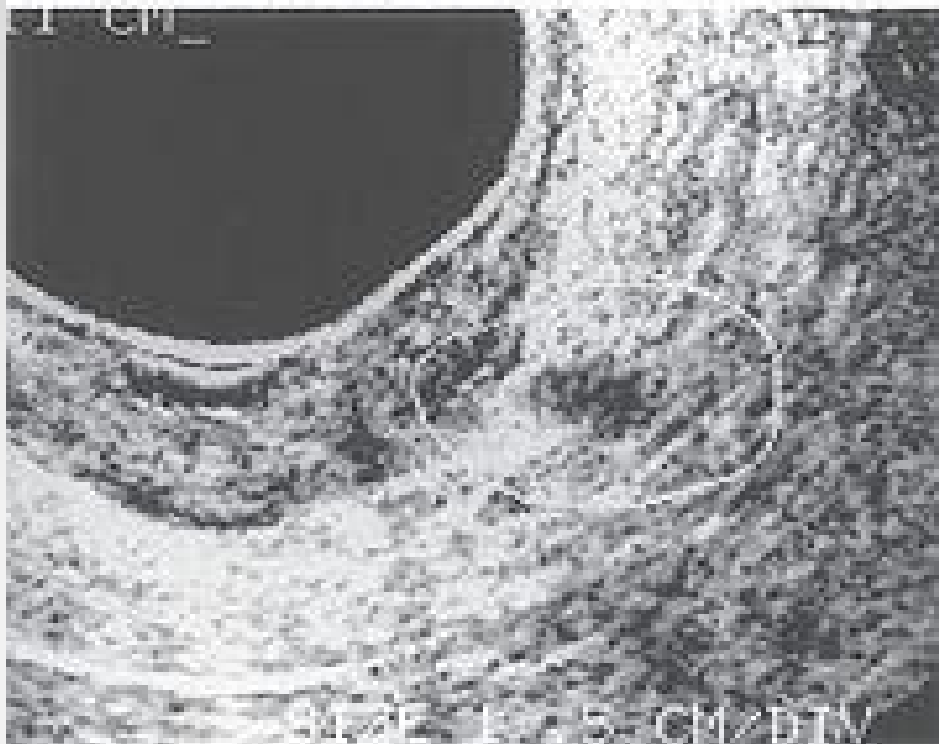
Adenocarcinoma of the colon may have a variety of appearances on endoscopy. Panel A: a typical exophytic mass; Panel B: a friable polypoid mass; Panel C: circumferential adenocarcinoma.



Double contrast barium enema showing a cancer in the ascending colon.

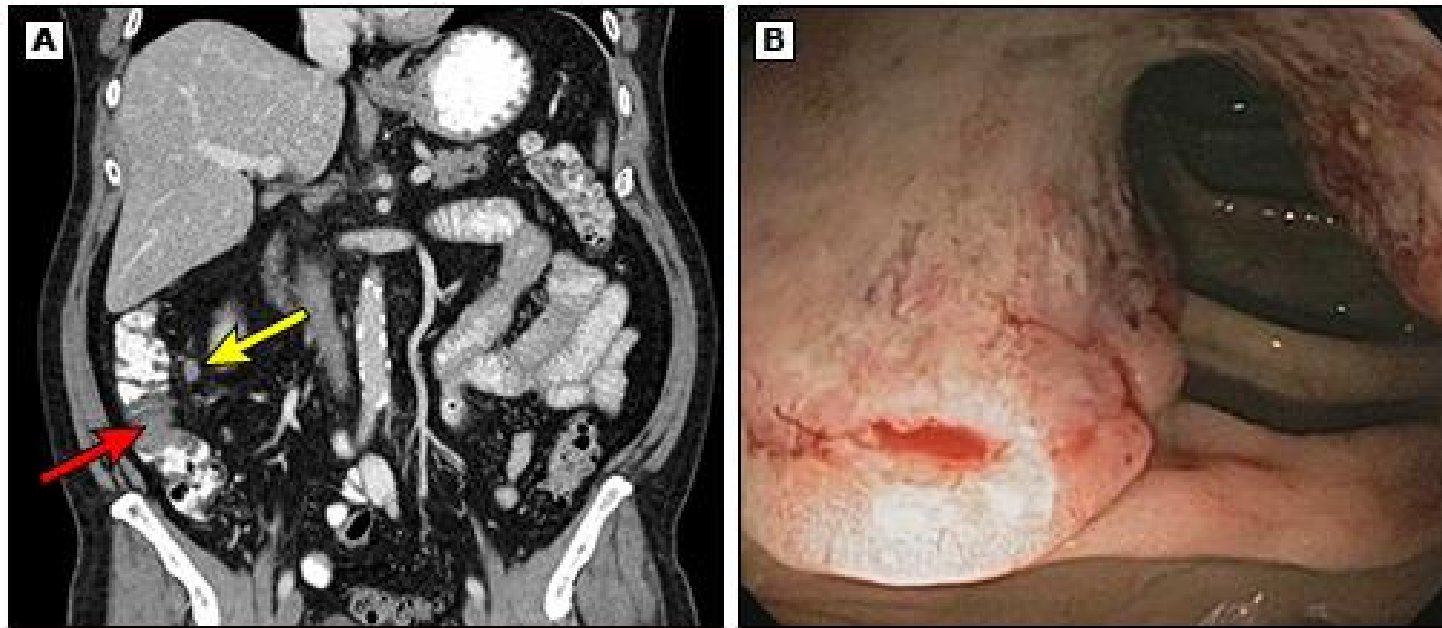


Barium enema showing a carcinoma of the sigmoid colon. It may have an 'apple core' appearance, i.e. a short, irregular stenosis with sharp shoulders at each end.

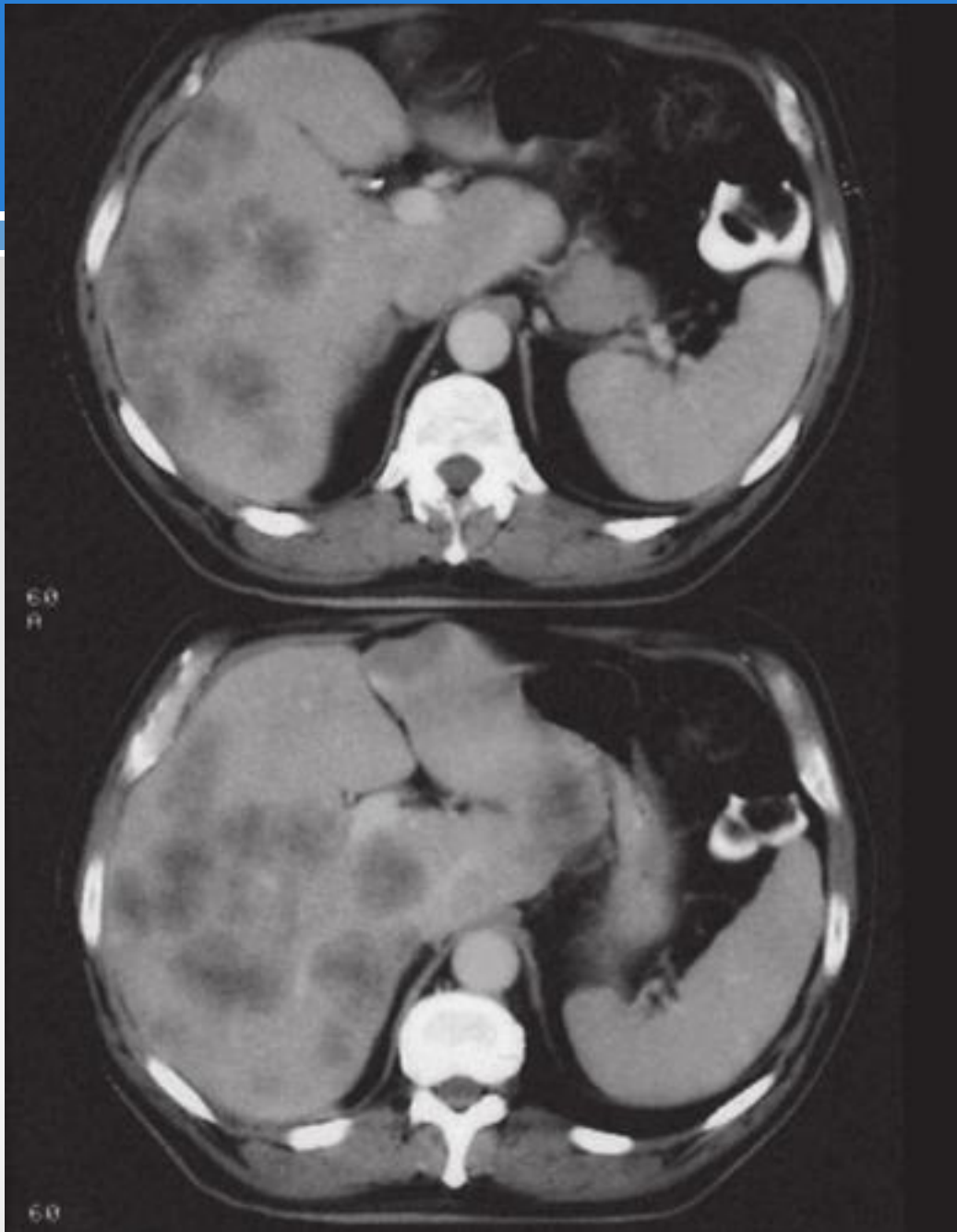


- Endorectal ultrasound showing (A) tumour invasion through the muscular wall to the perirectal fat, T3 and (B) an enlarged lymph node in the mesorectum.

Colon cancer seen on CT scan and colonoscopy



- (A) Computed tomographic (CT) scan showing a filling defect in the ascending colon (red arrow) along with an involved lymph node (yellow arrow).
- (B) Colon cancer identified in the ascending colon on subsequent colonoscopy.



Treatment

∞ Patient preparation

- Bowel preparation
- Blood transfusion, to correct anemia.
- **Antibiotics** → Metronidazole with gentamicin, or cephalosporins.

∞ Operation

Bowel preparation

When there is no intestinal obstruction

- ∞ Dietary restriction to fluids only for 48 hours before surgery.
- ∞ On the day before operation two sachets of Picolax (sodium picosulphate) are given to purge the colon.
- ∞ Rectal washout may be needed.
- ∞ Alternatives include prograde lavage via a nasogastric tube using water or balanced electrolyte solutions.

When intestinal obstruction is present

- ∞ Above mentioned preparation may precipitate abdominal pain & it should be left to the time of the operation using an on table lavage technique.

Operation

∞ **Incision**

- Abdomen is opened thru a short paramedian or midline incision, which is extended if growth is removable.

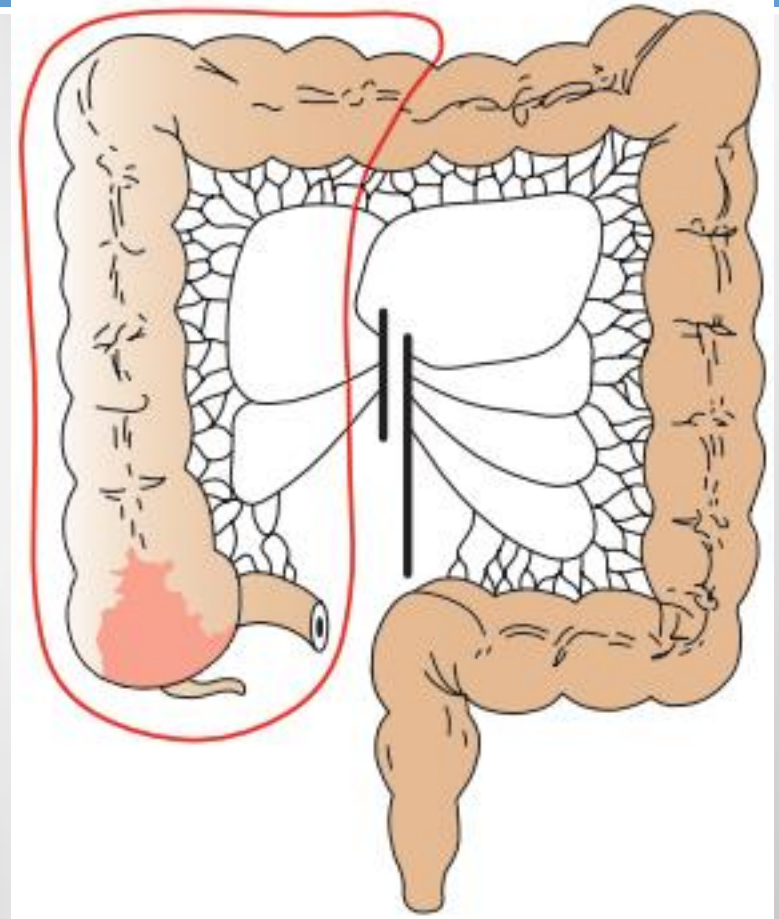
∞ **Look for signs of operability**

1. **Growth** → Whether, it is fixed or free.
2. **Lymph nodes** → For metastases.
3. **Peritoneum** esp. pelvic → For neoplastic implantations.
4. **Liver** → For secondary deposits.

In carcinoma of cecum or ascending colon

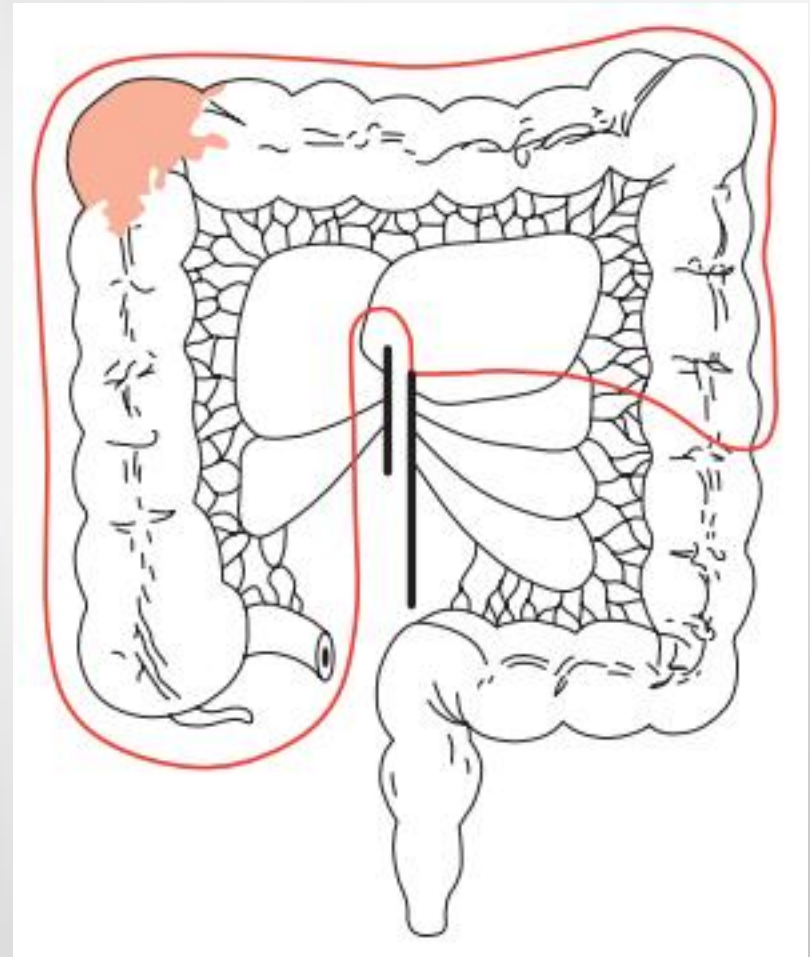
∞ Right hemicolectomy

- Note: A curable resection is one that involves the regional lymph nodes, & hepatic metastases, if present.



In carcinoma of hepatic flexure

- ∞ **Right hemicolectomy, extended upto the proximal half of descending colon.**



In carcinoma of transverse colon

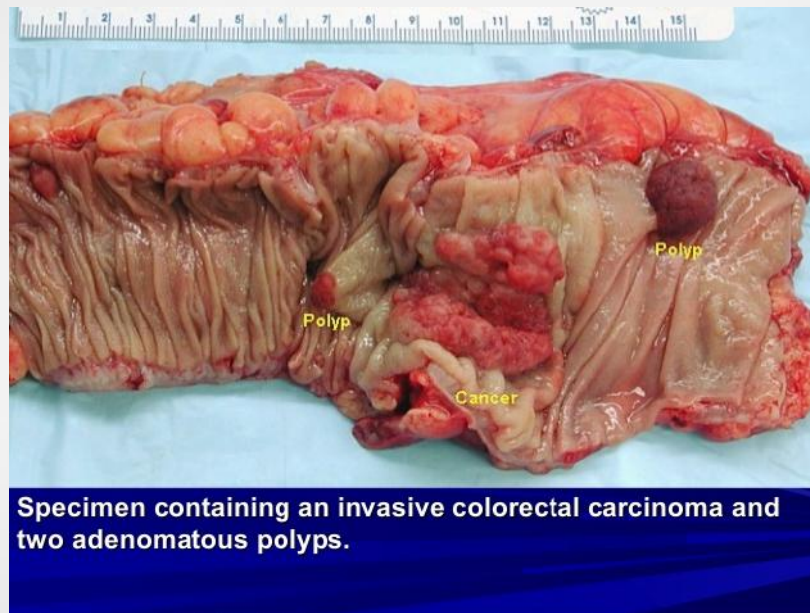
∞ Area of resection include

- upper 2/3 of ascending colon,
- transverse colon, &
- upper 1/3 of descending colon,
- together with the transverse mesocolon & the greater omentum,

∞ followed by end-to-end anastomosis.

In carcinoma of splenic flexure or descending colon

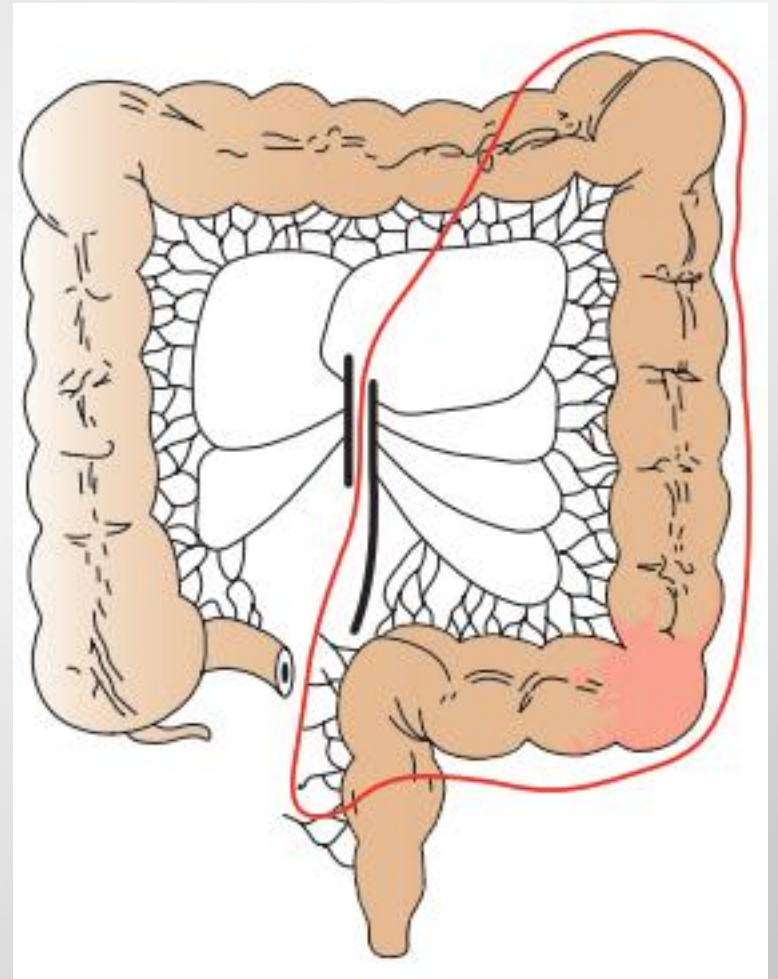
- ∞ Area of resection includes upper 2/3 of ascending colon, transverse colon, & upper 3/4 of descending colon.



Specimen containing an invasive colorectal carcinoma and two adenomatous polyps.

In carcinoma of sigmoid colon

- ∞ **Area of resection includes distal 1/3 of transverse colon, splenic flexure, descending colon, sigmoid colon, rectosigmoid junction, & upper 1/3 of rectum.**



When the growth is inoperable

- ∞ **Growth in left colon → Colostomy.**
- ∞ **Growth in ascending colon → Bypass using an ileocolic anastomosis.**

For hepatic metastasis

- ⌘ **Hepatic resection after recovery from colonic resection.**
- ⌘ **Irresectable hepatic lesions require cytotoxic drugs or ablative treatment.**

Chemotherapy

- ∞ In colonic cancer, there is no role for preoperative chemotherapy.
- ∞ There is evidence of improved outcome after surgery with **5-fluorouracil** (5-FU)-based chemotherapy in node-positive disease (Dukes' C/N1,2).
 - Systemic 5-FU alone or in combination with **oxaliplatin** can improve survival by 10–15% in node-positive disease.
- ∞ Similarly, 5-FU infused into the portal vein during and immediately after the primary operation have shown a small benefit.
- ∞ Recently, new drugs (**irinotecan and cetuximab**) shown some beneficial effect in disseminated disease.

Prognosis

Overall 5-year survival for colorectal cancer is about **50%**. The most important determinant is tumour stage, esp. lymph node status.

- ∞ **Dukes' stage A:** Usually cured by surgical resection, with **90%** disease-free survival at 5 years.
- ∞ **Dukes' stage B:** 5-year survival of **60–70%**.
- ∞ **Dukes' stage C:** 5-year survival of **30%**.
- ∞ **Dukes' stage D:** 5-year survival of **10%** or less.

The End!