Pathology of the large intestine and the peritoneum

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Oktatás, kutatás, gyógyítás: 250 éve az egészség szolgálatában

Topics

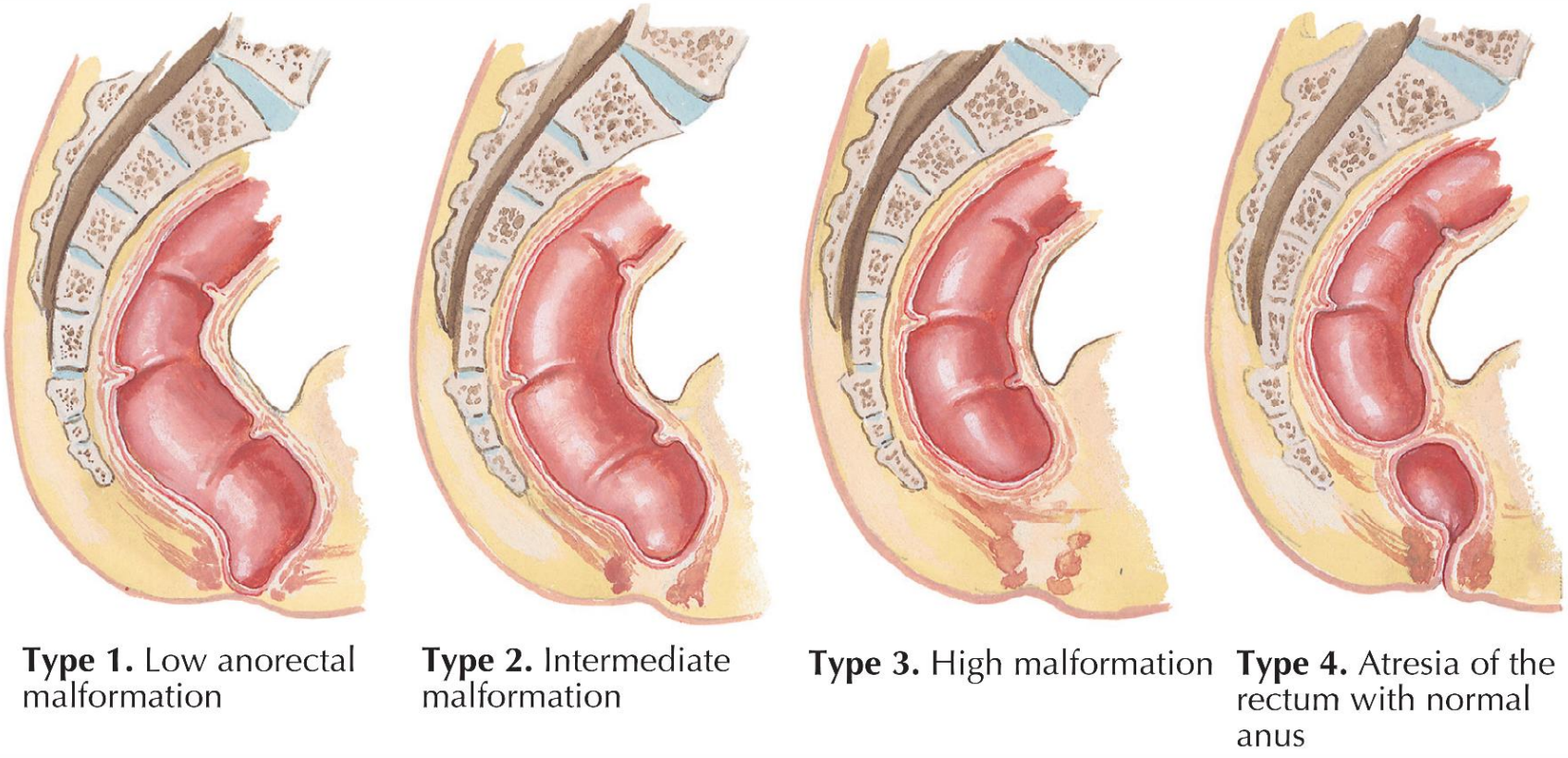
Colon
Appendix
Peritoneum

Developmental malformation
Inflammation
Tumor

Colon
Developmental malformation

ANUS IMPERFORATUS

A cloaca-membran is sill present and the anus is closed with a membrane
Th.: surgical



Type 1. Low anorectal malformation

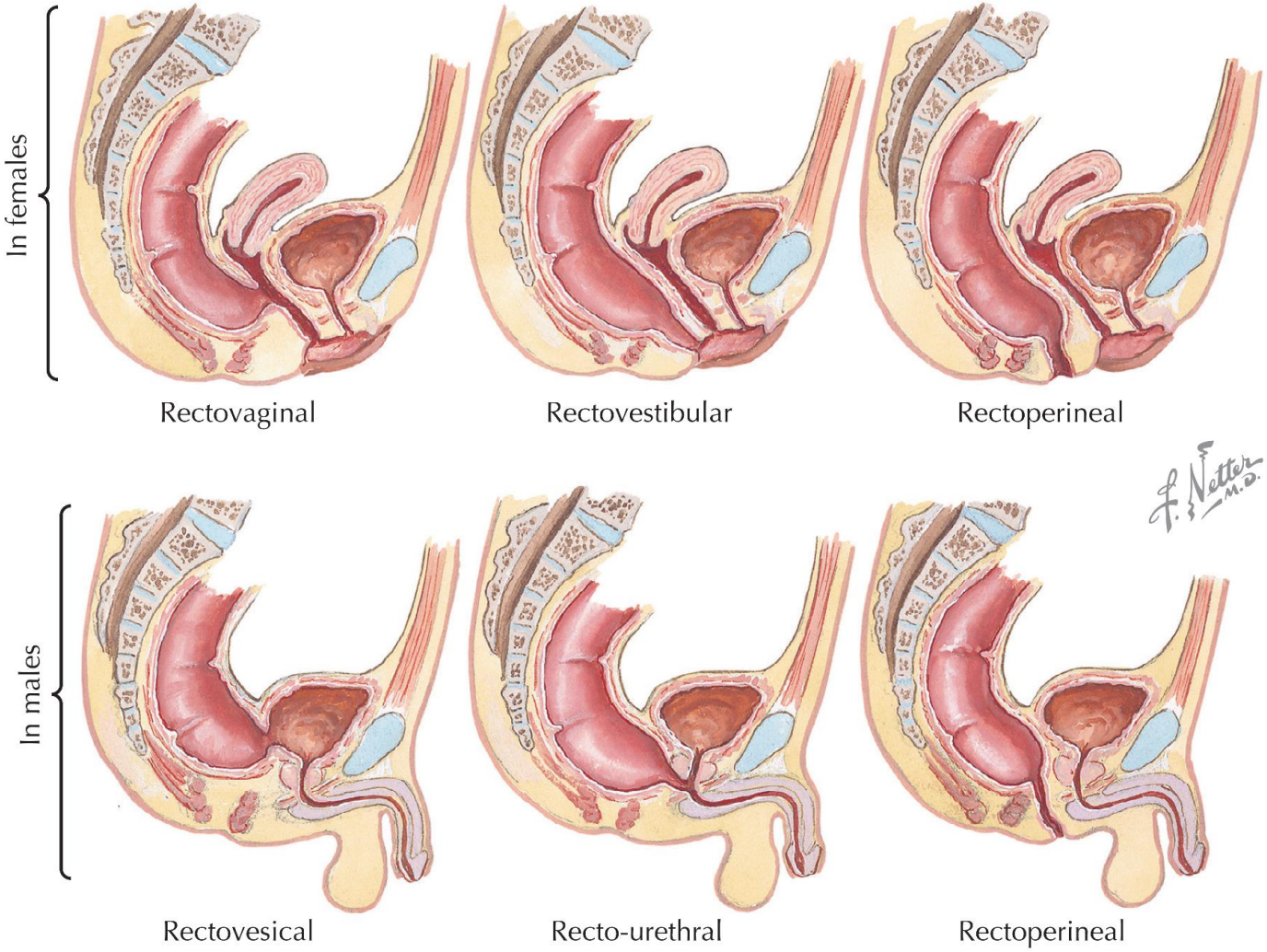
Type 2. Intermediate malformation

Type 3. High malformation

Type 4. Atresia of the rectum with normal anus

Colon
Developmental malformation

Perianal fistula



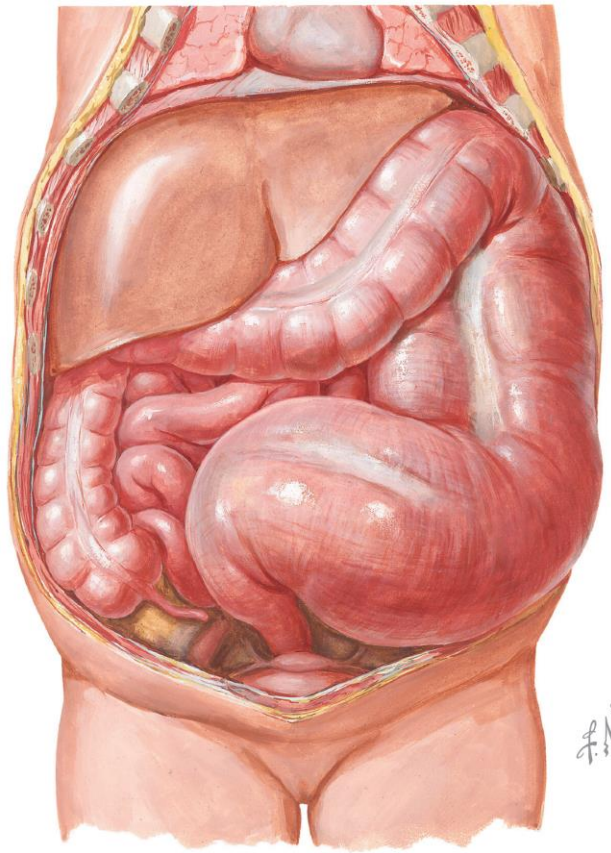
Colon
Developmental malformation
Hirschprung

Features

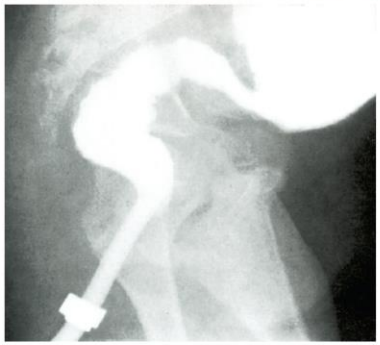
Chronic constipation and gradual bloating of the abdomen in infancy; danger of enterocolitis, perforation

Contrast enema is used to establish the diagnosis; full-thickness biopsy of the rectum reveals an absence of ganglion cells

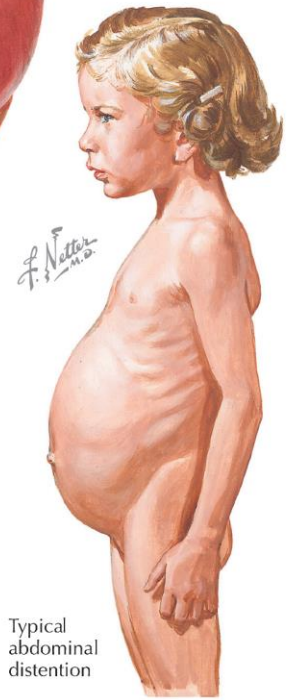
Th.: resection of the aganglionic segment



Tremendous distention and hypertrophy of sigmoid and descending colon; moderate involvement of transverse colon; distal constricted segment



Barium enema; characteristic distal constricted segment



Typical abdominal distention

Colon
Developmental malformation
Hirschprung

Absence of ganglia in the rectum + sigmoid colon

peristalsis is prevented in the rectum resulting in a functional obstruction

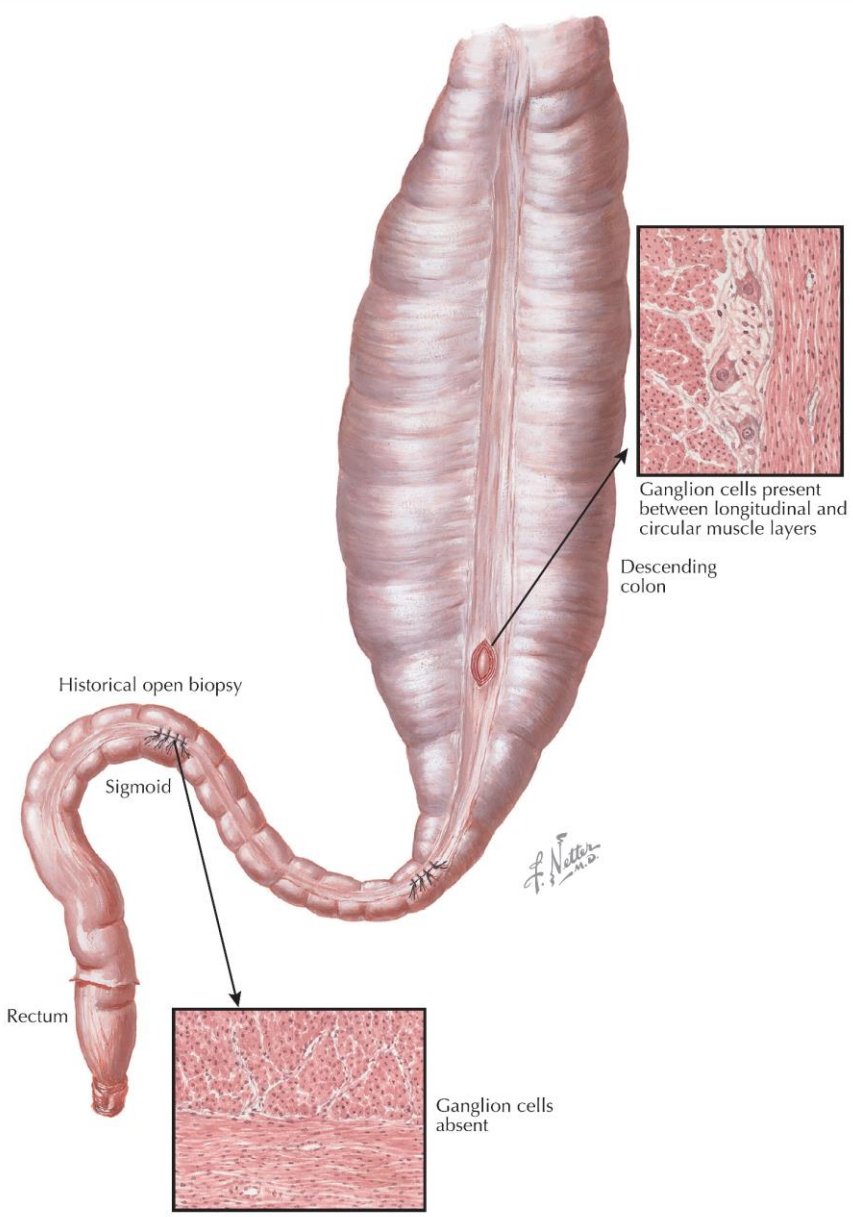
Males predominate

Morphology

The rectum lacks both Meissner'submucosal and Auerbach' myenteric plexuses

Extreme dilation of the normally innervated colon proximally to the aganglionic segment (megacolon)

The dilated bowel segment is filled with stool



Colon Diverticulosis

Small saclike outpouchings of the colon (5 to 10 mm in diameter)

Usually limited to the sigmoid colon

Frequent in Western countries in people above 60 ys

Pathomechanism

Low fiber diet, reduced bulk of stool, forced peristaltic contractions with abnormal elevations of intraluminal pressure to push stool towards the anal canal

Focal weakness of colonic wall beside the taeniae and the penetrating vessels allows mucosal outpouching when the intraluminal pressure is markedly increased

Morphology

Hypertrophied tunica muscularis + diverticula

Ulceration of the herniated mucosa by fecoliths

Purulent diverticulitis and peridiverticulitis

Complications

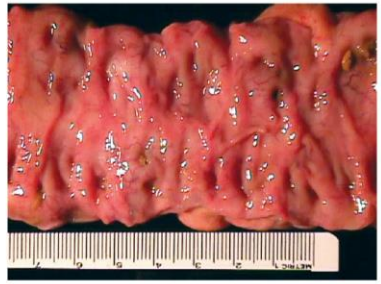
Perforation, pericolonic abscess or peritonitis

Relapses of diverticulitis, mural fibrosis, stenosis of sigmoid bowel

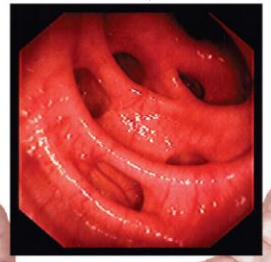
Clinical features

Left-sided lower-quadrant pain, bloody stool, fever, leukocytosis

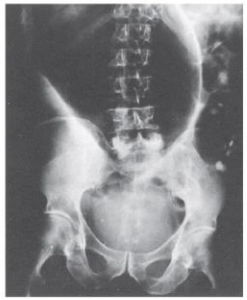
Colonic diverticulosis
(From King TC. Elsevier's Integrated Pathology, Elsevier, Philadelphia , 2006, F9-12.)



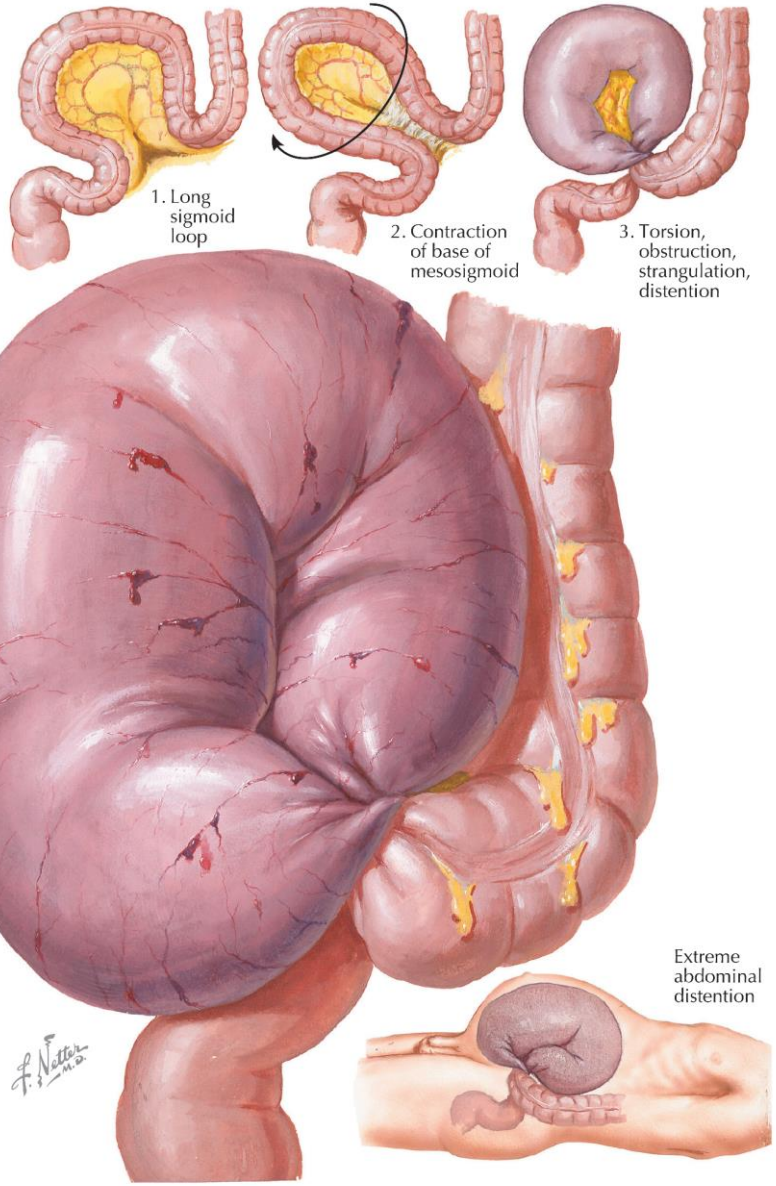
Colonoscopic view



Colon
Obstruction



Volvulus of sigmoid



Consequences of intestinal obstruction

The bowels proximal to the obstruction undergo progressive dilation, their wall becomes thinned, and their lumen is filled with large amount of fluid and gas because of increased secretion of fluid and electrolytes into the distended bowel segment and gas-producing bacterial overgrowth in the stagnating intestinal content

Strangulation-induced bowel infarction, peritonitis

Elevation of the diaphragm

Clinical features of intestinal obstruction

Colicky abdominal pain and distension, constipation without passage of wind, progressive

Volvulus

A rotation (axial twisting) of a mobile loop of intestine around its own mesenteric root

Mostly in the sigmoid colon

Consequence: strangulation, hemorrhagic infarction

Colon
Intussusception

Invagination (intussusception)

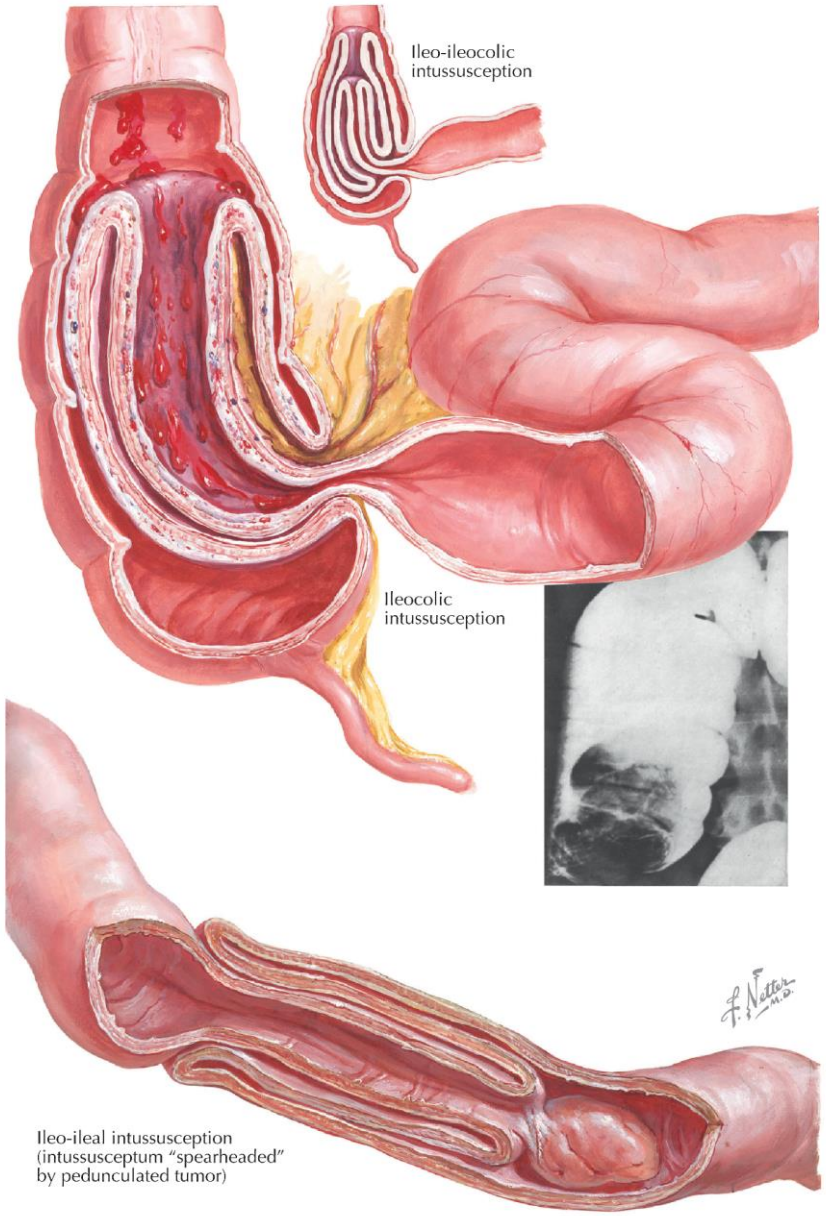
The proximal loop invaginates („telescopes”) into the immediately distal loop, like the finger of an inverted glove

Occurs

In children with very active peristalsis, e.g., in *rotavirus*-induced enteritis

Pedunculeted tumors carried by peristalsis may pull forward the loop to which such a tumor is attached

Consequence: the inner loop (intussusceptum) is strangulated by the outer intussuscipient, and becomes necrotic within hours



Colon
Inflammation
Pseudomembranous colitis

Clostridium difficile infection

Pathogenesis

Important nosocomial pathogen

Broad-spectrum antibiotic therapy eradicates the normal bacterial flora of the gut, the bacteria start to multiply and colonize in the intestinal crypts

Morphology

Pseudomembranous inflammation, most commonly in the rectosigmoid area

Plaques of yellow fibrin and inflammatory debris adherent to a reddened mucosa

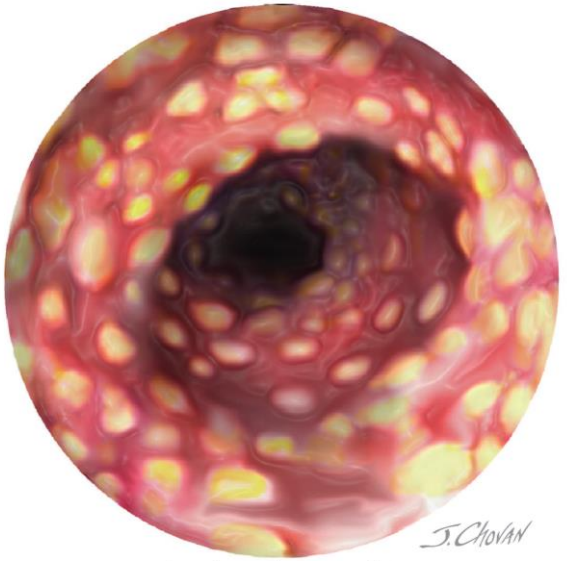
In fulminant cases, the entire colon becomes involved and displays marked dilation, termed toxic megacolon; the pseudomembrane formation can affect the ileum

Clinical features

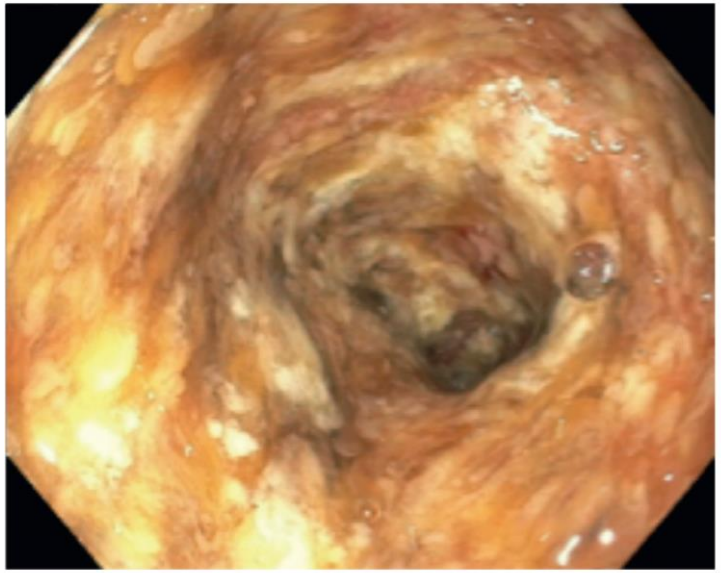
Abdominal pain, diarrhea that may be bloody; leukocytosis; fever, malaise

Fulminant colitis: severe abdominal pain with or without diarrhea, and symptoms of severe SIRS

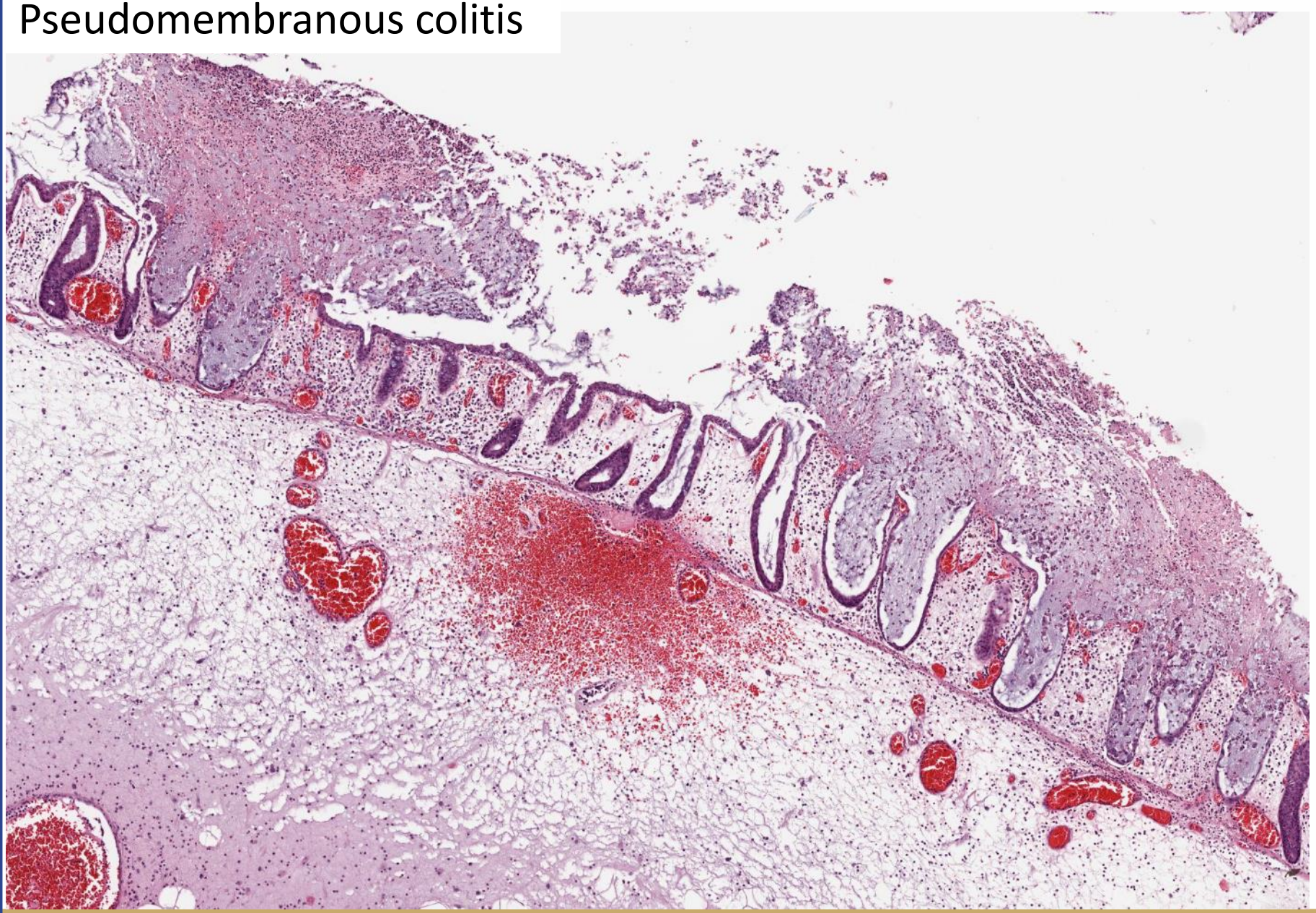
Complications: perforation, prolonged ileus, toxic megacolon, death



Pseudomembranous colitis


C. difficile colitis

Pseudomembranous colitis



Colon
Inflammation

Infectious enterocolitis

High incidence; frequent cause of death in the developing countries

Transmission: fecal-oral, by contaminated food and water

Dg.: by stool culture

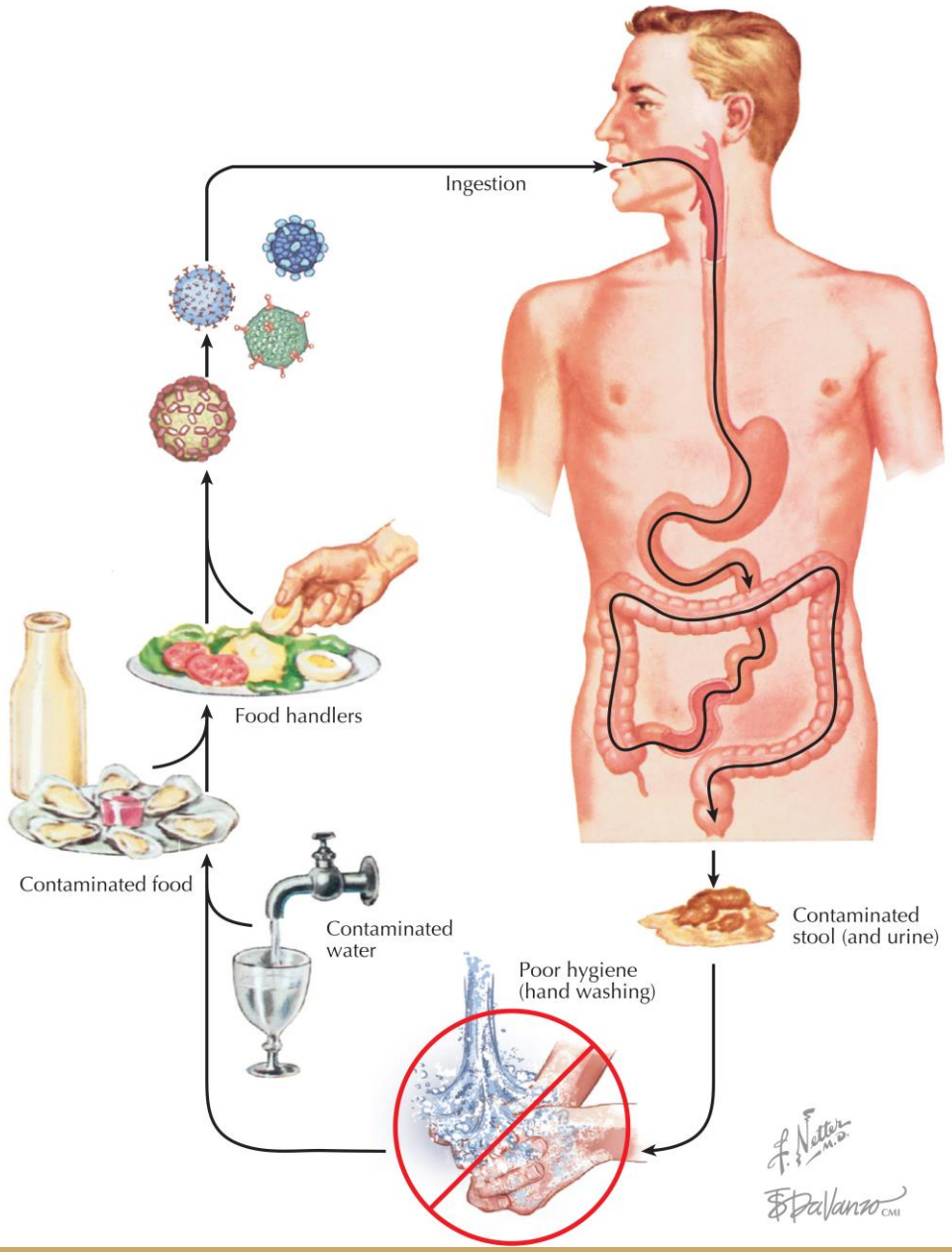
Acute viral enteritis

Rotavirus or adenovirus: mainly in infants and children;

Calicivirus: in all age groups

Pathomechanism: cytopathic damage to the small bowel epithelium, secretory diarrhea

Features: sudden onset of nausea, vomiting and profuse watery diarrhea, fluid and electrolyte loss



Colon Inflammation

Acute bacterial enterocolitis

Pathomechanism

Ingestion of preformed toxin in contaminated food as in St. aureus-induced food poisoning, vomits, explosive watery diarrhea and acute abdominal distress

Infection by secretory enterotoxin-producing E. coli (ETEC), traveller's diarrhea

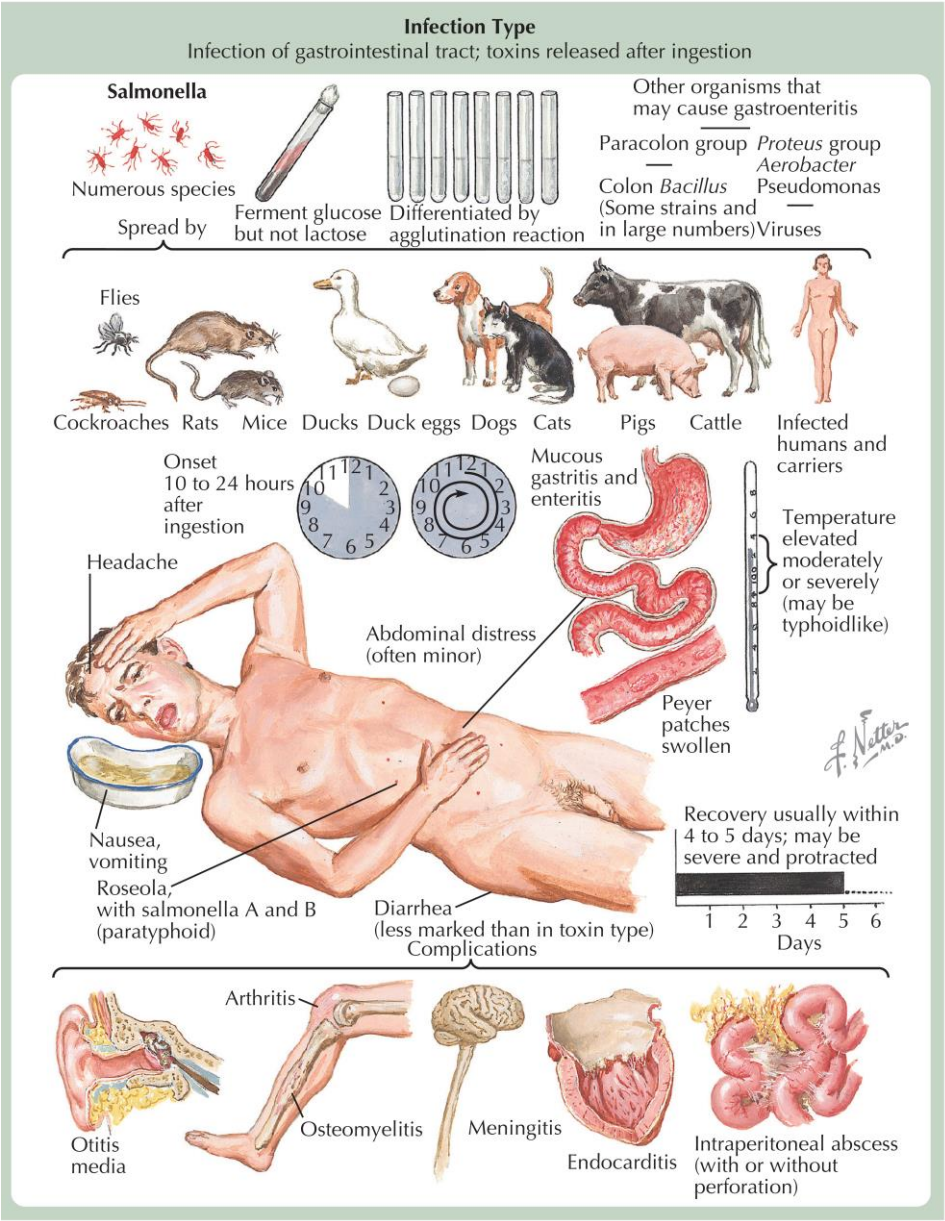
Infection by enteroinvasive organisms (Campylobacter, Salmonella, E. coli, Shigella, Yersinia, etc.) that proliferate, invade, and destroy mucosal epithelial cells; the Shigellae, the enterohemorrhagic E. coli (EHEC) organisms produce enterotoxin, too.

Features of enteroinvasive infections

Induce colitis + enteritis

Nonspecific morphology: the lamina propria is edematous, hyperemic and displays neutrophilic cryptitis, crypt abscess (crypts with accumulations of luminal neutrophils) + ulcers

Lead to dysentery: exudative, small-volume diarrhea; characterized by abdominal cramping and tenesmus in which loose stools contain blood, pus, mucus and necrotic tissue debris



Colon Inflammation

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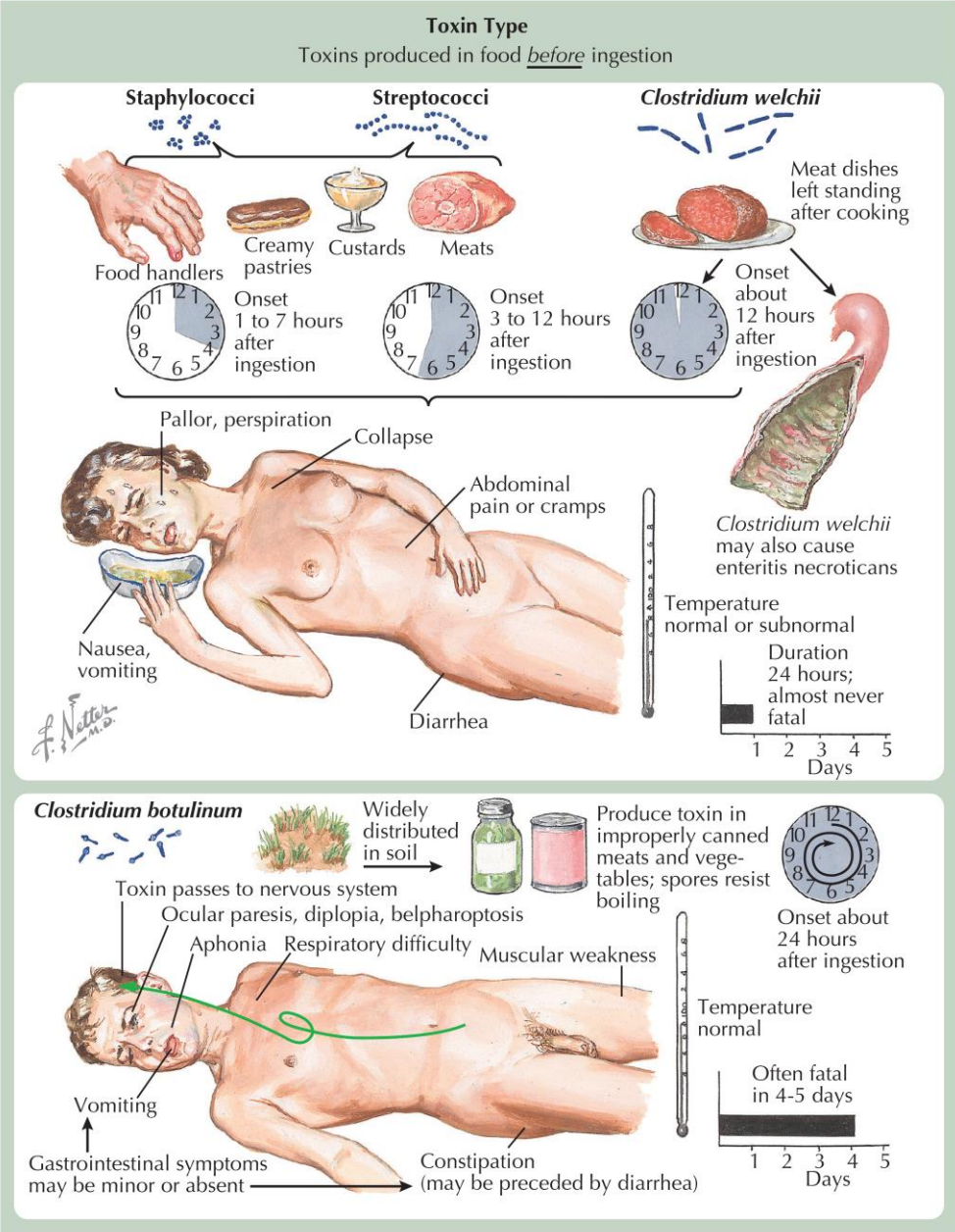


Colon
Inflammation

Non-Infectious enterocolitis

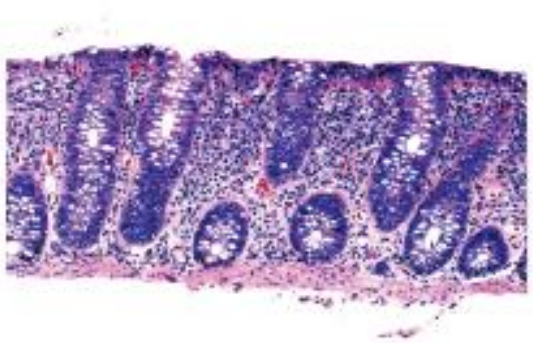
Microscopic colitis

Typically in middle-aged women; presents with chronic watery diarrhea and abdominal pain

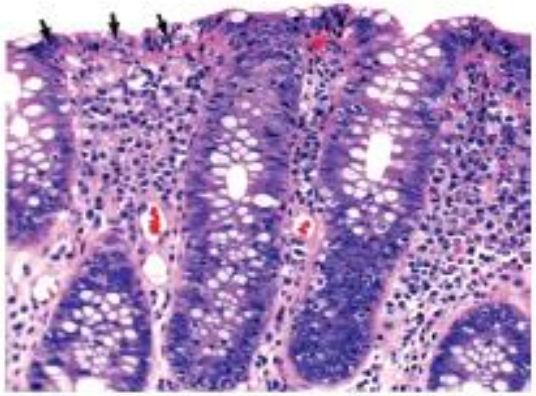
Colonoscopic findings are grossly normal

LM: band-like collagen under the surface epithelium (collageneous colitis) or prominent intraepithelial infiltrate of lymphocytes (lymphocytic colitis; association with autoimmune diseases and celiac disease)

Lymphocytic colitis

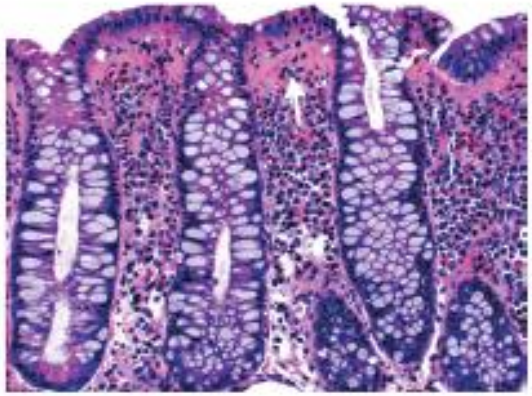


Low-power microphotograph of lymphocytic colitis that shows increased lymphocytic and round cell infiltration in the lamina propria. The crypts appear normal.



High-power microphotograph of lymphocytic colitis (same patient and biopsy as in figure at left). *Arrows* indicate the classic infiltrate of lymphocytes in the epithelium.

Collagenous colitis



Colon
Inflammation
Necrotizing enterocolitis

Pathogenesis

Obscure

In low-birth-weight or premature neonates after oral feeding is instituted

Pathology

Involves the terminal ileum, cecum, and ascending colon

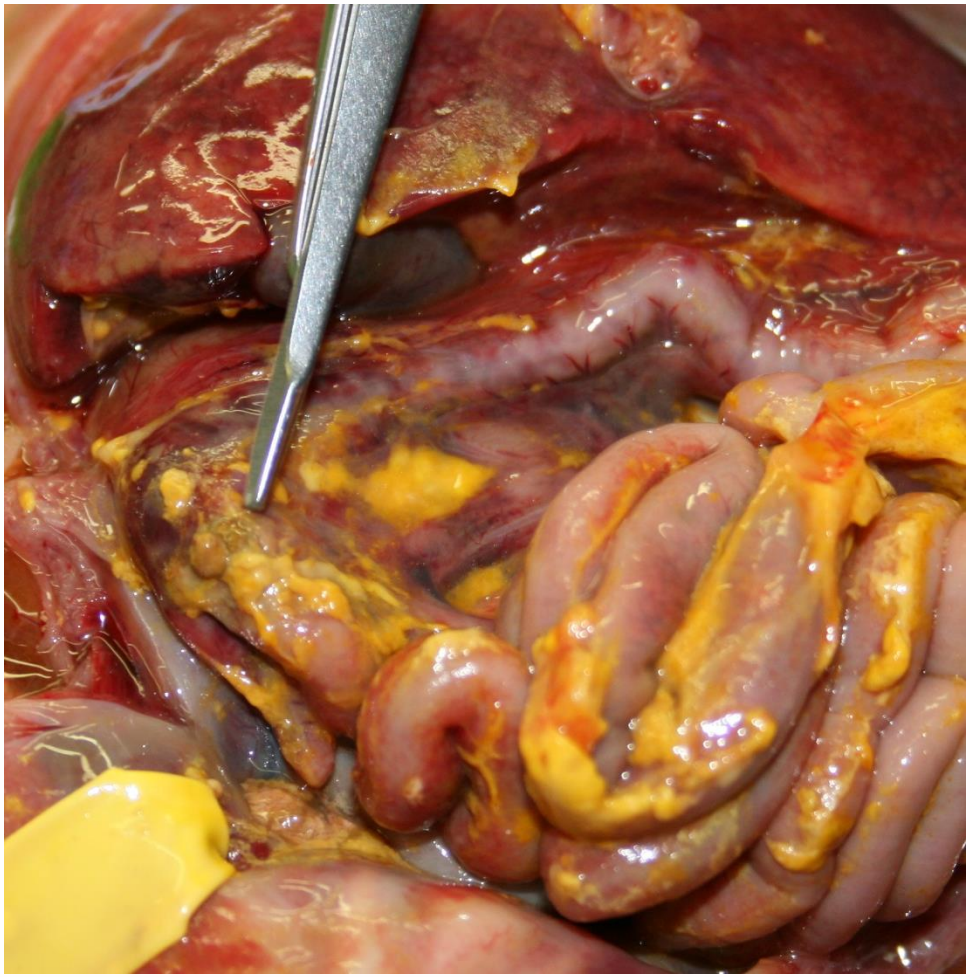
Mucosal edema, hemorrhage, and necrosis + submucosal gas bubbles (pneumatosis intestinalis)

Clinical features

Bloody stool, abdominal distention, shock

High mortality rate

Strictures in survivors



Colon
Inflammation
Inflammatory bowel disease (IBD)

Includes ulcerative colitis (UC) and Crohn's disease (CD); both are chronic, relapsing inflammatory disorders

UC and CD share many features but also differ from another so in typical cases each can be diagnosed

In 20% of cases, the disease cannot be classified as UC or CD: indeterminate colitis

Peak incidence: young adults

Pathogenesis

In a genetically susceptible host, IBD results from dysfunction of tight junctions of surface epithelial cells allowing bacterial antigens to enter the mucosa, where unregulated and exaggerated local inflammatory responses develop

Susceptibility genes, gene mutations.

Feature	Crohn Disease	Ulcerative Colitis
Macroscopic		
Bowel region	Ileum ± colon	Colon only
Distribution	Skip lesions	Diffuse
Stricture	Yes	Rare
Wall appearance	Thick	Thin
Microscopic		
Inflammation	Transmural	Limited to mucosa
Pseudopolyps	Moderate	Marked
Ulcers	Deep, knife-like	Superficial, broad-based
Lymphoid reaction	Marked	Moderate
Fibrosis	Marked	Mild to none
Serositis	Marked	Mild to none
Granulomas	Yes (~35%)	No
Fistulae/sinuses	Yes	No
Clinical		
Perianal fistula	Yes (in colonic disease)	No
Fat/vitamin malabsorption	Yes	No
Malignant potential	With colonic involvement	Yes
Recurrence after surgery	Common	No
Toxic megacolon	No	Yes

Colon
Inflammation
Ulcerative colitis

Affects the mucosa + submucosa of the rectum (proctitis) or rectosigmoid colon (distal colitis)

10%: the entire colon is affected (pancolitis)

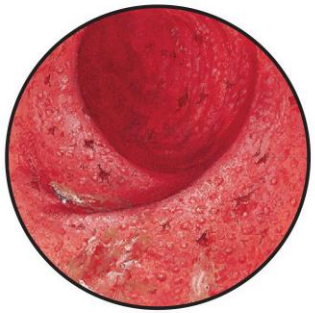
Extends in a continuous fashion proximally from the rectum

Exacerbations are often triggered by emotional or physical stress

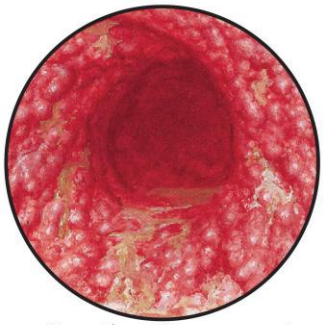
Macroscopy

Broad-based ulcers irregular in outline and orientation, ranging up to many cm-s

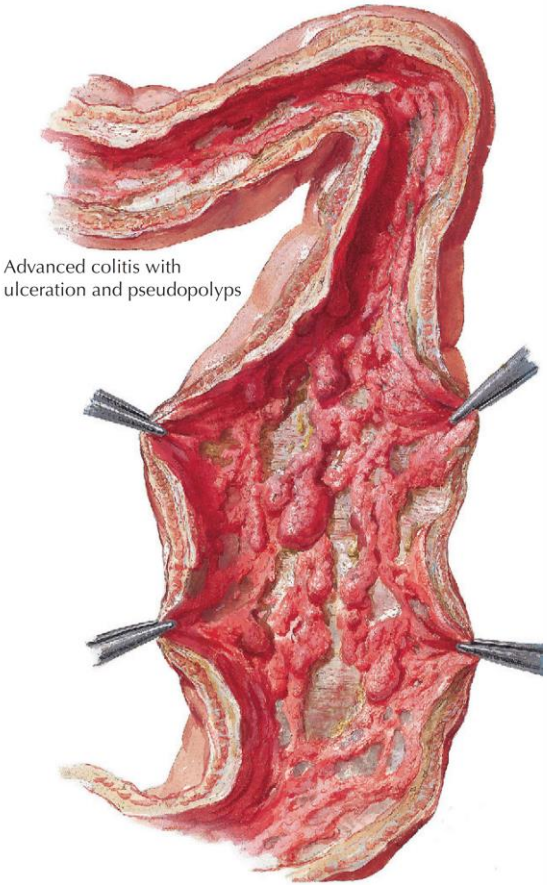
The ulcers are separated by narrow strands of edematous hyperemic mucosa bulging upwards, called pseudopolyps



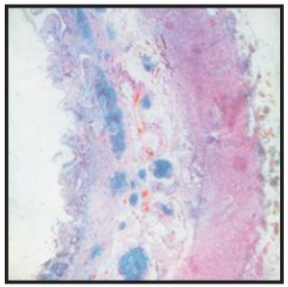
Sigmoidoscopic appearance in moderate colitis



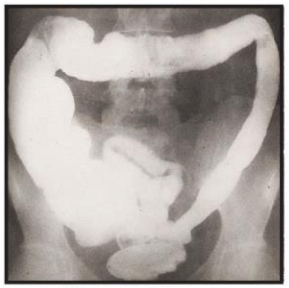
Sigmoidoscopic appearance in severe colitis



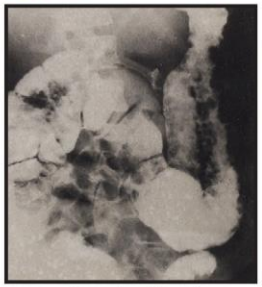
Advanced colitis with ulceration and pseudopolyps



Micropathology



Contracted bowel



Pseudopolyposis

Colon
Inflammation
Ulcerative colitis

LM

Intense infiltration of the mucosa by granulocytes, ly-s and plasma cells

Cryptitis, crypt abscesses

Necrosis of epithelium; enlargement of necrotic areas produce the grossly visible ulcers

Healing

Granulation tissue fills the ulcer craters, followed by regeneration of the mucosal epithelium

Clinical features, acute phase

Bleeding from ulcers

Severe bloody diarrhea; intense pain at defecation

Fever

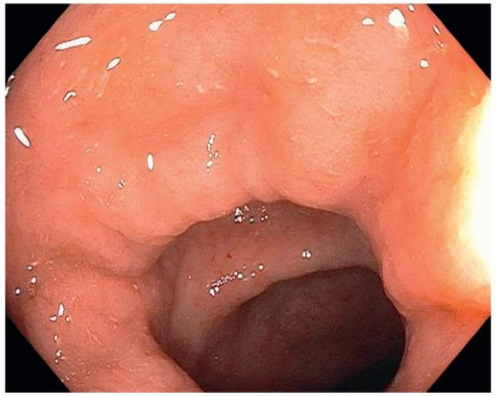
In severe cases: toxic megacolon; ulcers may perforate

Chronic phase

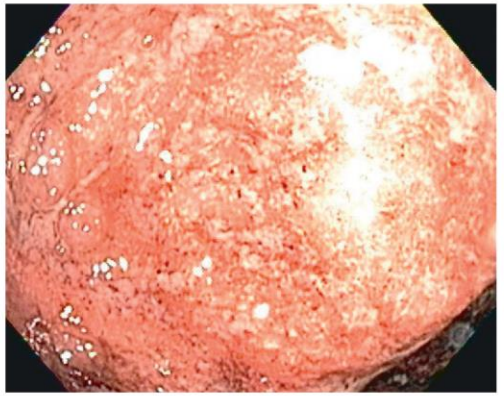
Progressive mucosal atrophy

Epithelial dysplasia

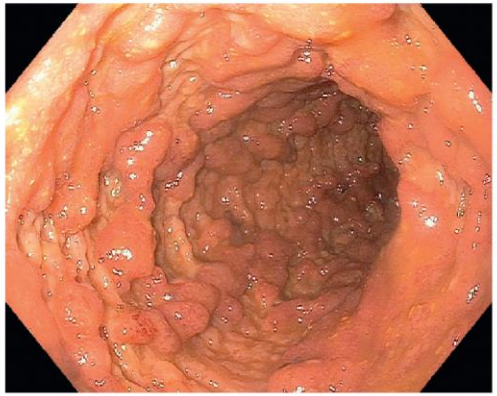
Development of colon cc - in relapsing disease lasting for 25 years, the risk is 10%



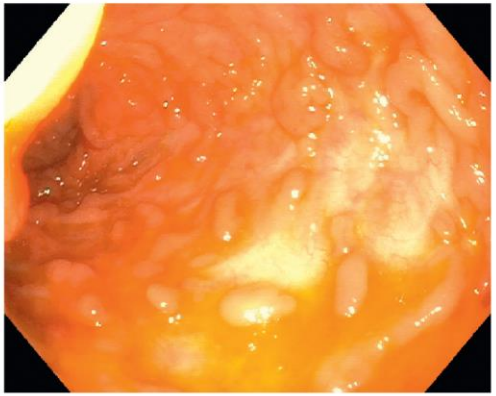
Mild ulcerative colitis



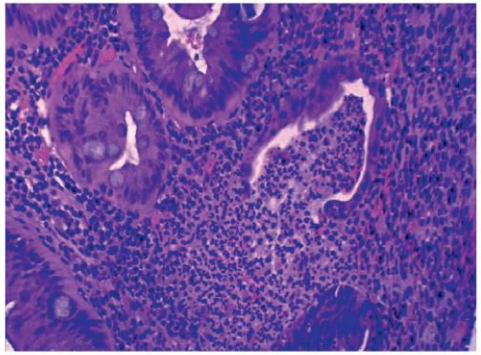
Moderate ulcerative colitis



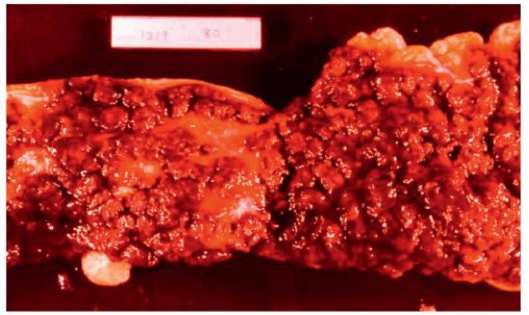
Severe ulcerative colitis



Pseudopolyps in ulcerative colitis



Active chronic colitis with crypt abscess characteristic of ulcerative colitis but not specific.



Ulcerative colitis, gross. Flat superficial ulcers with many inflammatory pseudopolyps.

Colon
Inflammation
Crohn

Involvement:

- small intestine 40% (terminal ileitis)
- large intestine 30%
- small and large intestine 30%

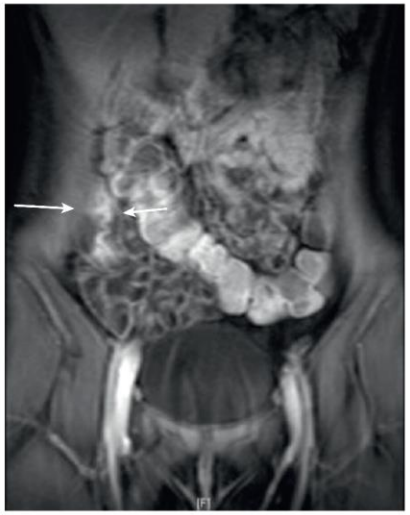
Gross features

Segmental involvement, sharply demarcated from adjacent normal bowel („skip lesion”)

Deep ulcers in the long axis of the bowel (**serpentine fissures**) separated by nodular mucosal thickenings, cobblestone appearance

Subsequent fibrosis of the wall, **stricture** of the involved segments, particularly of the terminal ileum

Extension of fissures leads **to fistula formation** to other loops of bowel, the urinary bladder, vagina or perianal skin + peritoneal abscess(es)



33-year-old woman with Crohn ileitis. MR enterography demonstrates a 5 cm distal terminal ileal stricture.

Colon
Inflammation
Crohn

LM

Fissure ulcers, extending into the tunica muscularis

Transmural aggregates of lymphocytes and fibrosis

Noncaseating granulomas in 35% of cases

Clinical features

Onset

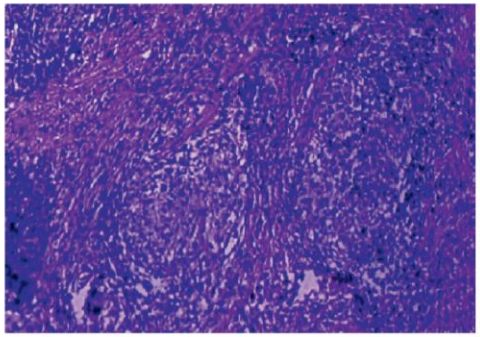
In most patients: intermittent attacks of relatively mild diarrhea, abdominal pain, and fever

In minority of patients: appendicitis-like symptoms + bloody diarrhea

Years later

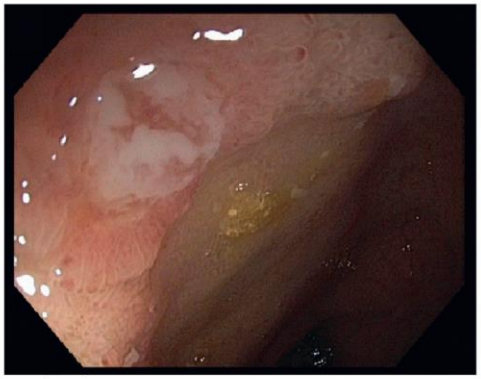
Small bowel manifestation - malabsorption, hypoalbuminemia (protein-loosing enteropathy)

Colonic manifestation - iron deficiency anemia

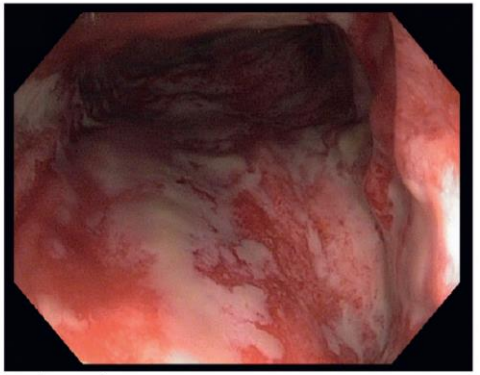


Epithelioid granulomatous reaction associated with chronic inflammation in the muscularis of colon in a patient with CD.

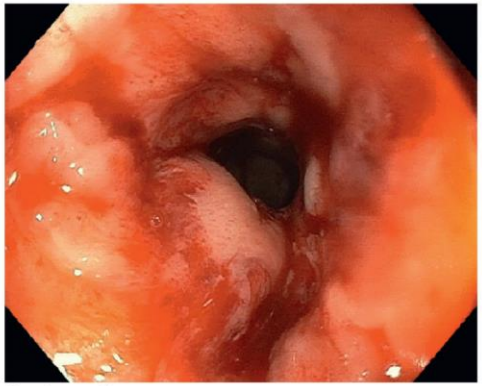
Linear deep fissure like ulcer, colon. Crohn.



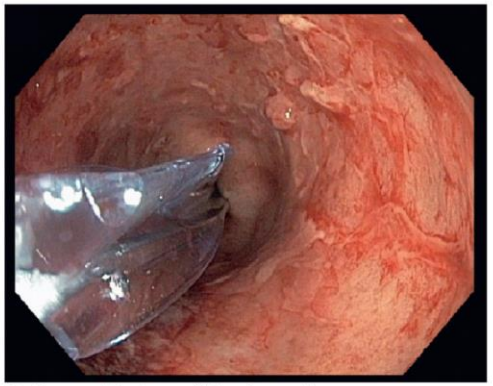
Crohn ileitis



Crohn coliitis



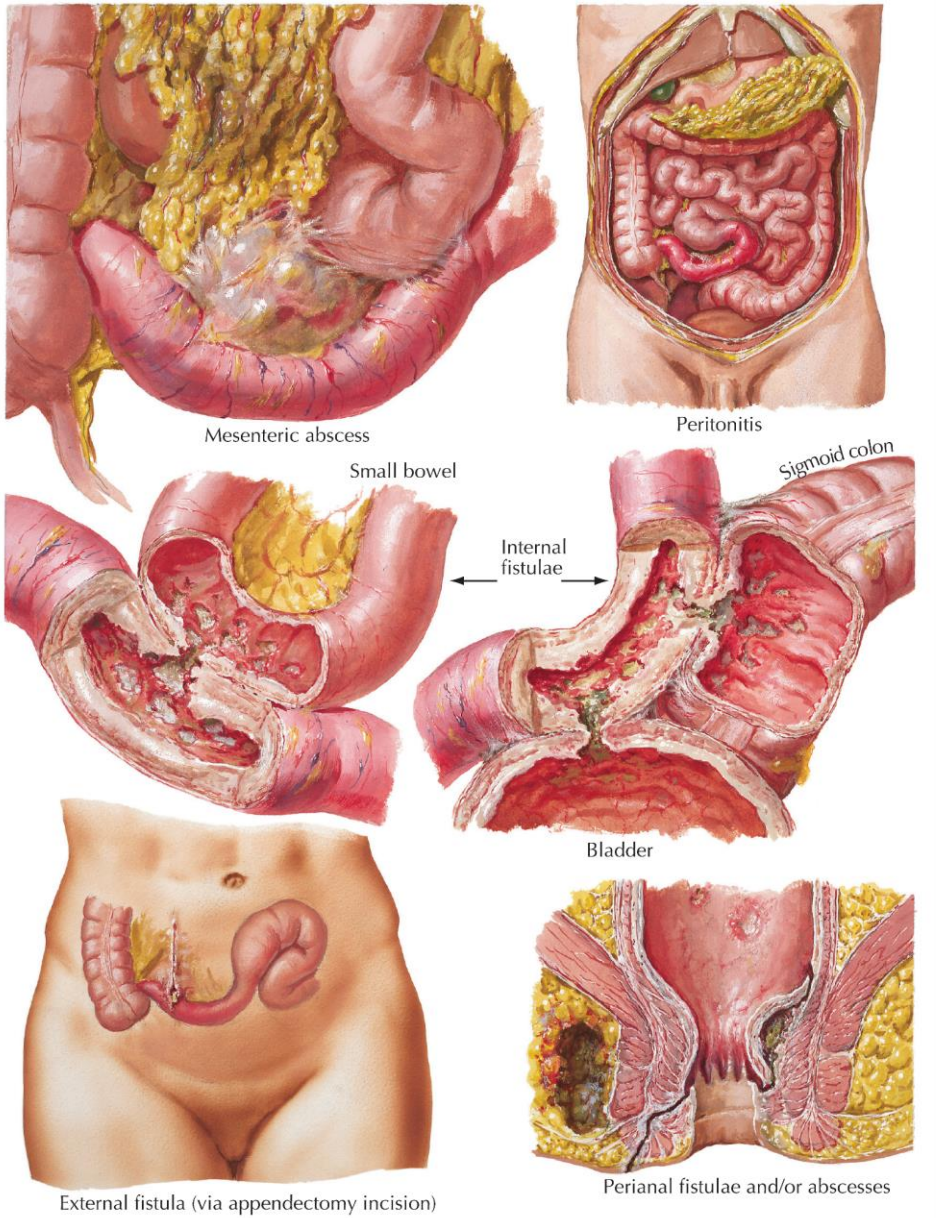
Crohn stricture



Balloon dilation of Crohn stricture

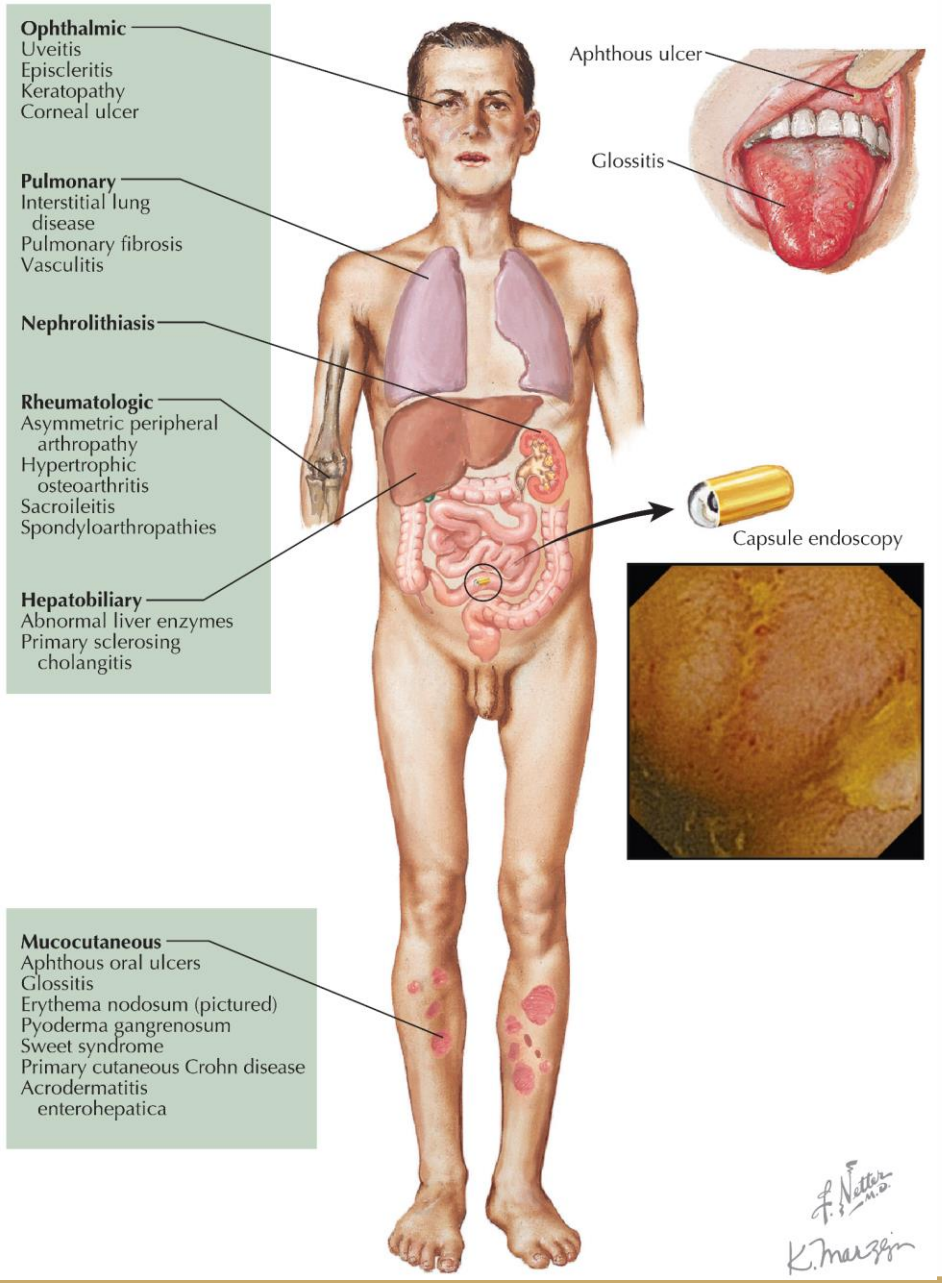
Colon
Inflammation
Crohn

Complications



Colon
Inflammation
Crohn

Extra-gastrointestinal complications of CD



Colon
Inflammation
Ischaemic colitis

Ischemia causes pathologic changes when the perfusion of the intestines declines below 50% of normal

Ischemia due to occlusion - transmural infarction

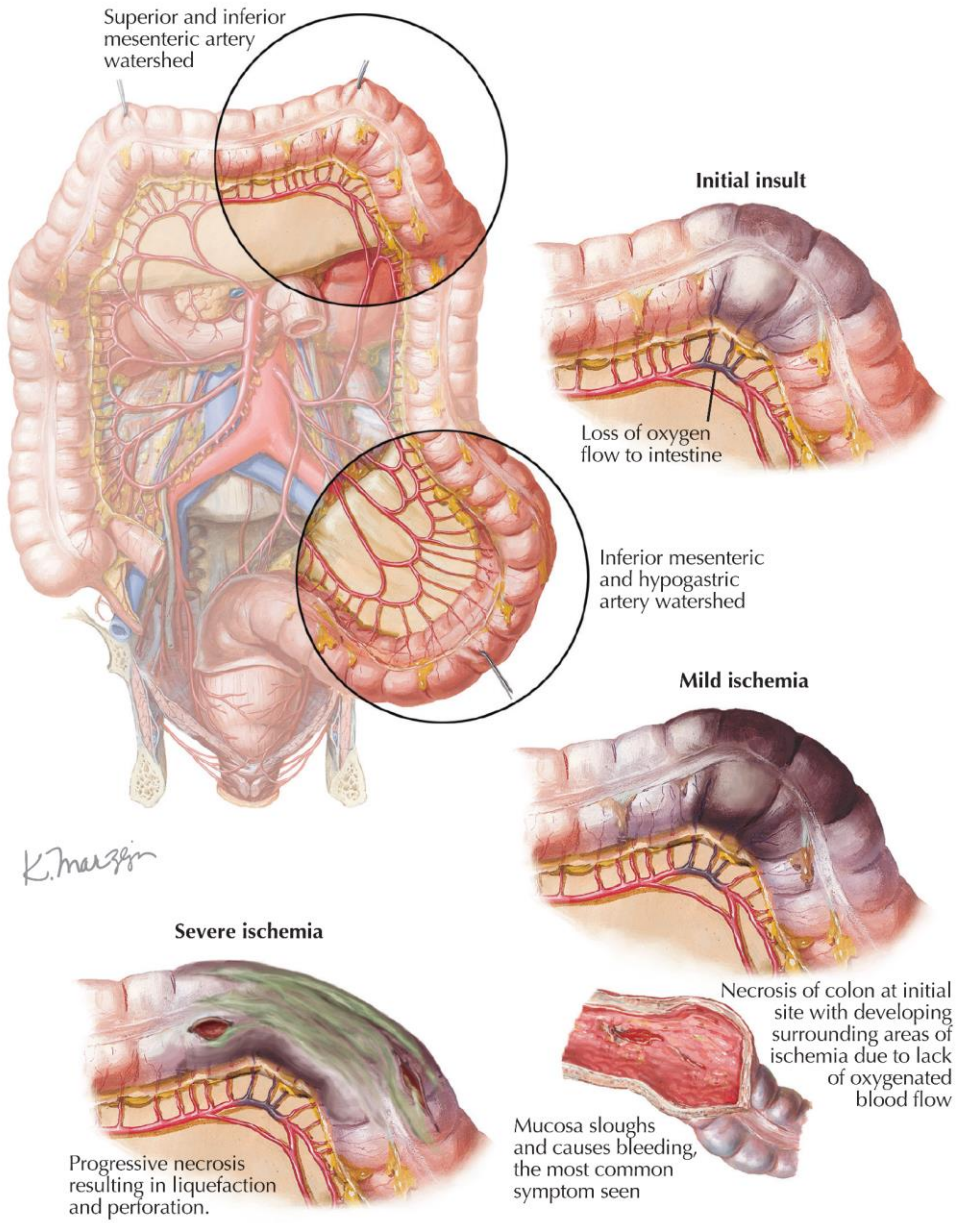
Ischemia due to stenosis - mucosal or mural infarction

Occlusive ischemia

The trunk of superior mesenteric artery is occluded by thrombosis over ruptured atheroma (frequent) or embolism

Consequence

Transmural hemorrhagic infarction of the bowel *(the necrotic area is reperfused by the blood coming from numerous anastomoses in the intestines)*



Colon
Inflammation
Ischaemic colitis

Clinical features

Progressively increasing abdominal pain (thrombosis) or sudden onset of abdominal pain (embolism) + bloody diarrhoea

Shock and vascular collapse within hours

Mortality rate: 90%

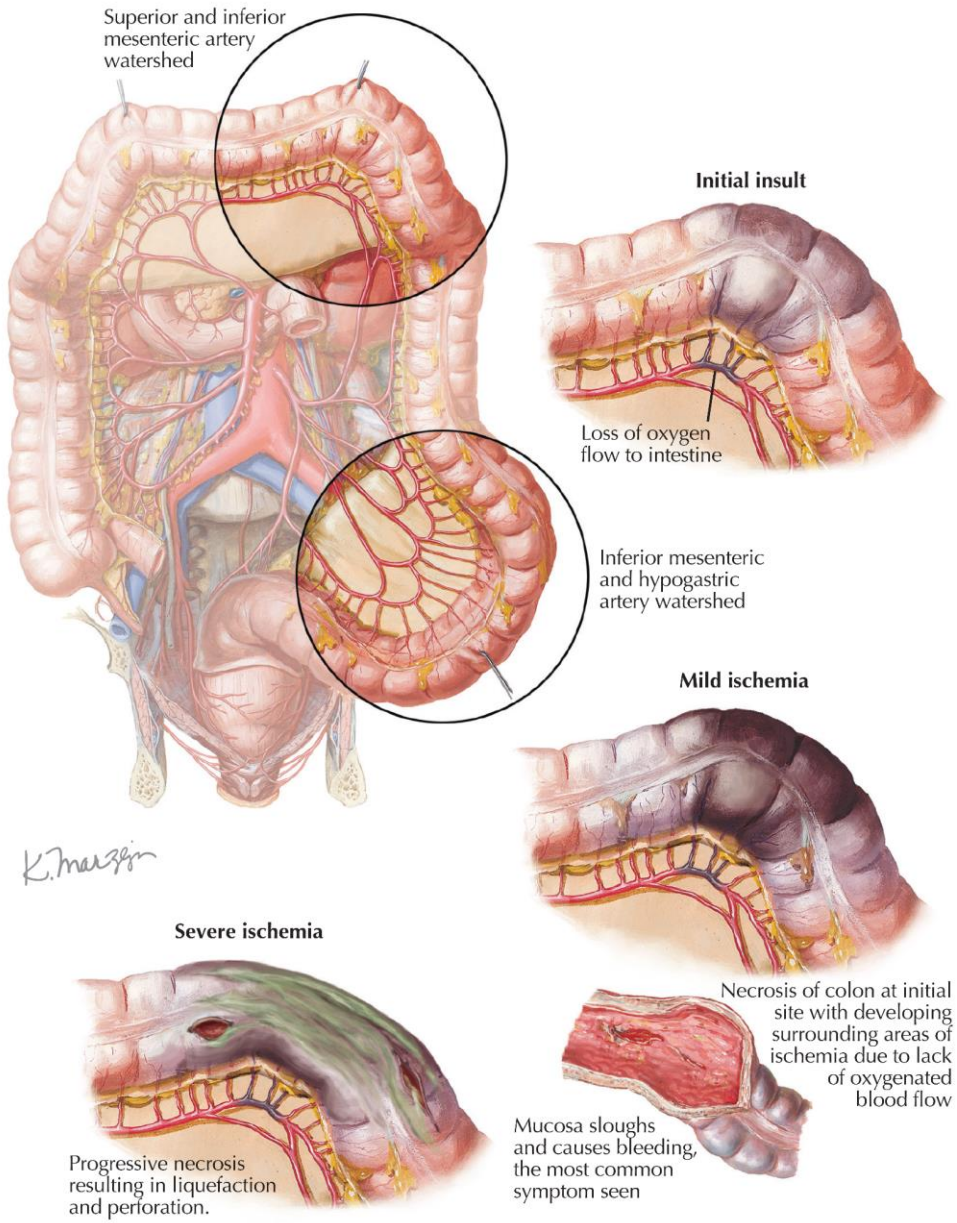
Nonocclusive ischemia

In elderly people with severe atherosclerosis of the intestinal arteries

The onset of ischemia is precipitated by sudden drop in intestinal perfusion due to acute myocardial infarction, pulmonary embolism, prolonged hypotension of any cause

Depending on the degree of narrowing of the arteries, the infarction may be **mucosal** or **mural** (mucosa + submucosa + tunica muscularis)

The watershed border zones are affected: the splenic flexure of the colon (watershed between the distribution of the superior and inferior mesenteric arteries); the rectosigmoid junction (between the branches of the inferior mesenteric artery)



Colon
Inflammation
Ischaemic colitis

Gross

Mucosa: edema, hemorrhagic thickening; several ulcerations (the necrotic mucosa detaches) which may be covered by pseudomembrane; serosal surface: normal

Healing state („ischemic colitis”): pseudopolyps may be present in addition to ulceration and fibrosis

Healing

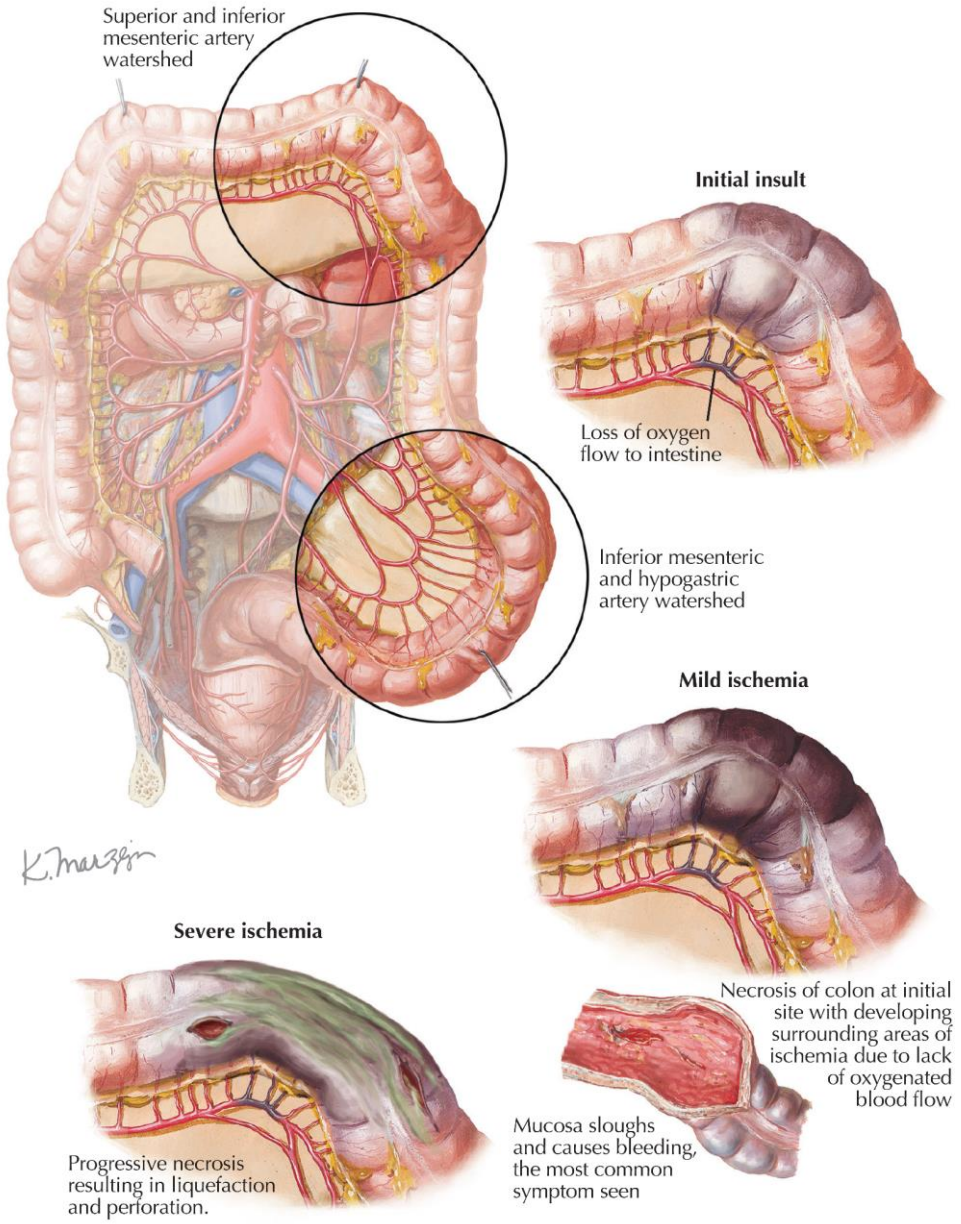
Mucosal ulcerations heal completely; mural infarctions heal with fibrosis, stricture formation

Clinical features

Abdominal pain/discomfort + bloody stool

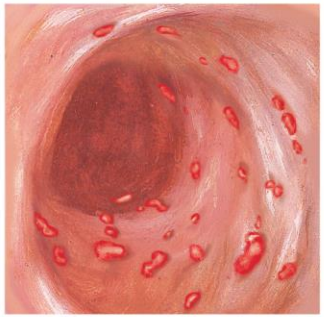
May mimic both acute enterocolitis from other causes and idiopathic inflammatory bowel disease

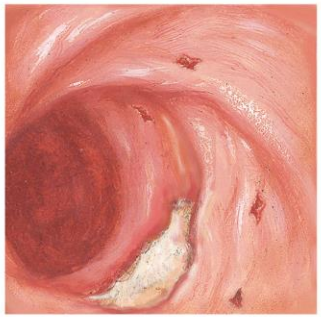
The bowel lesions, *per se*, are not lethal; if the cause of hypoperfusion can be corrected, the outcome is good

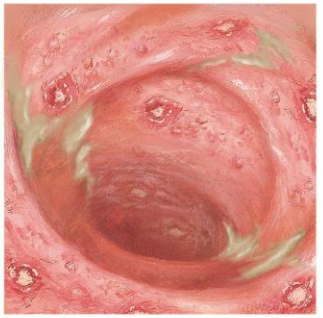


Colon
Inflammation
HIV/AIDS

HIV-Associated Gastrointestinal Pathogens			
Pathologic Process	**Small Bowel**	**Colon**	**Ano-Rectal**
Inflammatory	HIV enteropathy	HIV enteropathy	
Viral	CMV	CMV HSV	CMV HSV HPV HHV8
Bacterial	*Mycobacterium avium-intracellulare* *M. tuberculosis*	Bartonella *M. tuberculosis* *Clostridium difficile* Salmonella Shigella Campylobacter LGV	LGV Chlamydia Syphilis
Parasitic	Cryptosporidia Cystoisospora Giardia Microsporidia Strongyloides		
Fungal	Histoplasmosis	Cryptococcus Histoplasmosis	
Neoplastic	Kaposi sarcoma	Kaposi sarcoma (HHV8) Lymphoma	Anal carcinoma


Bacillary angiomatosis of colon


CMV ulcer, colon


CMV colitis

Colon Tumor Polyps

Tumor-like or tumorous protrusions of the intestinal mucosa

Classification

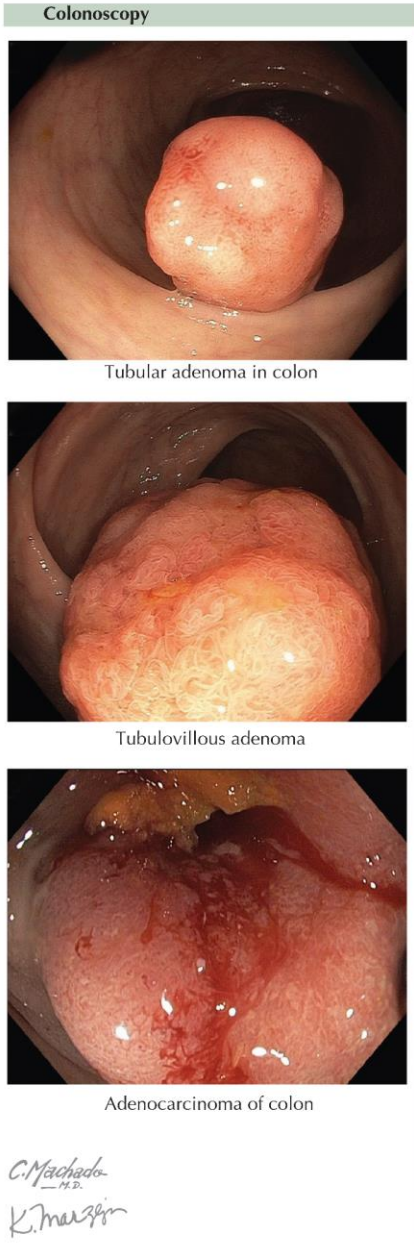
1. Non-neoplastic

Inflammatory (pseudopolyps): in ischemic colitis, ulcerative colitis

Hyperplastic: sessile lesions of the distal colon < 0.5 cm in size; ***no*** malignant potential; should be distinguished from sessile serrated adenomas

Hamartomatous

2. Neoplastic: adenomas, high risk of malignant transformation



Colon
Tumor
Polyps (adenoma)

Sporadic adenomas

Precursors of colorectal adenocarcinoma

Classified based on architecture: tubular, villous, or serrated

Tubular adenoma

About half are found in the rectosigmoid; may be single or multiple

Usually <1 cm and pedunculated; LM: tubular glands lined by dysplastic columnar epithelium

>2.5 cm: areas of intramucosal carcinoma can be present (invasion of the lamina propria with no extension through the muscularis mucosae into the submucosa)

Villous adenoma

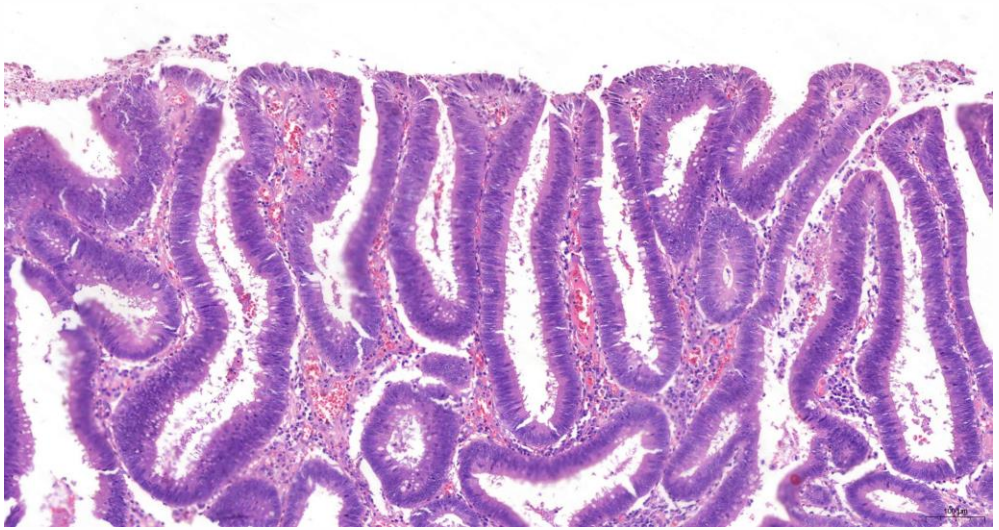
Most often in the rectum; solitary, sessile, diameter: up to 10 cm

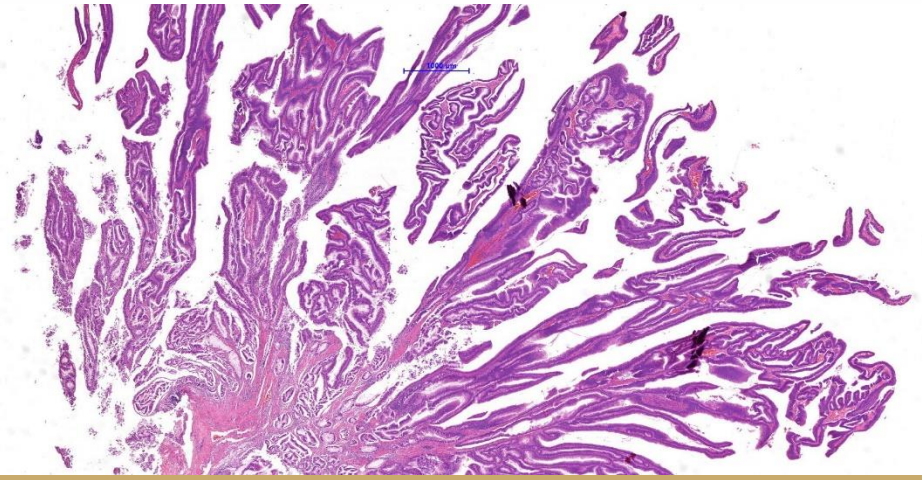
Composed of villi (finger-like protrusions lined with dysplastic columnar epithelium)

Adenocarcinoma frequently arises in VA-s >4 cm in diameter

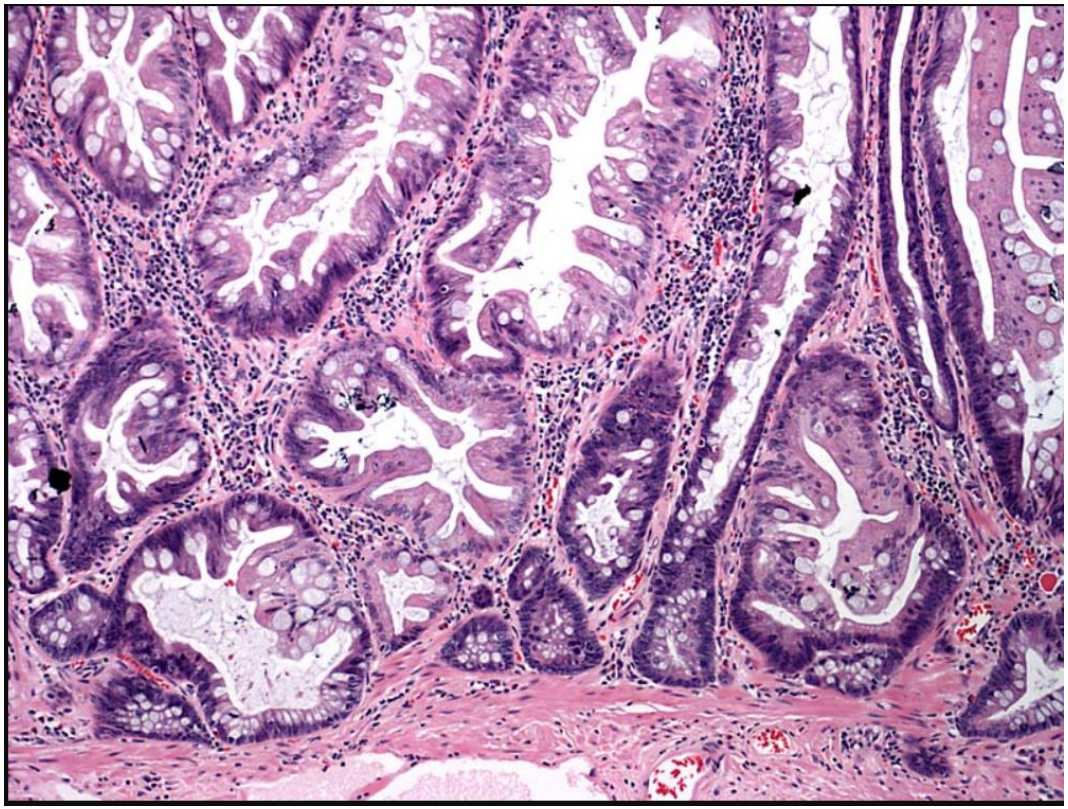
New terminology:

Mucosal neoplasia (with low/high grade dyplasia)





Colon
Tumor
Polyps



Sessile serrated adenoma

Common in the proximal colon; 5 to 15 mm in size

LM: the full gland length exhibits serrated architecture

Despite malignant potential, dysplastic changes are not present

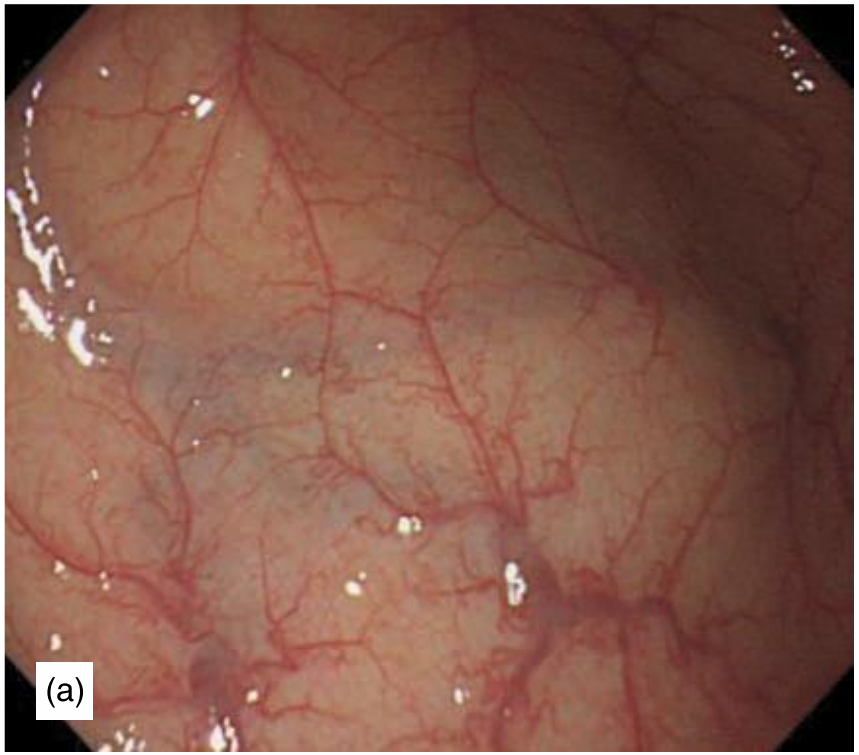
Clinical features

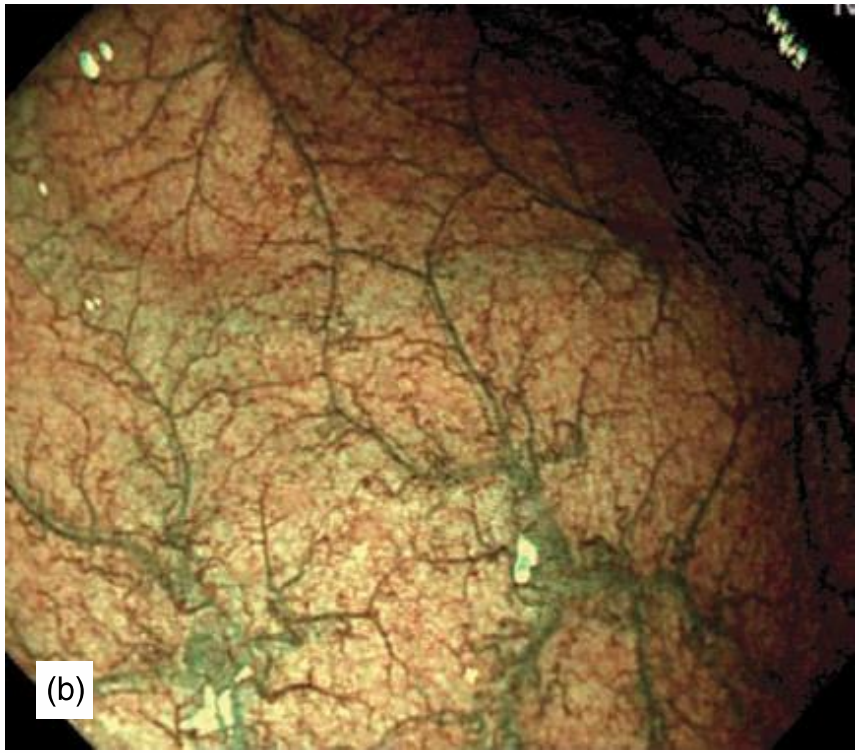
Common after age 60, the incidence increases with age

Most are silent; can cause occult/fresh bleeding; large villous adenomas can hypersecrete protein and K+ into the stool, hypoproteinemia and hypokalemia

Some of them can be removed endoscopically

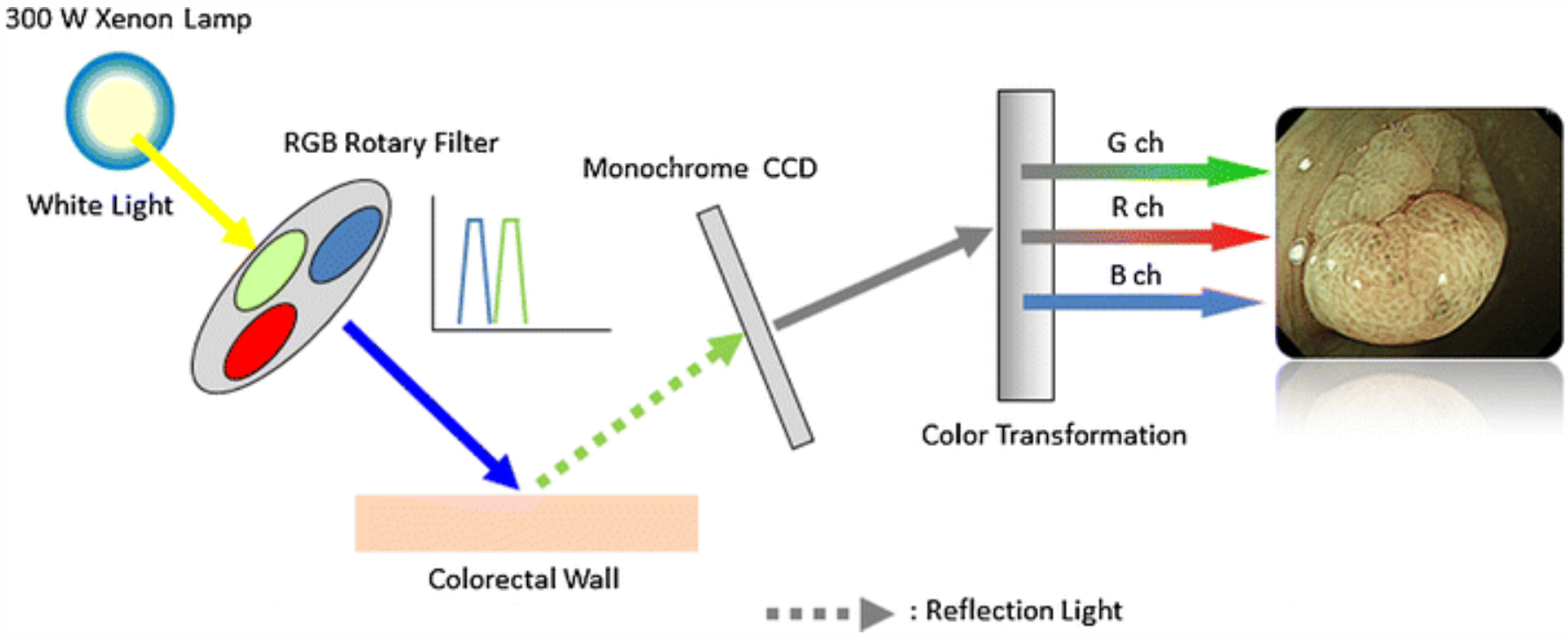
Colon with normal mucosa (brightfield light and NBI=Narrow-band imaging)





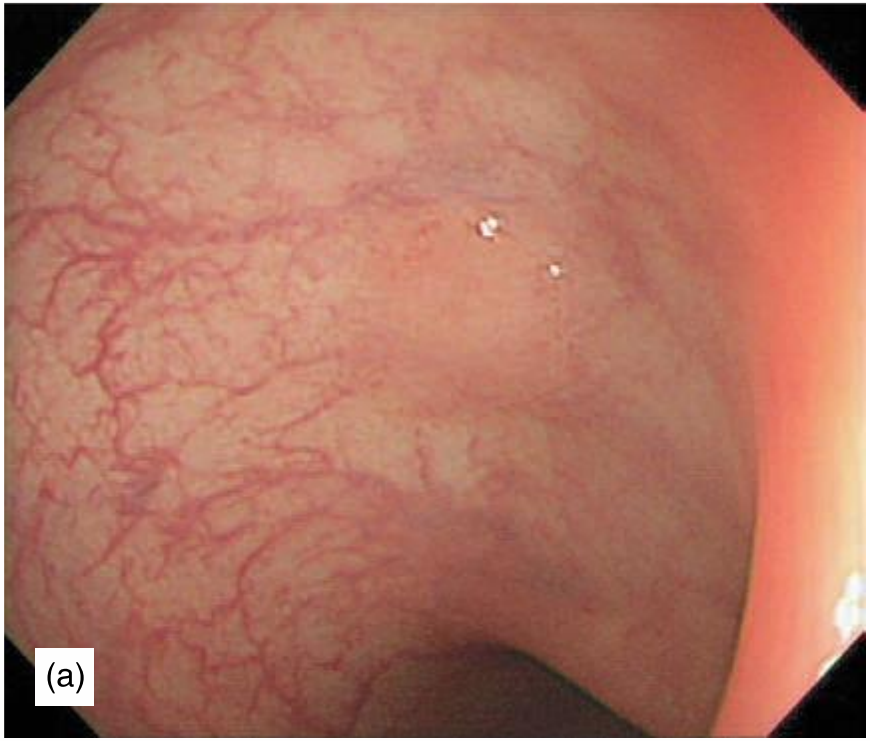
Colon
Tumor
Polyps

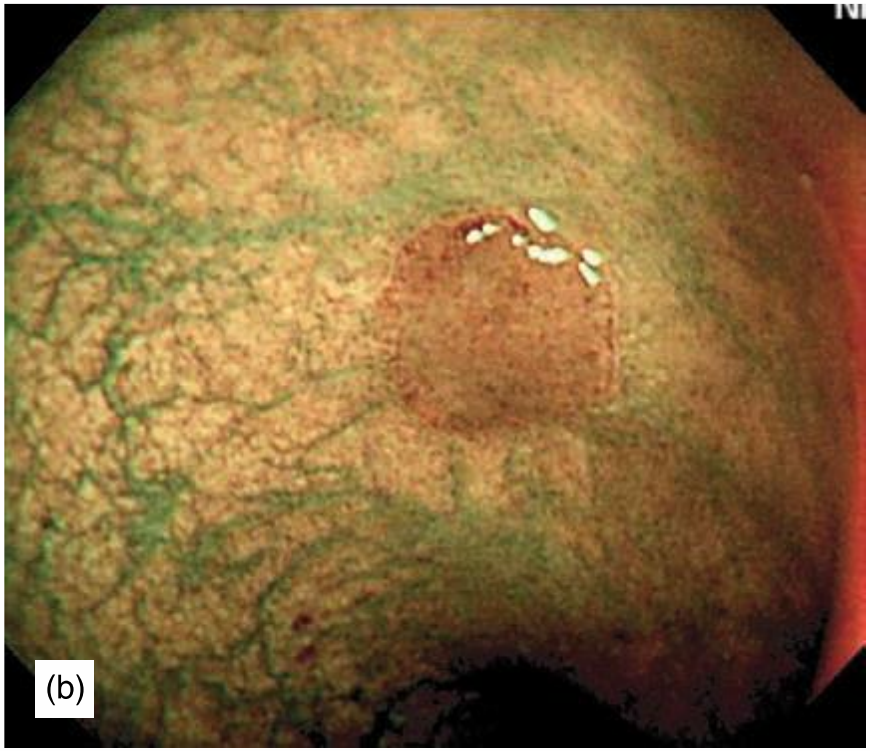
Narrow-band imaging



Colon Tumor Polyps

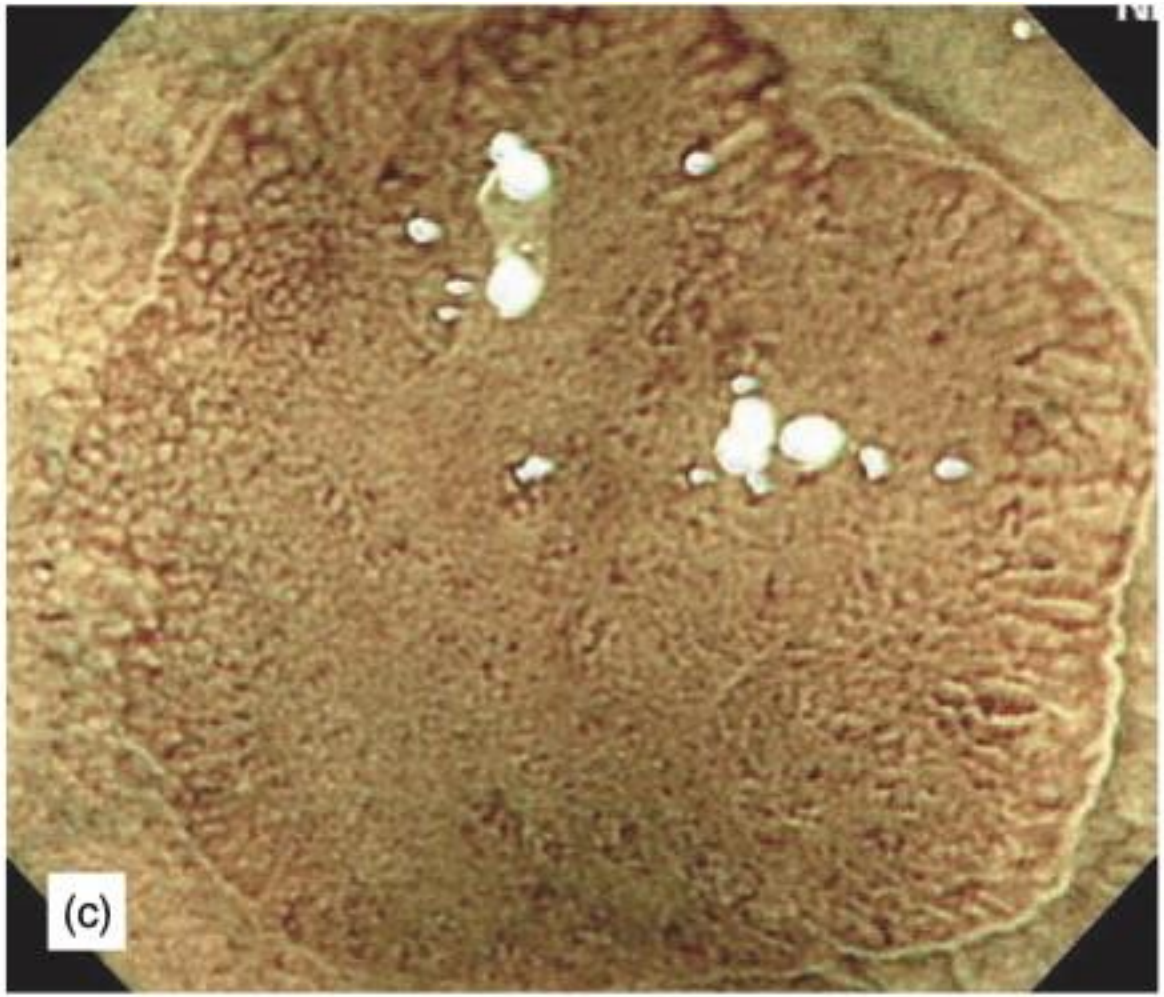
Tubular adenoma (brightfield és NBI)





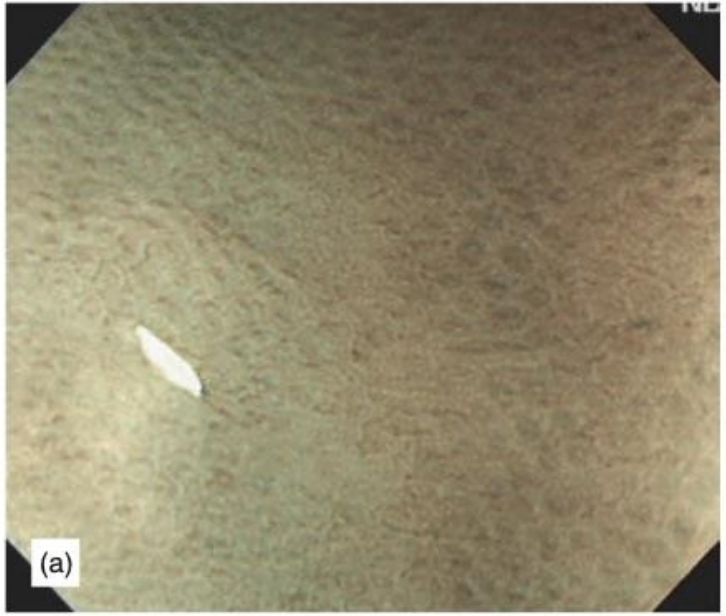
Colon
Tumor
Polyps

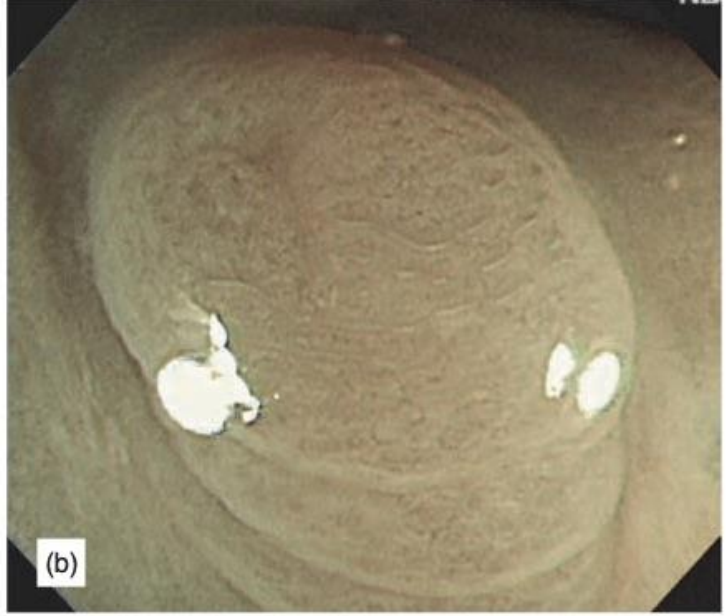
Tubular adenoma (NBI) with high magnification

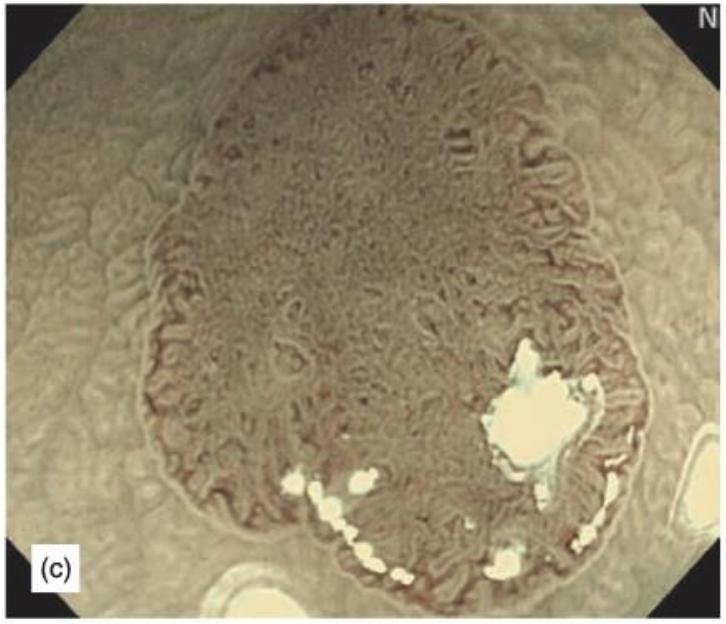


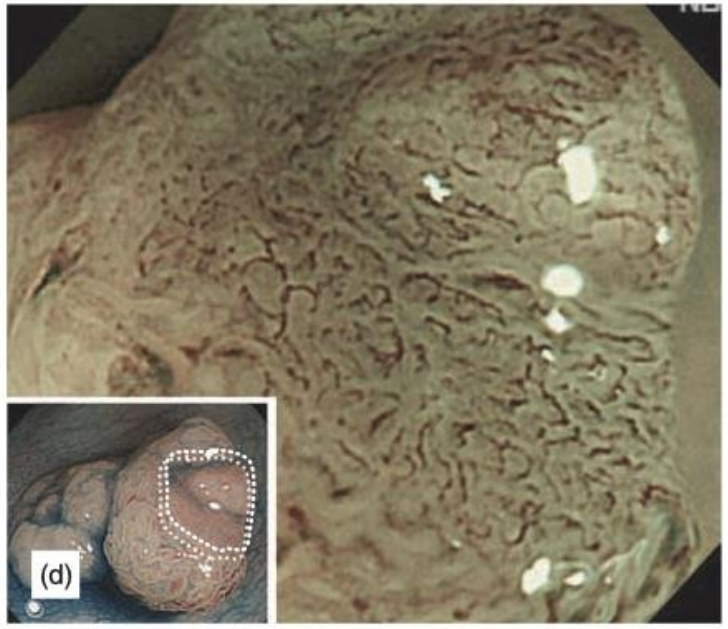
Colon Tumor Polyps

Normal mucosa
Hyperplastic
Adenoma
Carcinoma

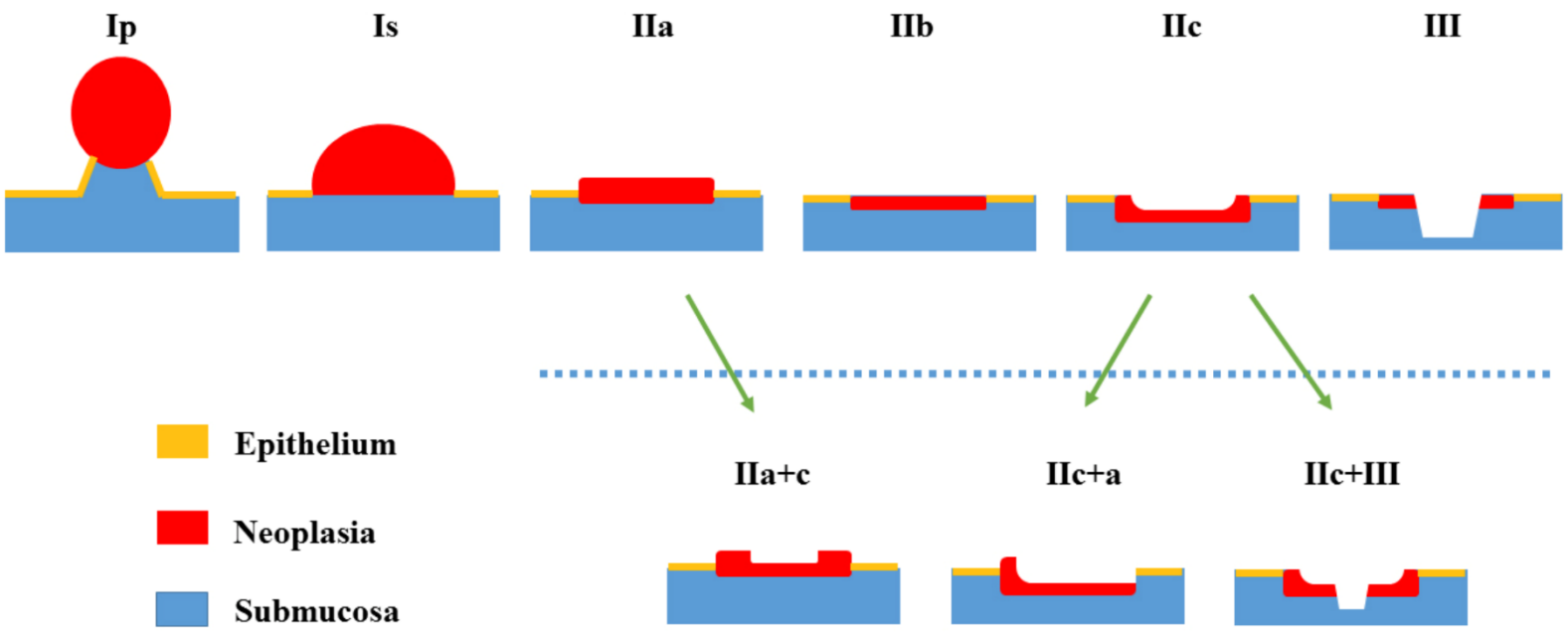






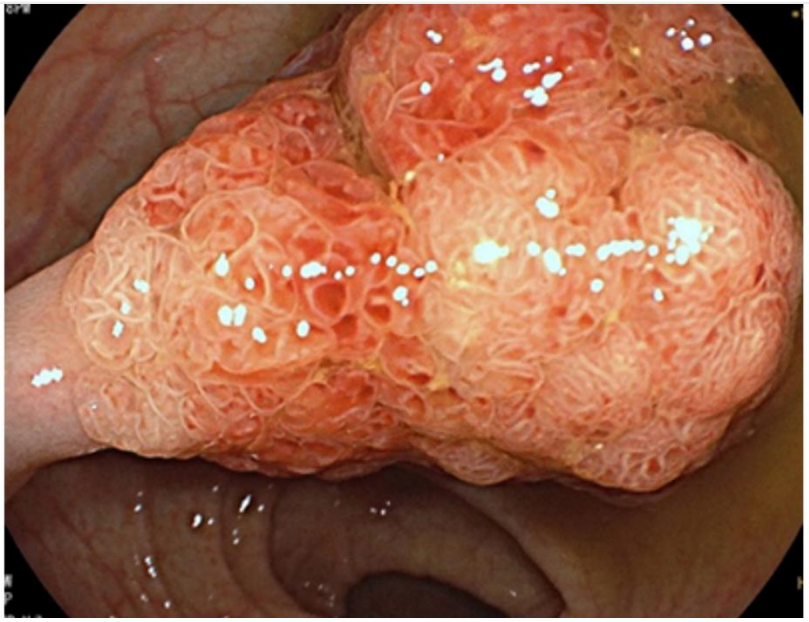


Paris-classification of polyps

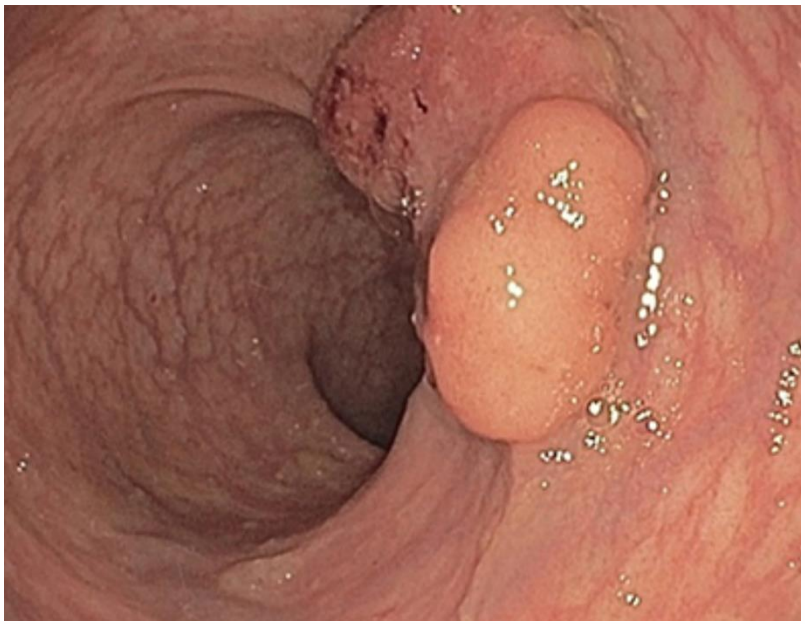


Colon Tumor Polyps

Mucosal neoplasia (high grade dysplasia)



Colon carcinoma (low risk) (pT1a)



Colon
Tumor
Carcinoma

General features

One of the most common carcinomas in developed countries

Arises in individuals who are exposed to life style risk factors and have acquired or inherited genetic changes

95% of cc-s are sporadic

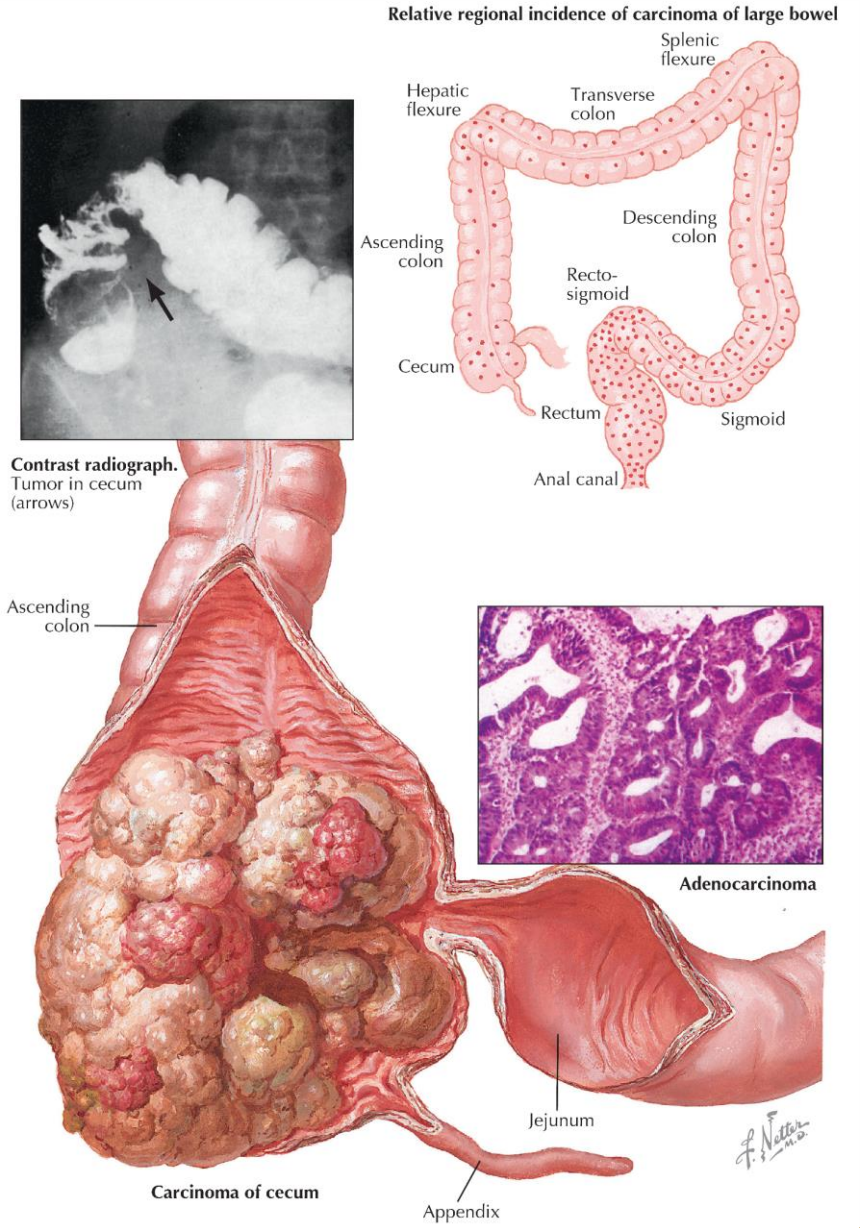
Peak incidence: 60 to 70 ys

The overall prognosis is bad

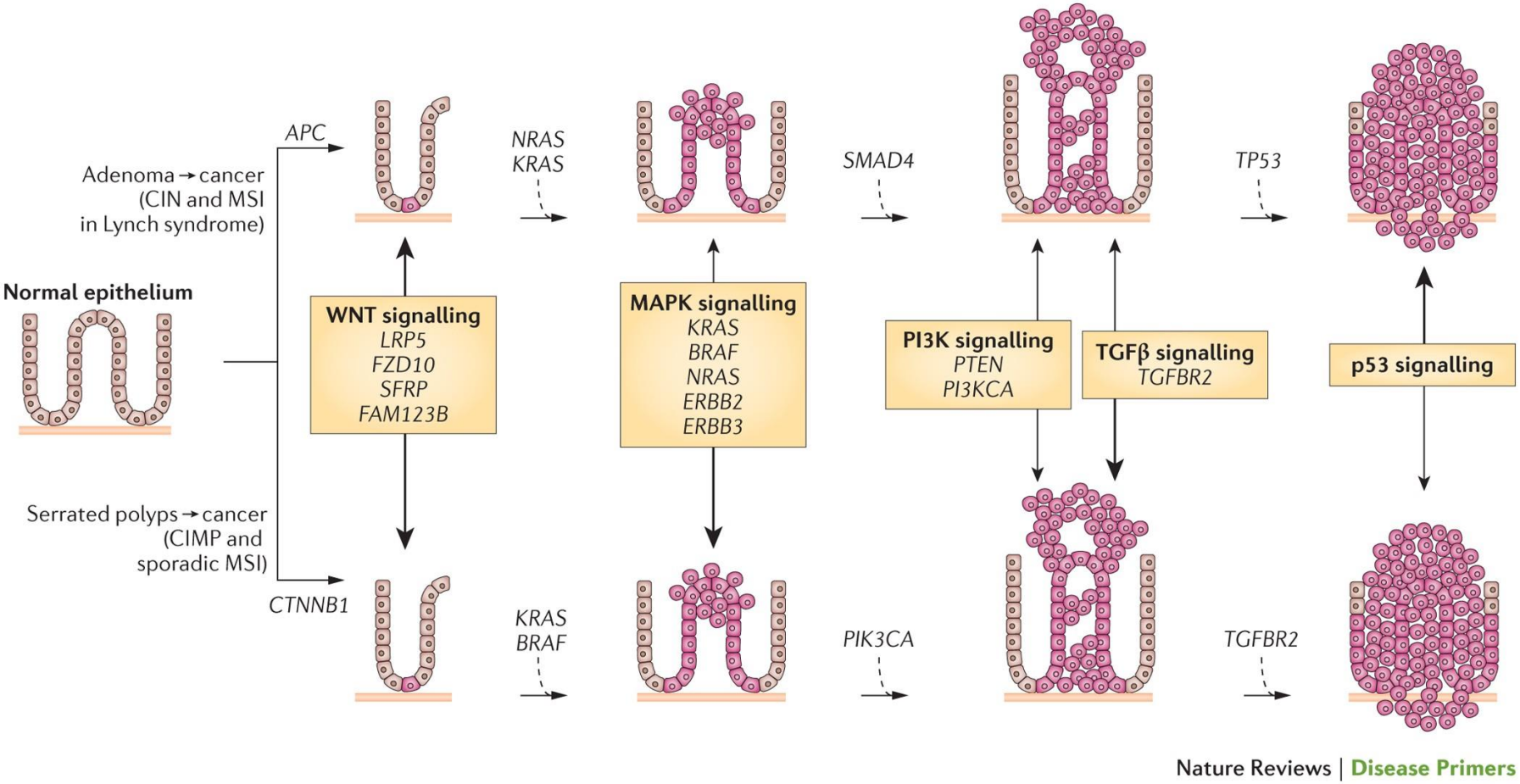
Pathogenesis

Risk factors: obesity, physical inactivity and Western diet (low intake of unabsorbable vegetable fiber, high intake of refined carbohydrates and red meat)

Two genetic pathways: 80%: chromosomal instability pathway; 20%: microsatellite instability pathway



Colon
Tumor
Carcinoma



Colon
Tumor
Carcinoma

Spread of colorectal carcinomas

The cancers eventually penetrate the wall, infiltrate the subserosa, and then the peritoneum

Metastases

- lymphatic: colonic cc-s mesocolonic lymph nodes; rectal cc-s mesorectal lymph nodes
- hematogeneous: liver; distal rectal carcinomas, lungs
- transcelomic: carcinosis of peritoneum

Clinical features

Asymptomatic for years

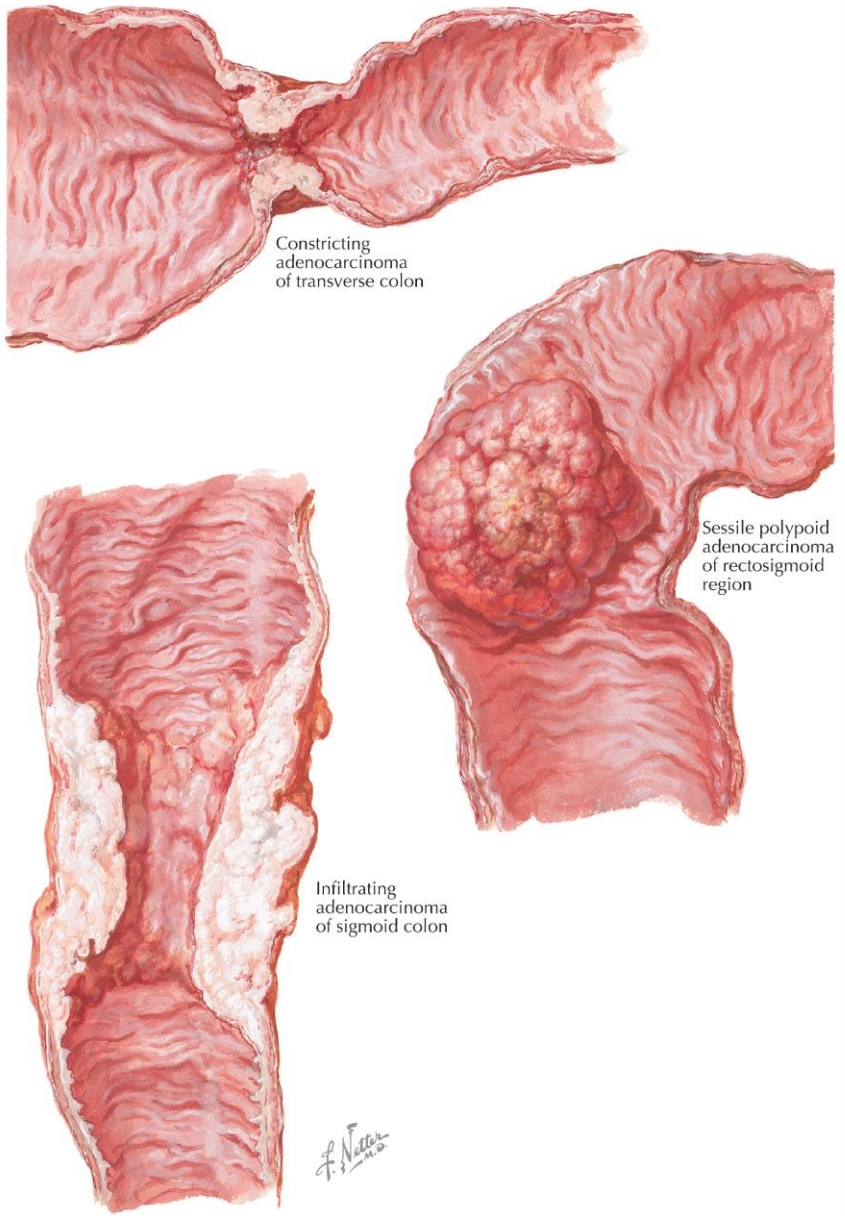
Distal colon carcinomas: can produce changes in bowel habit, occult bleeding, iron deficiency anemia, weakness; on occasion, intestinal obstruction or hematochezia

Proximal colon carcinomas: occult bleeding, iron deficiency anemia, weakness

Prognosis

Depends on the extent of tumor

At the time of the diagnosis, the majority of carcinomas infiltrated the subserosa and produced metastases in the regional lymph nodes



Colon
Tumor
Familial syndromes

1. Familial adenomatous polyposis

Autosomal dominant; biallelic loss of the tumor suppressor *APC* (Adenomatous Polyposis Coli) gene

In adolescence >100 colonic adenomatous polyps; by age 30 ys 500-2500 polyps, malignant transformation

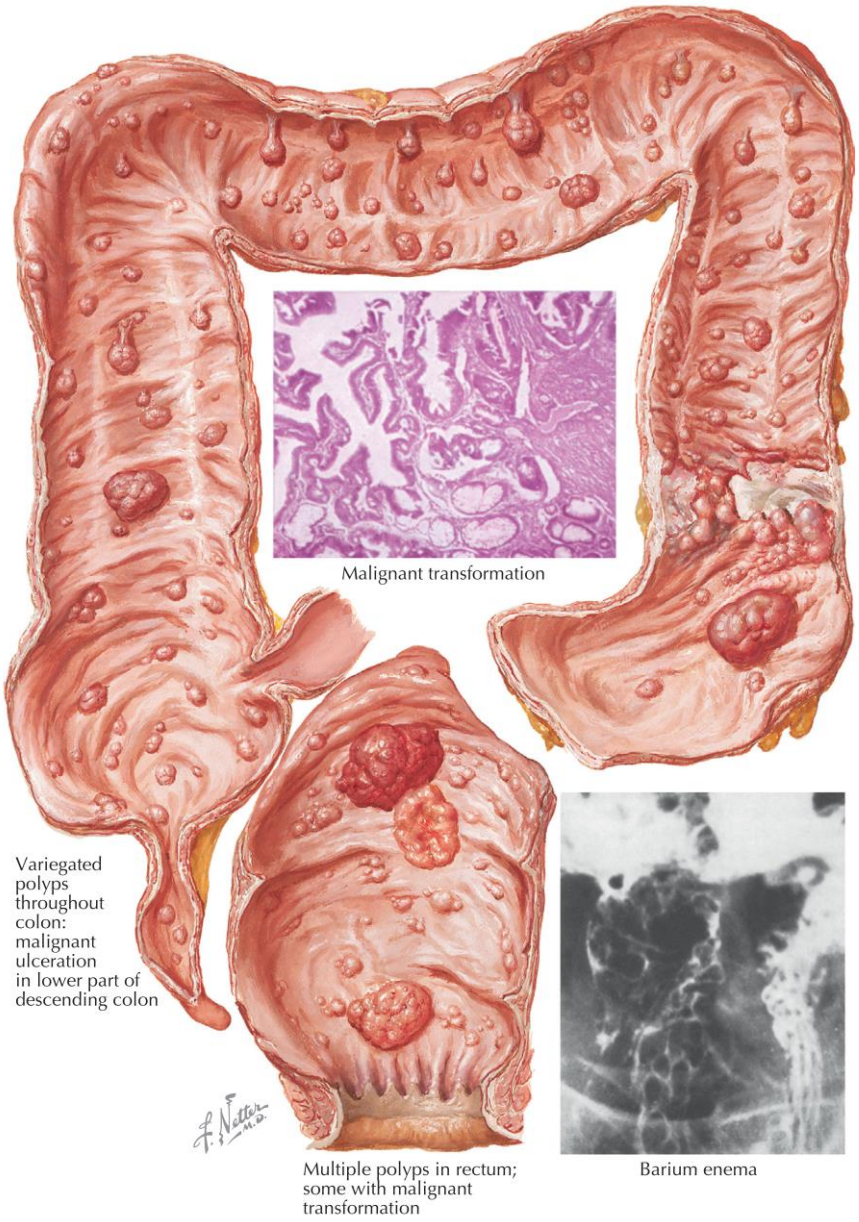
Therapy: total colectomy

2. Lynch sy (hereditary nonpolyposis colorectal carcinoma)

Autosomal dominant; biallelic mutation in the DNA mismatch repair genes, such as *MSH2 or MLH1*, microsatellite instability, adenomas in the colon

Carcinoma of right colon at age 45 ys; often multiple

Affected individuals have high risk of endometrial and ovarian carcinomas



Appendix
Inflammation
Appendixitis acuta

Acute bacterial infection of the appendix, usually precipitated by the obstruction of the lumen by fecalith, enlarged mucosal lymphoid follicles, worms, *etc*.

Frequent condition affecting mainly older children and young adults

Morphology of inflammation

Purulent

6th hour - acute early ~: focal erosions in the mucosa, filled and covered with fibrin and ng-s

24th hour - acute ulcerophlegmonous ~: multiple ulcers, intense transmural infiltration of ng-s, fibrinopurulent exudate on the serosa (*this is the stage when surgical removal is usually performed)*

48th hour - acute suppurative ~ : grossly visible abscesses in the wall of appendix + perforation

Gangrenous

Rapid course

Perforation is frequent

Complications

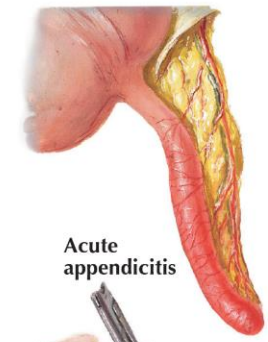
Focal or diffuse acute fibrinopurulent peritonitis

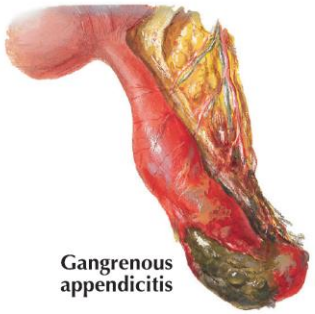
Periappendicular abscess

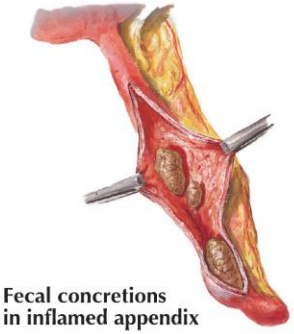
Rare: suppurative inflammation of the portal vein (pylephlebitis) with thrombosis, hepatic abscesses and septicemia

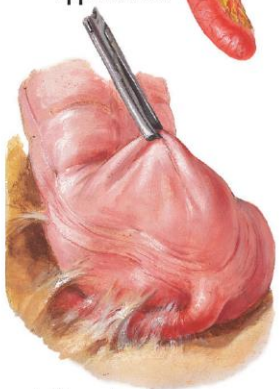
Clinical features

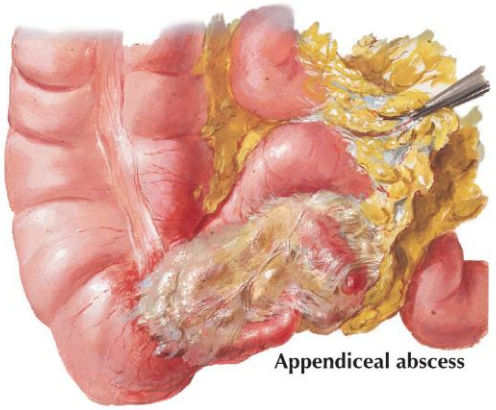
Presents itself as acute abdomen

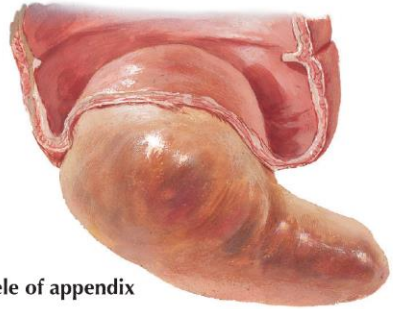

Acute appendicitis

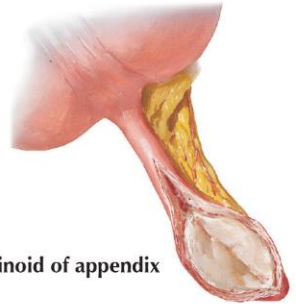

Gangrenous appendicitis

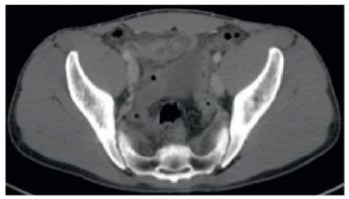

Fecal concretions in inflamed appendix


Inflamed retrocecal appendix with adhesions


Appendiceal abscess


Mucocele of appendix


Carcinoid of appendix


Diffusely dilated appendix in a patient with acute appendicitis

Appendix
Mucocele

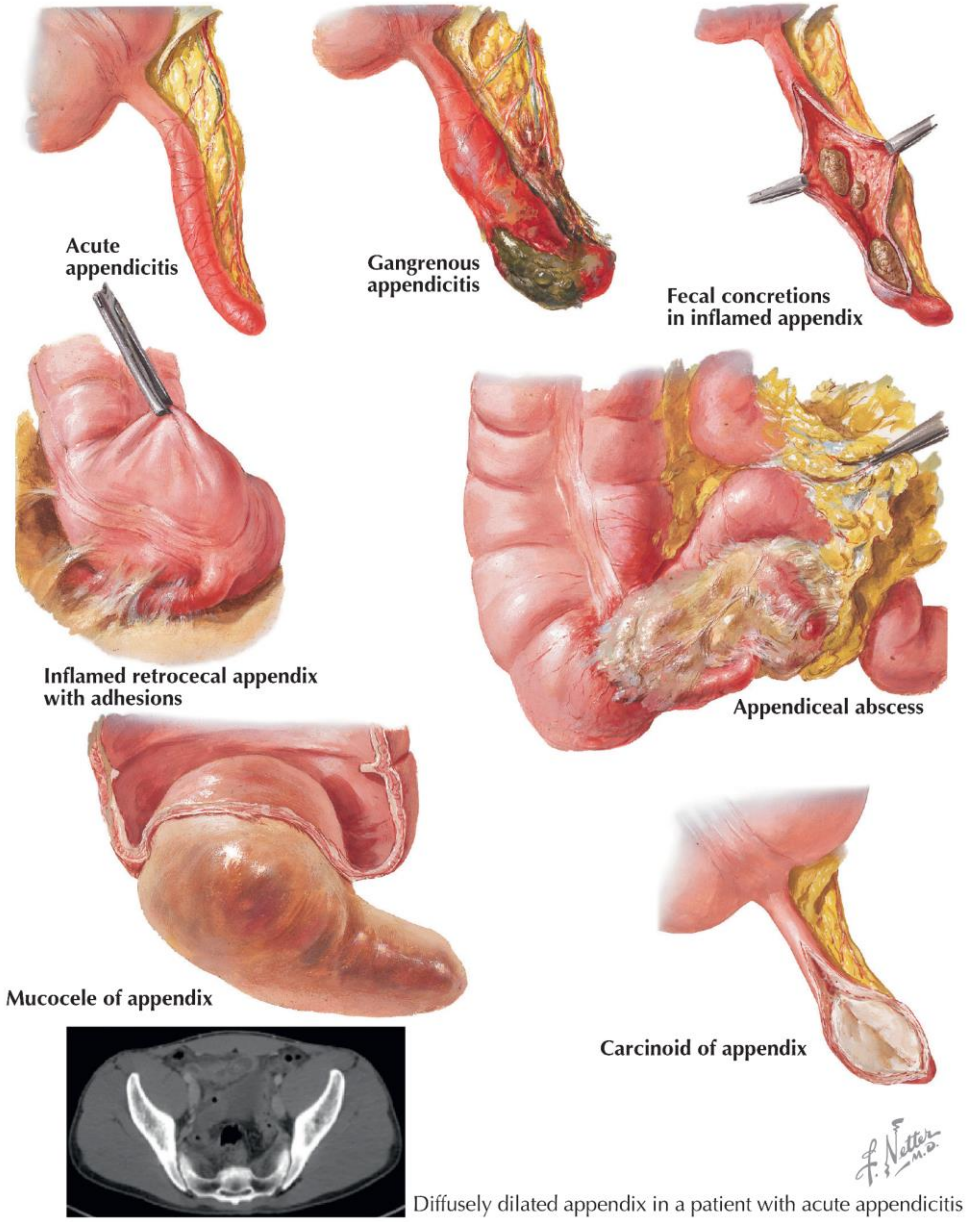
Mucocele of appendix

Macroscopic description of a dilated appendix filled with mucin

Obstructive ~: fecalith plugs the orifice of the appendix

Neoplastic ~: results from a mucinous cystadenoma or a mucinous cystadenocarcinoma of the appendix

Rupture of cystadenocarcinoma, implantation of carcinoma cells throughout the peritoneal cavity, which becomes filled with mucin: pseudomyxoma peritonei



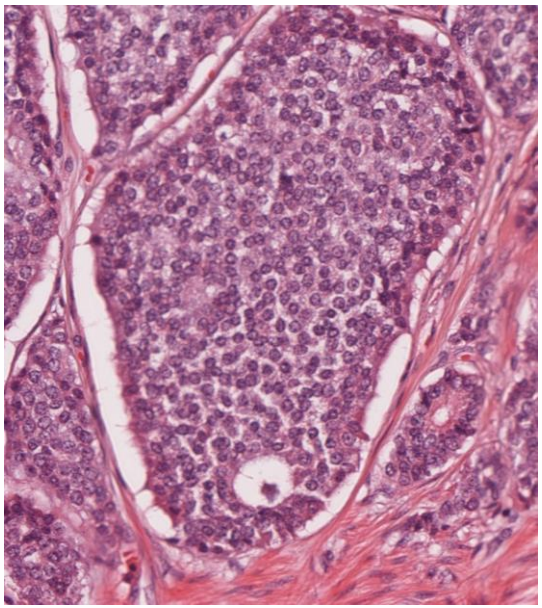
Appendix
Tumor
Neuroendokrin

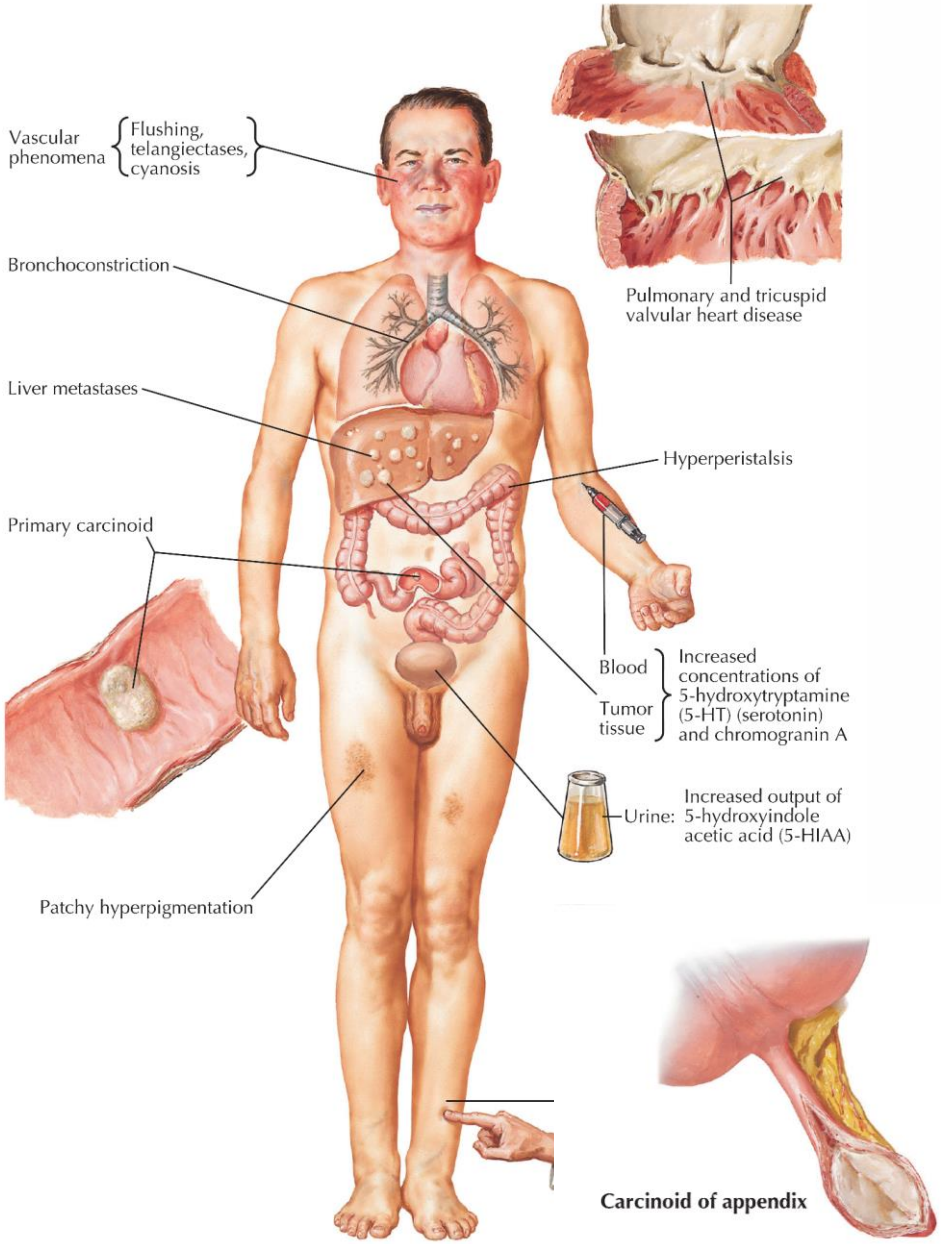
Neuroendocrine tumor of the appendix

Often discovered incidentally in appendices removed for appendicitis

Usually in the distal tip

Malignant - invades through the wall, and give metastasisis

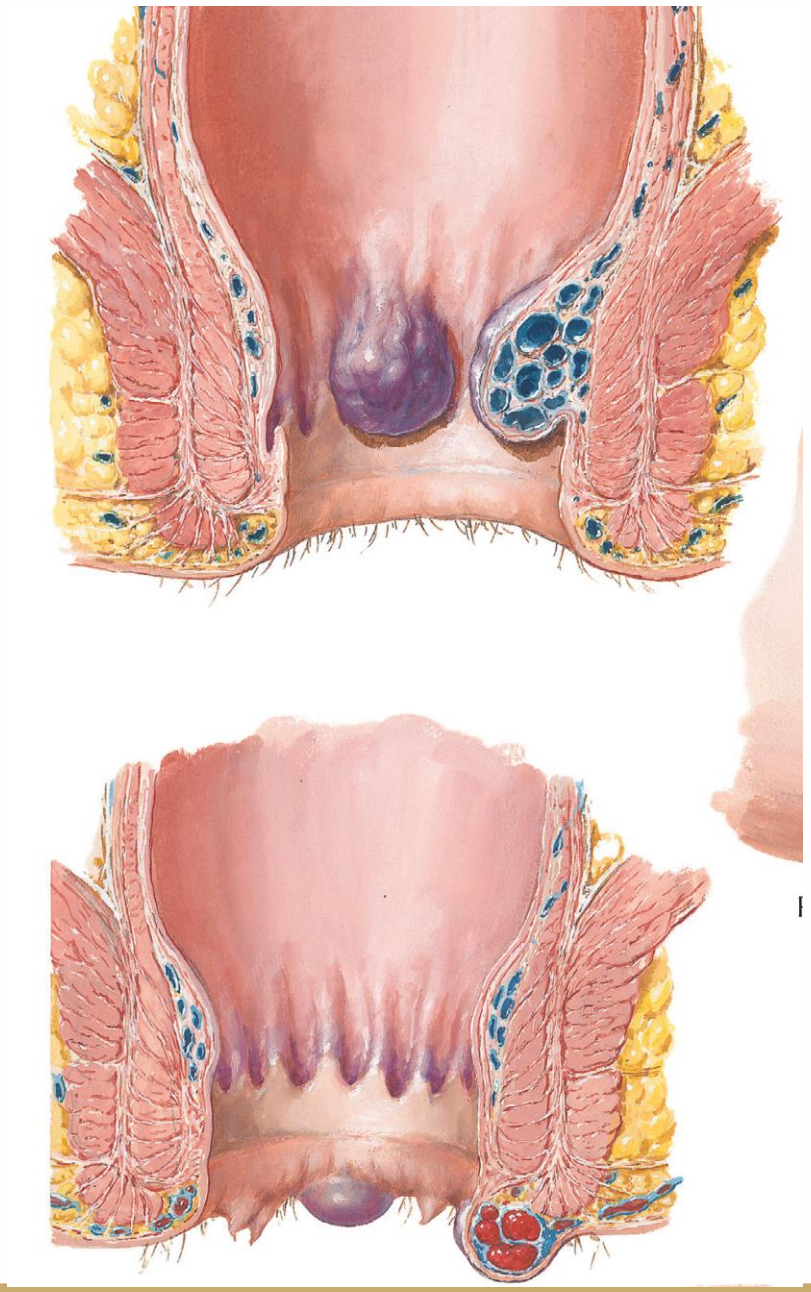




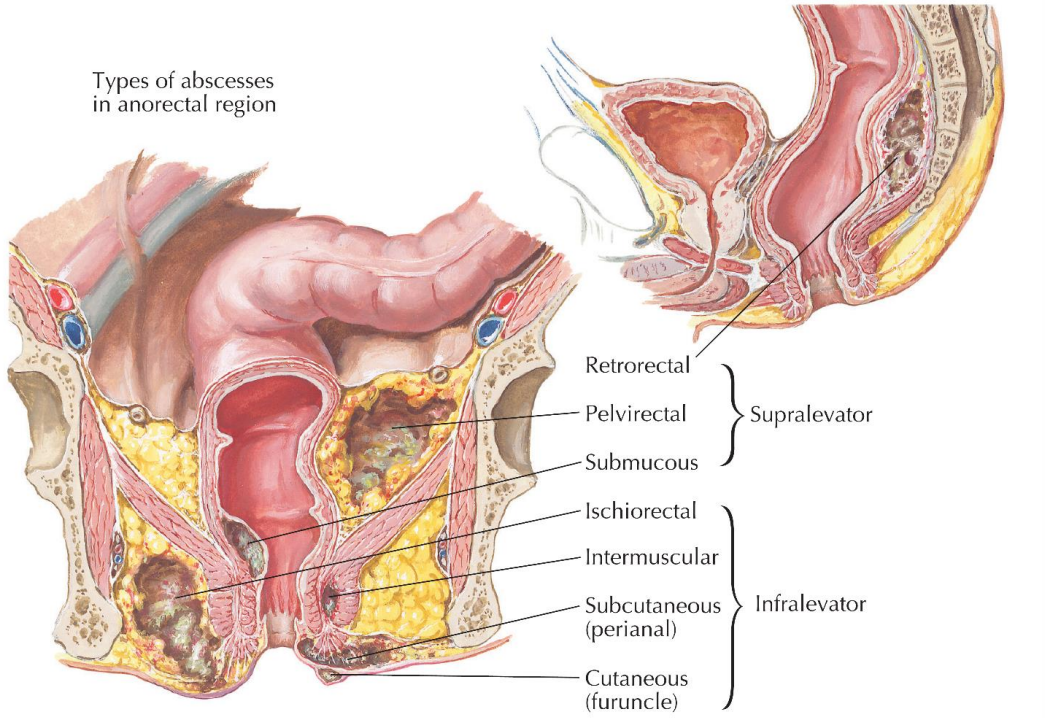
Anus
Haemorrhoids

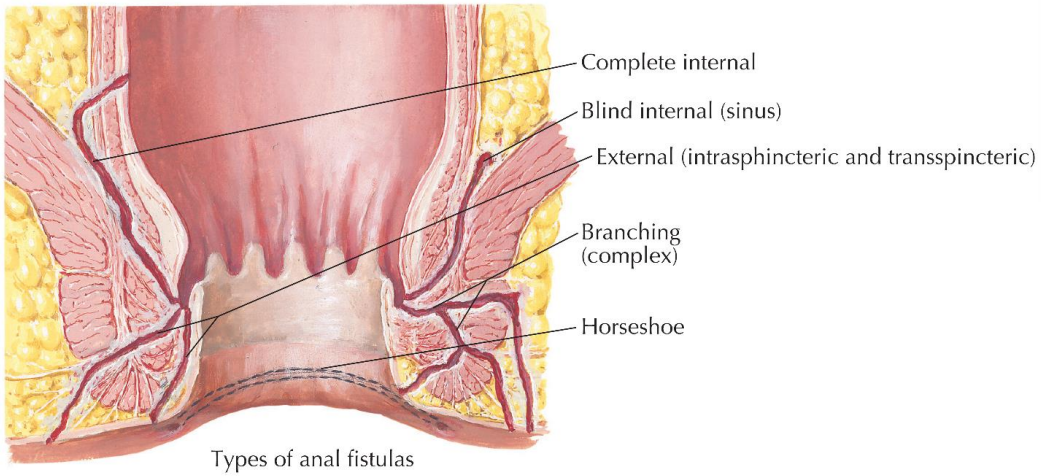
Dilated venes

Thrombosis
Inflammation
Pain



Anus
Inflammation
Fistula



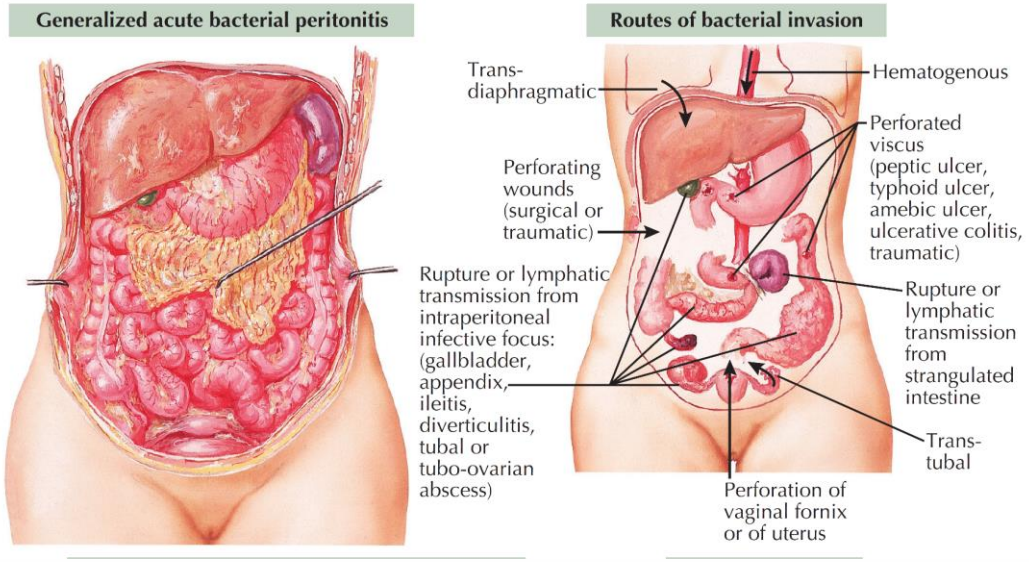


Types of anal fistulas

Peritoneum
Inflammation
Bacterial peritonitis

Pathogenesis

Three subsets



1. **Perforation** of an abdominal viscus, as in peptic ulcer, cholecystitis, appendicitis, colon carcinoma, mixed infection of E. coli, enterococci, Gram-negative rods, Streptococci, and Clostridia.

Leakage of bile, gastric juice or pancreatic enzymes, first sterile peritonitis, within hours bacteria appear in the exudate, turns into bacterial peritonitis

2. Bacterial **permigration** through the intact, but severely inflamed viscus wall, as in cholecystitis or salpingitis

3. **Spontaneous** bacterial peritonitis develops without an obvious source; occurs in the setting of ascites (e.g., cirrhosis, nephrotic sy).

Peritoneum Inflammation

Morphology

The serosal surface is hyperemic, its glistening sheen is lost, and a creamy (fibrino)purulent exudate covers the surface of the intestines

First it is localized, then becomes diffuse (generalized)

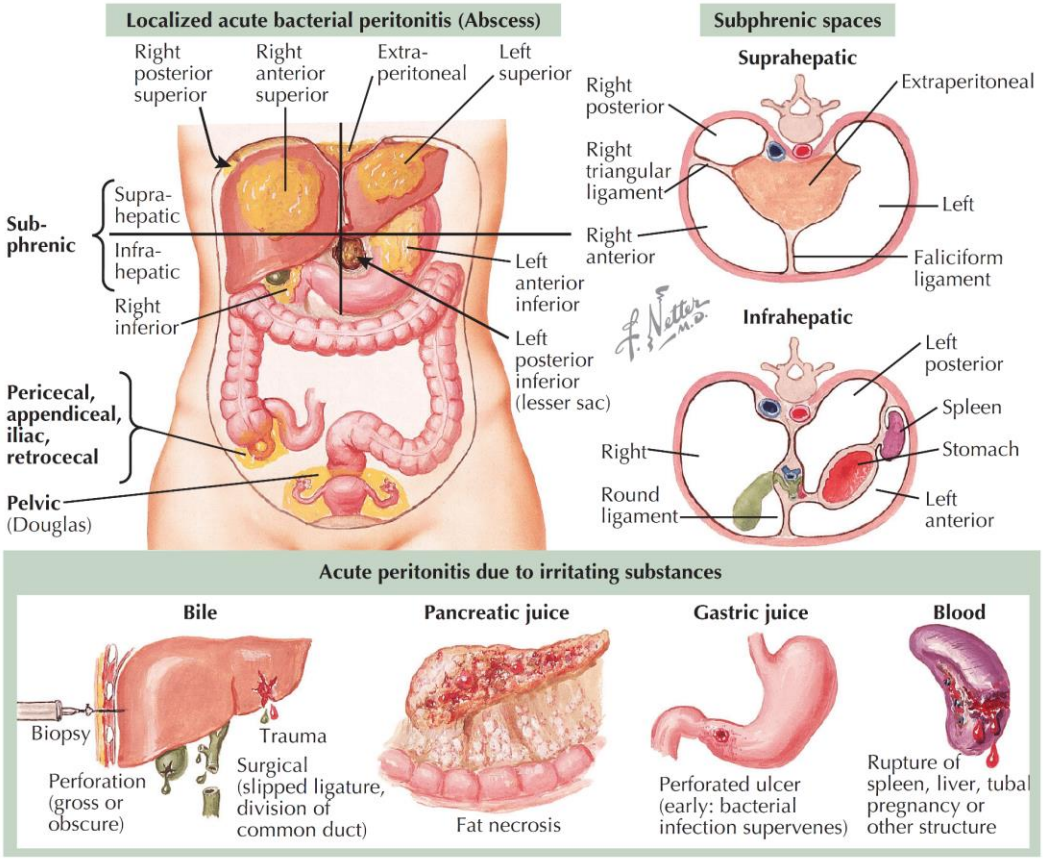
Inflammation of the peritoneal cavity in the minor pelvis: pelveoperitonitis

Events in survivors

The perforation is „walled off" by loops of intestine, omentum and abdominal parietes, localized abscess(es): intestinal or subdiaphragmatic or subhepatic or pelvic cul-de-sac abscess

Organization: granulation tissue, fibrous tissue (adhesion) between loops of bowel, which become joined to each other + intestinal obstruction

Walled-off abscesses serve as new infectious foci and, therefore, require surgical drainage



Clinical features

Sy of acute abdomen

In the stage of diffuse peritonitis, paralytic ileus and septic shock ensue

Despite proper surgical treatment, diffuse peritonitis has a **high mortality rate**; especially in the elderly

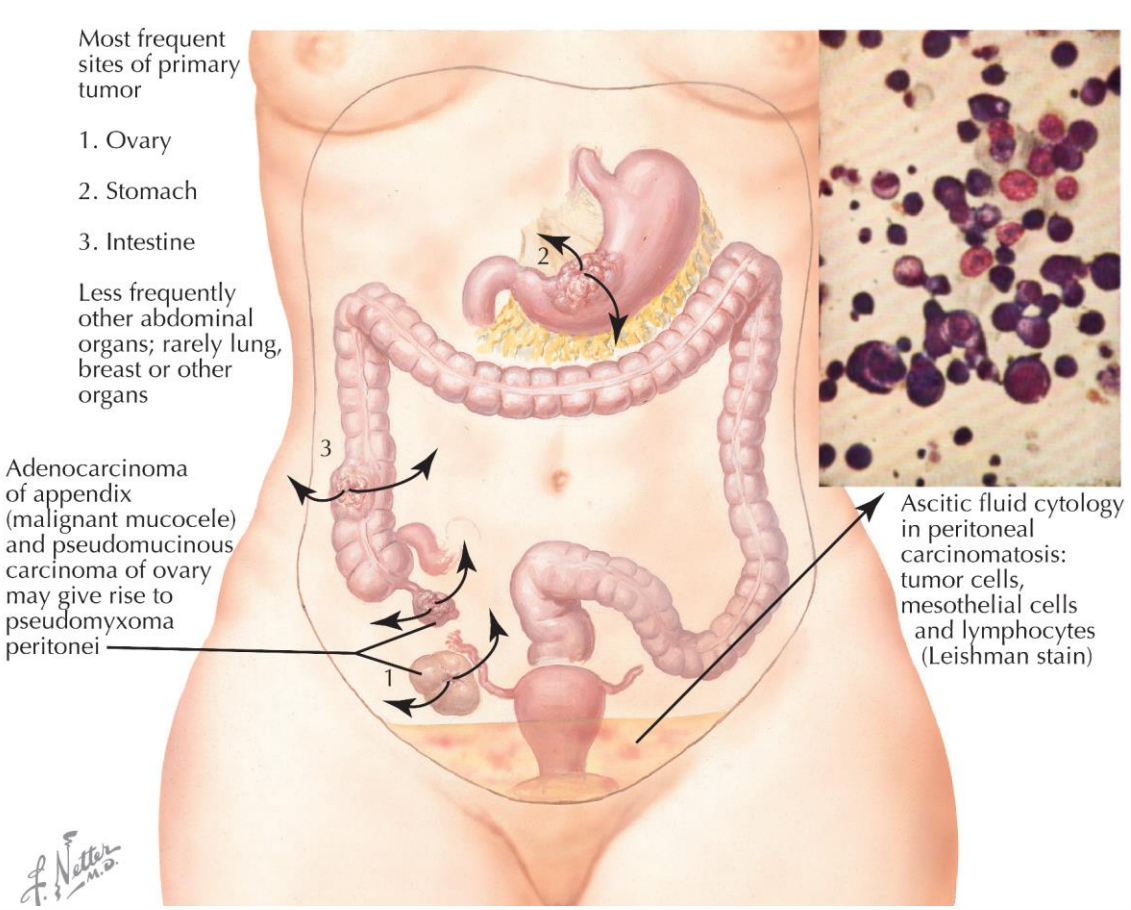
Peritoneum Tumor

Malignant

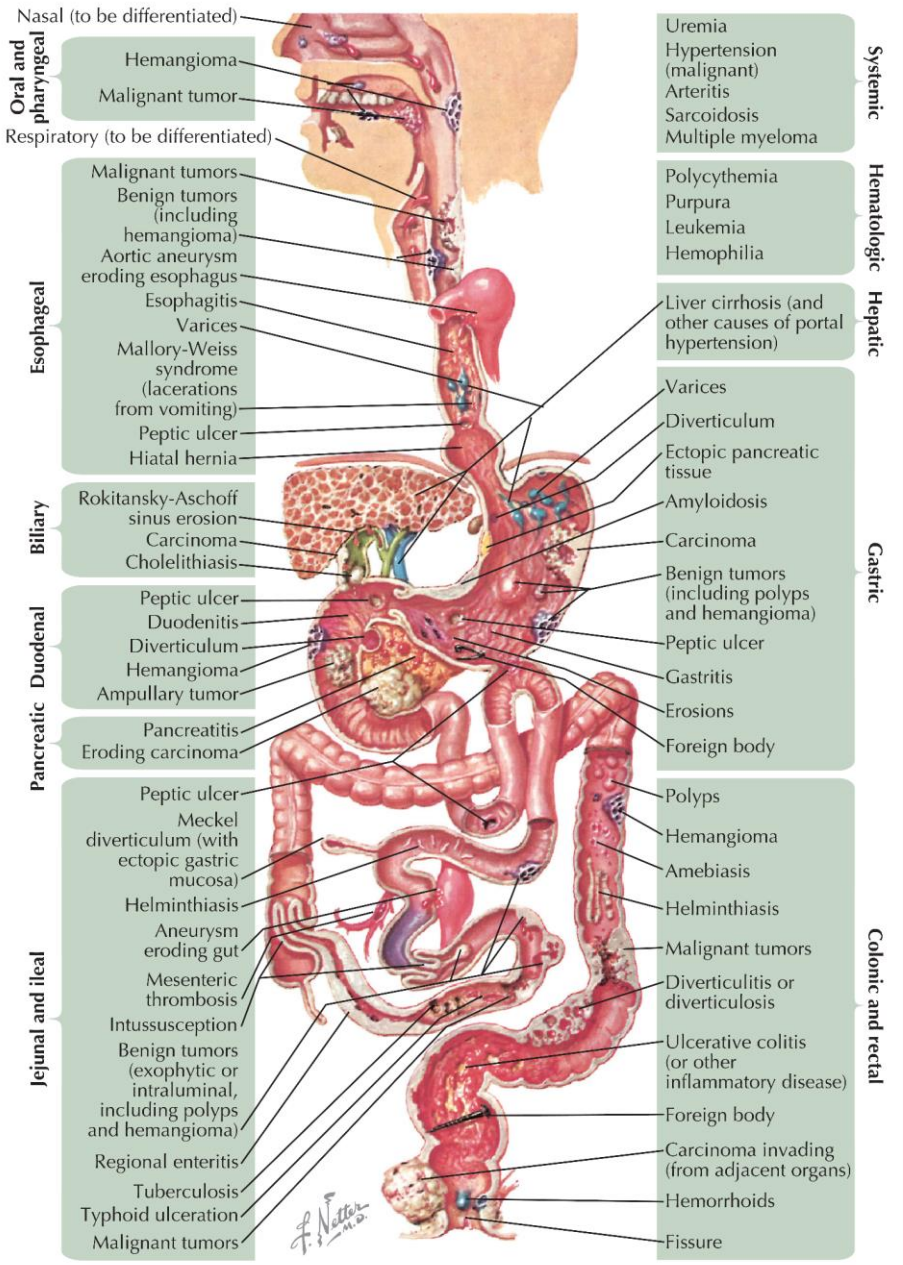
Metastatic involvement: very common; in any form of advanced cancer of abdominal organs, direct spread to the peritoneal surface, leading to carcinosis of peritoneum, death within months

Ovarian or pancreatic adenocarcinomas regularly cause carcinosis of peritoneum

Primary: rare; peritoneal mesothelioma due to asbestos exposure



GI bleeding


Nasal (to be differentiated)
Oral and pharyngeal
Hemangioma
Malignant tumor
Respiratory (to be differentiated)
Esophageal
Malignant tumors
Benign tumors (including hemangioma)
Aortic aneurysm eroding esophagus
Esophagitis
Varices
Mallory-Weiss syndrome (lacerations from vomiting)
Peptic ulcer
Hiatal hernia
Biliary
Rokitansky-Aschoff sinus erosion
Carcinoma
Cholelithiasis
Duodenal
Peptic ulcer
Duodenitis
Diverticulum
Hemangioma
Ampullary tumor
Pancreatic
Pancreatitis
Eroding carcinoma
Jejunal and ileal
Peptic ulcer
Meckel diverticulum (with ectopic gastric mucosa)
Helminthiasis
Aneurysm eroding gut
Mesenteric thrombosis
Intussusception
Benign tumors (exophytic or intraluminal, including polyps and hemangioma)
Regional enteritis
Tuberculosis
Typhoid ulceration
Malignant tumors
Systemic
Uremia
Hypertension (malignant)
Arteritis
Sarcoidosis
Multiple myeloma
Hematologic
Polycythemia
Purpura
Leukemia
Hemophilia
Hepatic
Liver cirrhosis (and other causes of portal hypertension)
Gastric
Varices
Diverticulum
Ectopic pancreatic tissue
Amyloidosis
Carcinoma
Benign tumors (including polyps and hemangioma)
Peptic ulcer
Gastritis
Erosions
Foreign body
Colonic and rectal
Polyps
Hemangioma
Amebiasis
Helminthiasis
Malignant tumors
Diverticulitis or diverticulosis
Ulcerative colitis (or other inflammatory disease)
Foreign body
Carcinoma invading (from adjacent organs)
Hemorrhoids
Fissure
F. Netter M.D.

Acute abdomen

Syndrome of acute abdomen

Severe abdominal pain, abdominal guarding, absence of bowel sounds, medical emergency

The pain can either be constant usually owing to inflammation (raised temperature, tachycardia + raised white cell count) or colicky because of an obstruction of the gut, biliary system, urinary tract or torsion of an ovarian cyst

Abdominal guarding: involuntary spasm of the abdominal wall; indicates peritonitis

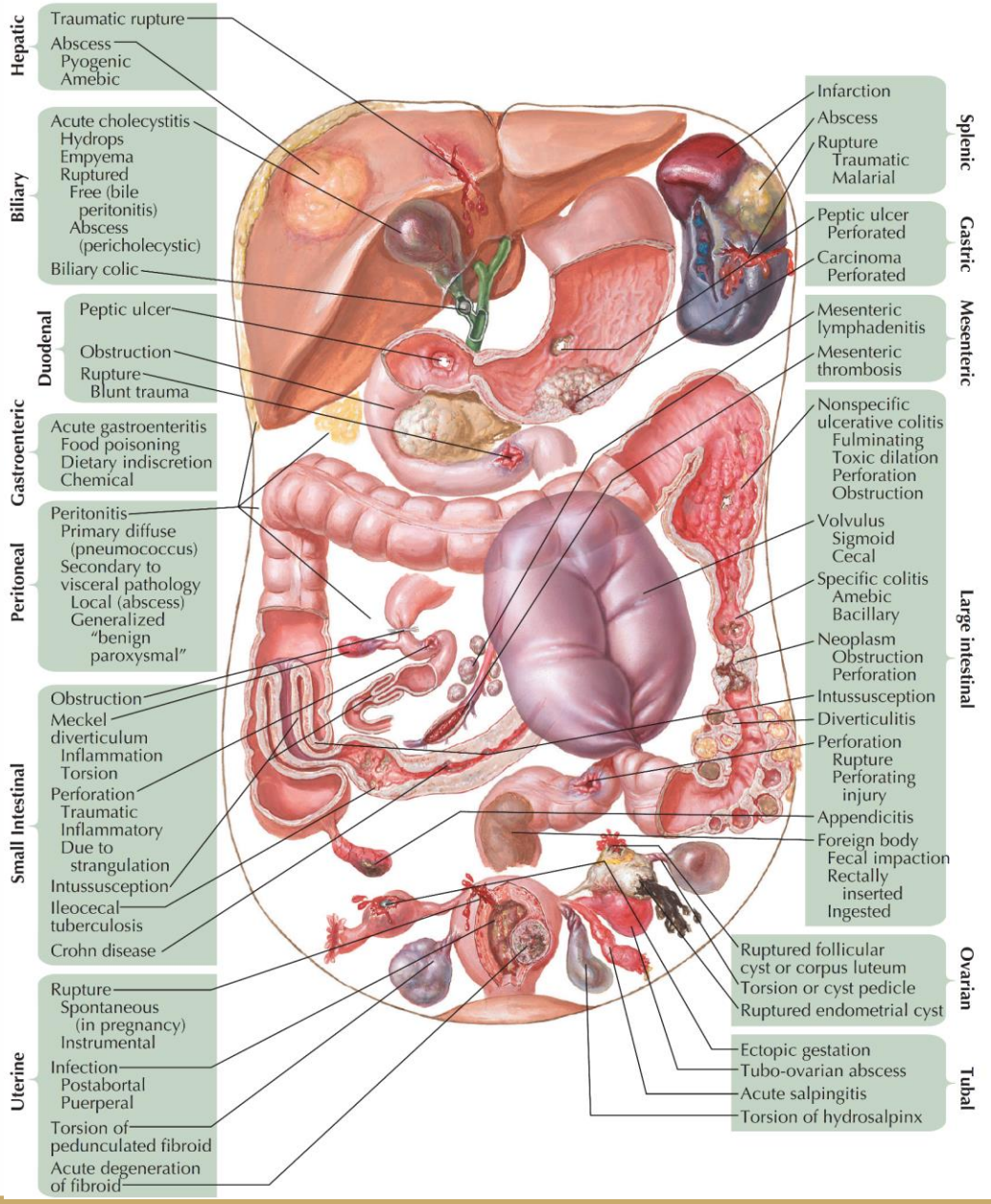
Common causes of acute abdomen

Perforation of an abdominal organ (X-ray: free air under the diaphragm)

Biliary colic

Thrombosis of superior mesenteric artery with infarction of the small bowels

Acute necrotizing-hemorrhagic pancreatitis



Acute abdomen 2

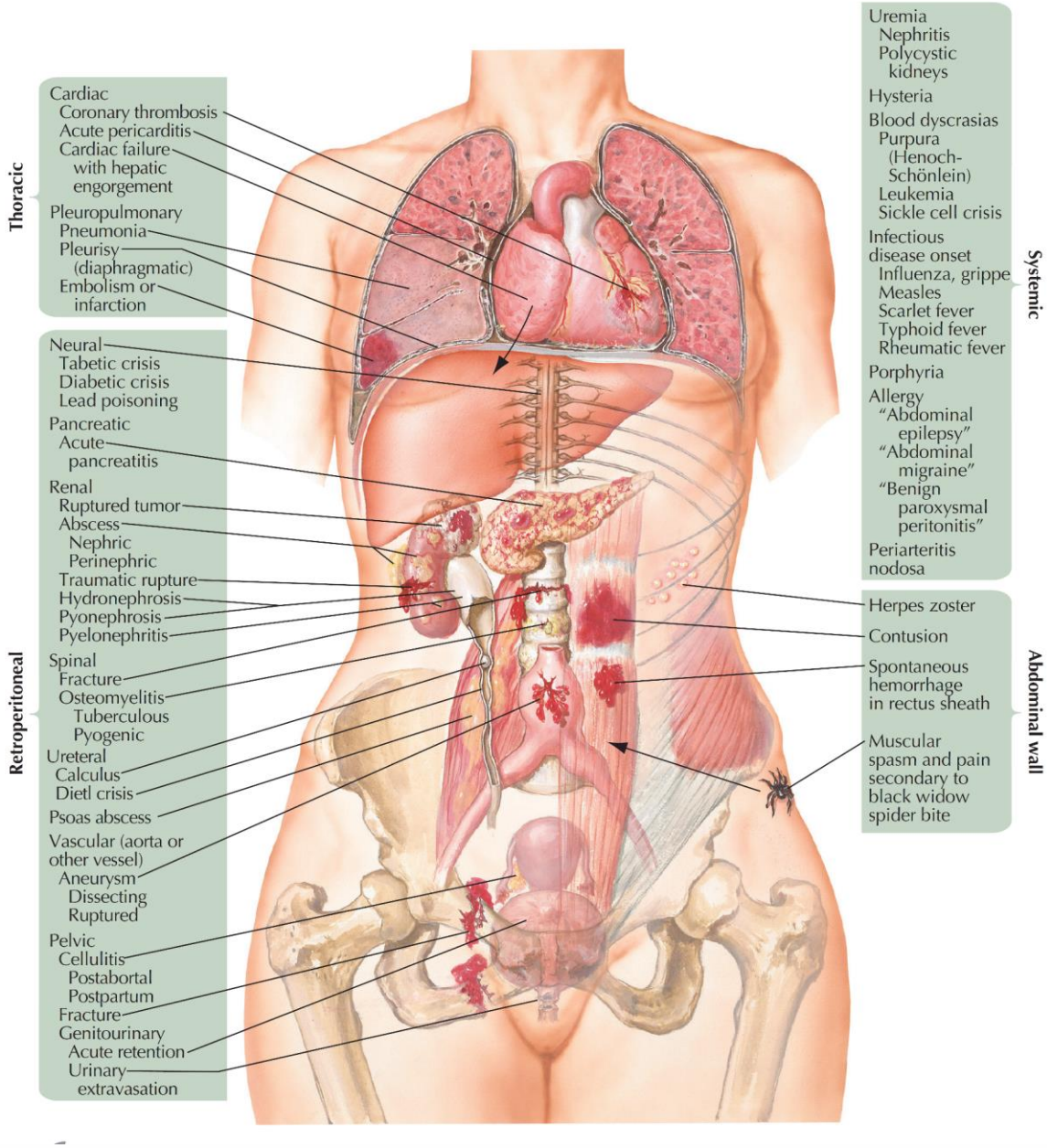
Immune complex-induced peritonitis

Called serositis

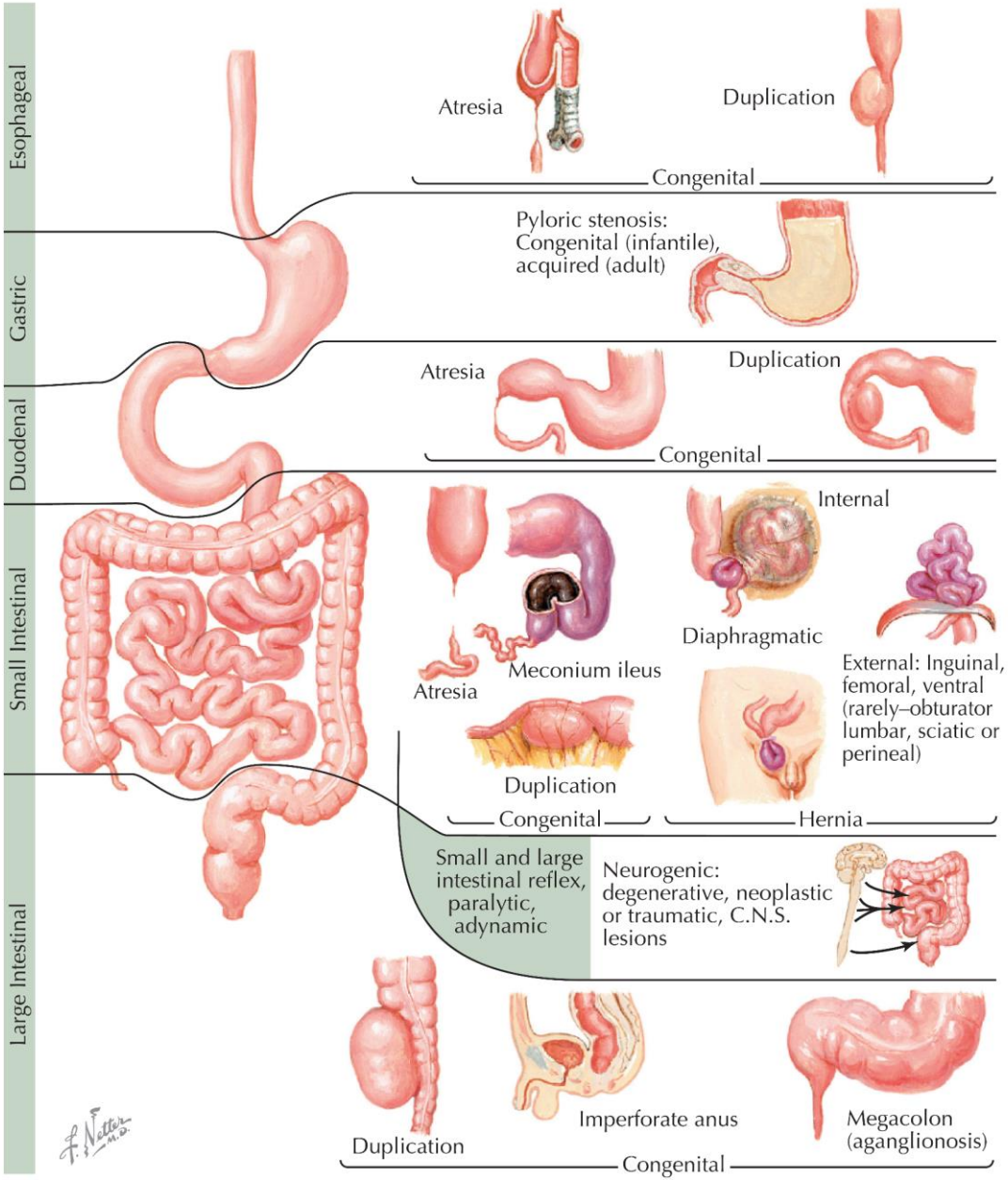
Frequent in SLE, together with pleuritis and pericarditis; bacterial invasion of the peritoneal cavity **does not** occur

Morphology: acute serous or serofibrinous peritonitis

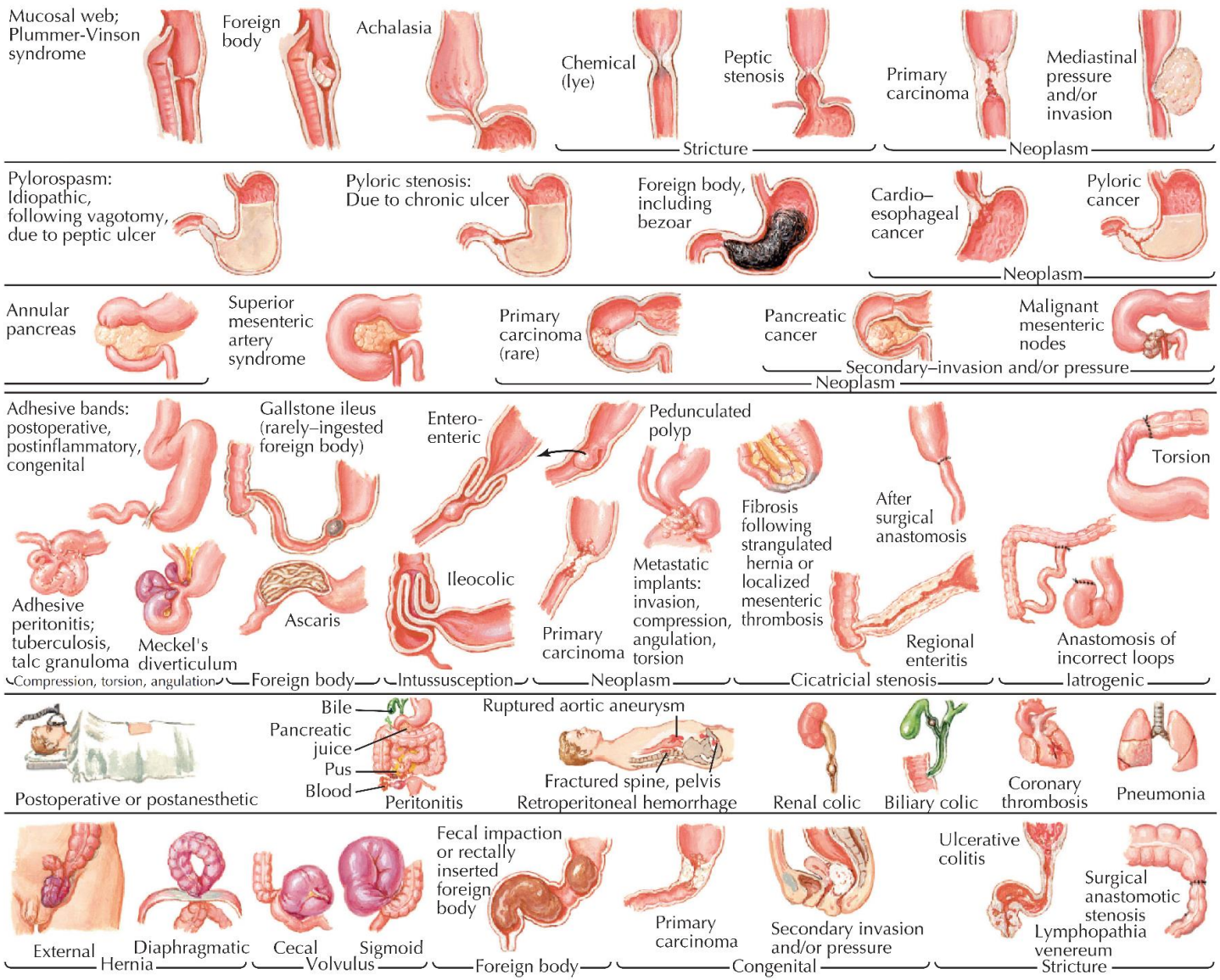
Heals with fibrous adhesions



Obstructions



Obstructions



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