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Bowel obstruction and tumors

I added extra information from Pathoma so you can understand more clearly .
it's written in green color so you can distinguish them from what the doctor said.

Intestinal Obstruction

- Obstruction of the GI tract may occur at any level, but the small intestine is most often involved because of its relatively narrow lumen.

these are the causes of intestinal obstruction

- Causes:

Hernias

intestinal adhesions

Intussusception

volvulus

Tumors

Infarction----- strictures

Crohn disease----- strictures

they cause bowel obstruction by forming strictures

the major causes of intestinal obstruction are :
Herniation
Adhesions
Volvulus
Intussusception

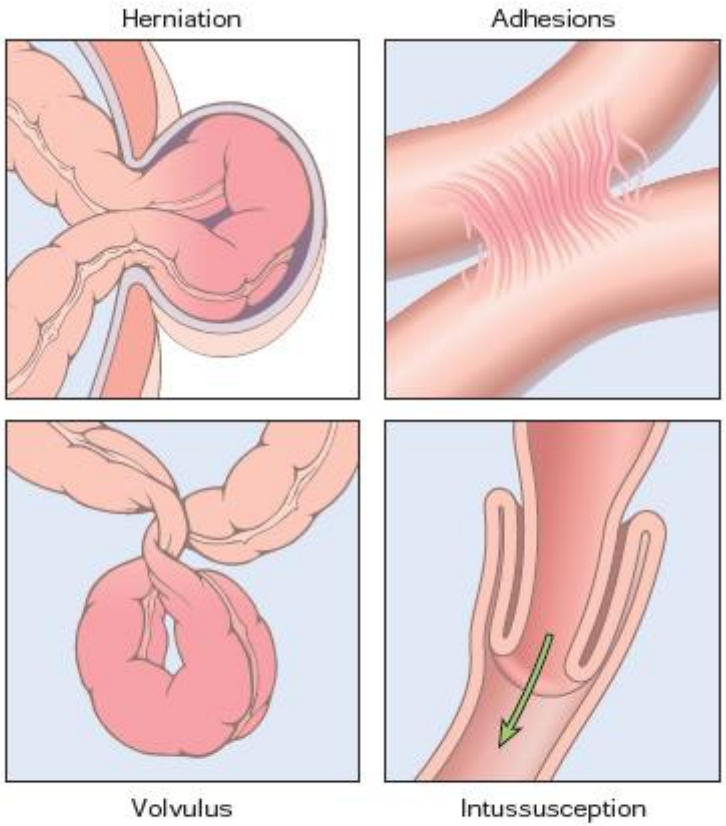

Figure 17-22 Intestinal obstruction. The four major causes of intestinal obstruction are (1) herniation of a segment in the umbilical or inguinal regions, (2) adhesion between loops of intestine, (3) volvulus, and (4) intussusception.

- The clinical manifestations of intestinal obstruction include:

abdominal pain and distention

Vomiting

constipation

The type of vomiting depends on the site of obstruction
if the obstruction was in high level of the small intestine
the vomiting will contain green material
and if the obstruction was in the large intestine ,the
vomiting may contain some feces .

Hernia must be repaired before developing into infarction

- **Hernias:**

- Hernias are the most frequent cause of intestinal obstruction worldwide.

- Any weakness or defect in the abdominal wall may permit protrusion of a serosa-lined pouch of peritoneum called a hernia sac.

- inguinal and femoral canals, umbilicus, or at sites of surgical scars.

if Herniation occur in SI it will lead to SI obstruction, and if it occurred in the LI , LI obstruction will develop

- Small bowel loops are typically involved, but omentum or large bowel may also protrude, and any of these may become entrapped.

- Incarceration----------strangulation---------infarction

How does the obstruction develop ?

Adhesions can be acquired or congenital , but mostly acquired

- **Adhesions:**

Acquired may be due to :

- causes: Surgical procedures, peritoneal inflammation (such as infection, endometriosis)

- resulting in internal herniation

- Fibrous adhesions are most often acquired, but can be congenital in rare cases. Therefore, internal herniation must be considered even in the absence of a history of peritonitis or surgery.

these adhesions usually develop internal herniation which causes the obstruction.

- **Volvulus:**

- Twisting of a loop of bowel about its mesenteric point of attachment is termed volvulus; it results in both luminal and vascular compromise.

it cause obstruction mechanically and due to infarction

- It occurs most often in large redundant loops of **sigmoid colon**, followed in frequency by the cecum, small bowel, stomach, or, rarely, transverse colon.

why sigmoid colon ?
because it's mesentry is very redundant, so it is more easier to twist

usually in young children, due to viral gastroenteritis that cause hyperplasia of peyer patches that will telescope into another segment

Intussusception is telescoping of proximal segment of bowel forward into distal segment

- **Intussusception:**

- Intussusception occurs when a segment of the intestine, constricted by a wave of peristalsis, telescopes into the immediately distal segment. Once trapped, the invaginated segment is propelled by peristalsis and pulls the mesentery along.

- Intussusception is the most common cause of intestinal obstruction in **children younger than 2 years of age.**

- Some cases are idiopathic, but many cases have been associated with viral infection and rotavirusvaccines, perhaps due to reactive hyperplasia of Peyer patches and other mucosa-associated lymphoid tissue which can act as the leading edge of the intussusception.

- Intussusception is rare in older children and adults, and is generally caused by an intraluminal mass or tumor that serves as the initiating point of traction.

if the cause is tumor a sergical elemination must be done

in the children, the most common cause is lymphoid hyperplasia due to rotavirus

in adults , the most common cause is tumor

Polyps

polyps are protrusions of mucosa above the mucosal level

it can occur in any part of the GI tract

- Polyps are most common in the **colo-rectal region** but may occur in the esophagus, stomach, or small intestine.
- intestinal polyps can be classified as non-neoplastic or neoplastic in nature.
- The most common neoplastic polyp is the adenoma, which has the potential to progress to cancer.
- The non-neoplastic polyps can be further classified as inflammatory, hamartomatous, or hyperplastic. sometimes they can develop into benign tumors

These are morphological descriptions

- Pedunculated polyp: with stalks
- sessile polyp: without stalks

the first type of non-neoplastic polyps

- **hyperplastic Polyps**: Hyperplastic polyps are due to hyperplasia of glands , classically show a serrated appearance on microscopy.

- The pathogenesis of hyperplastic polyps is incompletely understood, but they are thought to result from decreased epithelial cell turnover and delayed shedding of surface epithelial cells. comes usually with chronic inflammation
- It is now appreciated that these lesions are **without malignant potential**.
- most commonly found in the left colon

notice the increased number of epithelial cells

serrated architecture

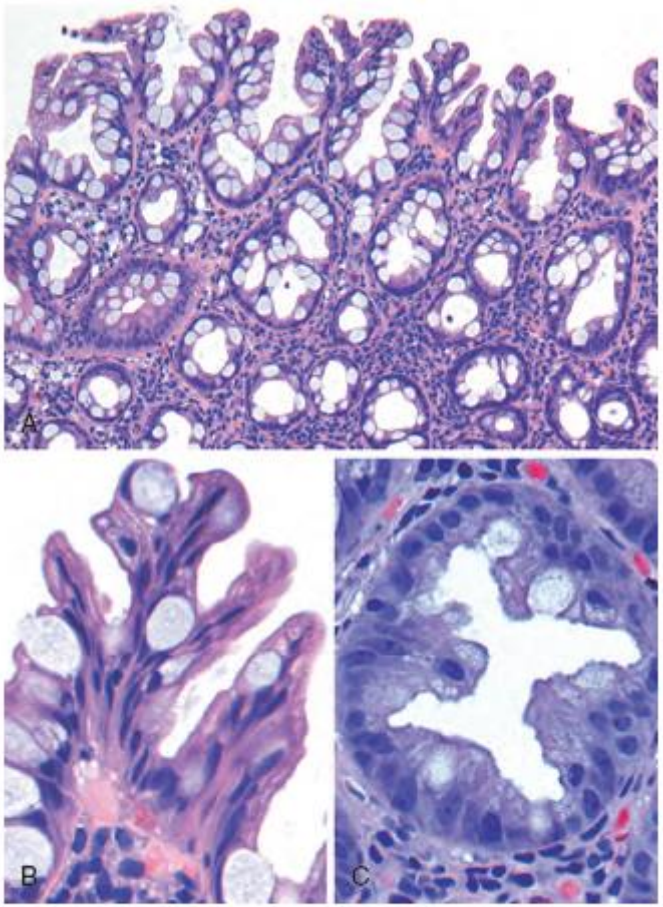

Figure 17-41 Hyperplastic polyp. **A,** Polyp surface with irregular tufting of epithelial cells. **B,** Tufting results from epithelial overcrowding. **C,** Epithelial crowding produces a serrated architecture when crypts are cut in cross-section.

normal tissue in normal location but with abnormal configuration

- **Hamartomatous Polyps**:

- Hamartomatous polyps occur sporadically or as components of various genetically determined or acquired syndromes.

- **Juvenile Polyps**:

may be sporadic (solitary lesions) or syndromic (3 to as many as 100 polyps , AD disorder, 30% to 50% of patients with juvenile polyposis develop colonic adenocarcinoma by age 45).

occur in children younger than 5 years of age, Most juvenile polyps are located in the rectum, typically present with rectal bleeding.

Peutz-Jeghers Syndrome: there must be 2 symptoms

This rare autosomal dominant syndrome presents at a median age of 11 years with multiple GI hamartomatous polyps and mucocutaneous hyperpigmentation.

The polyps of Peutz-Jeghers syndrome are most common in the small intestine.

associated with a markedly increased risk of several malignancies. in other sites of the body , not in GIT

All of these are types of hamartomatous polyps

Syndrome	Mean Age at Presentation (yr)	Mutated Gene(s); Pathway	Gastrointestinal Lesions	Selected Extra-Gastrointestinal Manifestations
Juvenile polyposis	<5	*SMAD4*,*BMPR1A*; TGF-β signaling pathway	Juvenile polyps; risk of gastric, small intestinal, colonic, and pancreatic adenocarcinoma	Congenital malformations, digital clubbing
Peutz-Jeghers syndrome	10-15	*STK11*; AMP kinase-related pathways	Arborizing polyps; Small intestine > colon > stomach; colonic adenocarcinoma	Pigmented macules; risk of colon, breast, lung, pancreatic, and thyroid cancer
Cowden syndrome, Bannayan-Ruvalcaba-Riley syndrome*	<15	*PTEN*: PI3K/AKT pathway	Hamartomatous/ inflammatory intestinal polyps, lipomas, ganglioneuromas	Benign skin tumors, benign and malignant thyroid and breast lesions; no increase in GI cancers
Cronkhite-Canada syndrome	>50	Nonhereditary, unknown cause	Hamartomatous polyps of stomach, small intestine colon; abnormalities in nonpolypoid mucosa	Nail atrophy, hair loss, abnormal skin pigmentation, cachexia, and anemia. Fatal in up to 50%.

The second type of polyps which are the neoplastic ones :

- **Adenomas:** to call apolyp adenoma there must be dysplasia

colonic adenomas are precursors to the majority of colorectal adenocarcinomas. But majority of adenomas do not progress to become adenocarcinomas.

characterized by the presence of epithelial **dysplasia.**

Adenomas can be classified as **tubular, tubulovillous,** or **villous** based on their architecture. These categories, however, have little clinical significance in isolation. **Size is the most important characteristic that correlates with risk of malignancy**.

Most adenomas are clinically silent, with the exception of large polyps that produce occult bleeding and anemia and rare villous adenomas that cause hypoproteinemic hypokalemia by secreting large amounts of protein and potassium.

they come as 2 syndromes :

- **Familial adenomatous polyposis** (FAP): inherited autosomal dominant disorder

is an autosomal dominant disorder in which patients develop numerous colorectal adenomas as teenagers.

At least 100 polyps are necessary for a diagnosis

of classic FAP, but as many as several thousand may be present.

caused by mutations of the adenomatous polyposis coli (**APC) gene.** 75% of cases are inherited, while the remaining appear to be caused by de novo mutations.

Colorectal adenocarcinoma develops in 100% of untreated FAP patients, often before age 30 and nearly always by age 50. As a result, prophylactic colectomy is the standard therapy for individuals carrying APC mutations.

if the patient was under the age of 30 we should do prophylactic colectomy or it will develop to adenocarcinoma

- **Hereditary non-polyposis colorectal cancer (HNPCC):** caused by mutation in DNA mismatch repair genes

Lynch syndrome

HNPCC is caused by inherited mutations in genes that encode proteins responsible for the detection, excision, and repair of errors that occur during DNA replication.

Etiology	Molecular Defect	Target Gene(s)	Transmission	Predominant Site(s)	Histology
Familial adenomatous polyposis	APC/WNT pathway	*APC*	Autosomal dominant	None	Tubular, villous; typical adenocarcinoma
Hereditary nonpolyposis colorectal cancer	DNA mismatch repair	*MSH2, MLH1*	Autosomal dominant	Right side	Sessile serrated adenoma; mucinous adenocarcinoma

these 2 syndromes cause adenoma but differ in morphology

Adenocarcinoma

- Adenocarcinoma of the colon is the most common malignancy of the GI tract and is a major cause of morbidity and mortality worldwide.
- In contrast, the small intestine, which accounts for 75% of the overall length of the GI tract, is an uncommon site for benign and malignant tumors.

we see it usually in old age people

- Colorectal cancer incidence peaks at 60 to 70 years of age,
- The dietary factors most closely associated with increased rates of colorectal cancer are low intake of unabsorbable vegetable fiber and high intake of refined carbohydrates and fat. it is theorized that reduced fiber content leads to decreased stool bulk and altered composition of the intestinal microbiota. This change may increase synthesis of potentially toxic oxidative by-products of bacterial metabolism, which would be expected to remain in contact with the colonic mucosa for longer periods of time as a result of reduced stool bulk. High fat intake also enhances hepatic synthesis of cholesterol and bile acids, which can be converted into carcinogens by intestinal bacteria.
- aspirin or other NSAIDs have a protective effect.

- Pathogenesis:

At least two genetic pathways have been described:

- APC/β-catenin pathway, which is activated in the classic adenoma-carcinoma sequence. accounts for up to 80% of sporadic colon tumors.

- microsatellite instability pathway, which is associated with defects in DNA mismatch repair and accumulation of mutations in microsatellite repeat regions of the genome.

APC mutation

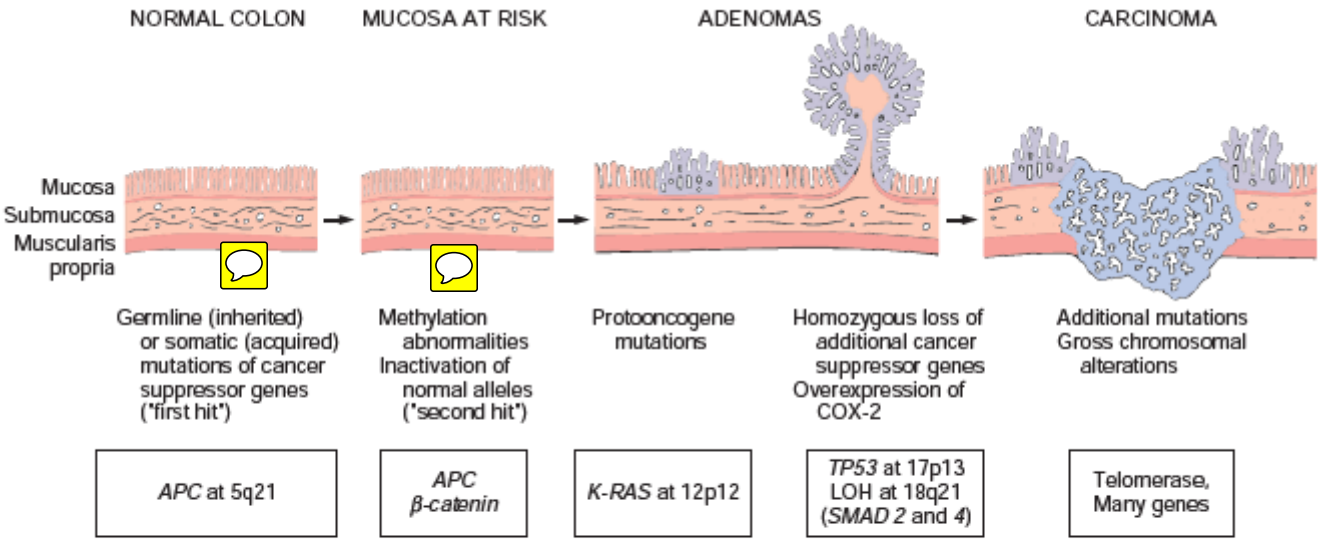

Figure 17-49 Morphologic and molecular changes in the adenoma-carcinoma sequence. Loss of one normal copy of the tumor suppressor gene *APC* occurs early. Individuals born with one mutant allele are therefore at increased risk of developing colon cancer. Alternatively, inactivation of *APC* in colonic epithelium may occur later in life. This is the "first hit" according to the Knudson hypothesis (Chapter 7). The loss of the intact second copy of *APC* follows ("second hit"). Other changes, including mutation of *KRAS*, losses at 18q21 involving *SMAD2* and *SMAD4*, and inactivation of the tumor suppressor gene *TP53*, lead to the emergence of carcinoma, in which further mutations occur. Although there seems to be a temporal sequence of changes, the accumulation of mutations, rather than their occurrence in a specific order, is most critical.

to develop colorectum cancer there must be mutation in APC gene by 2 hits , that means the 2 allels of the APC gene must be mutated to develop the cancer

mutation in DNA mismatch repair

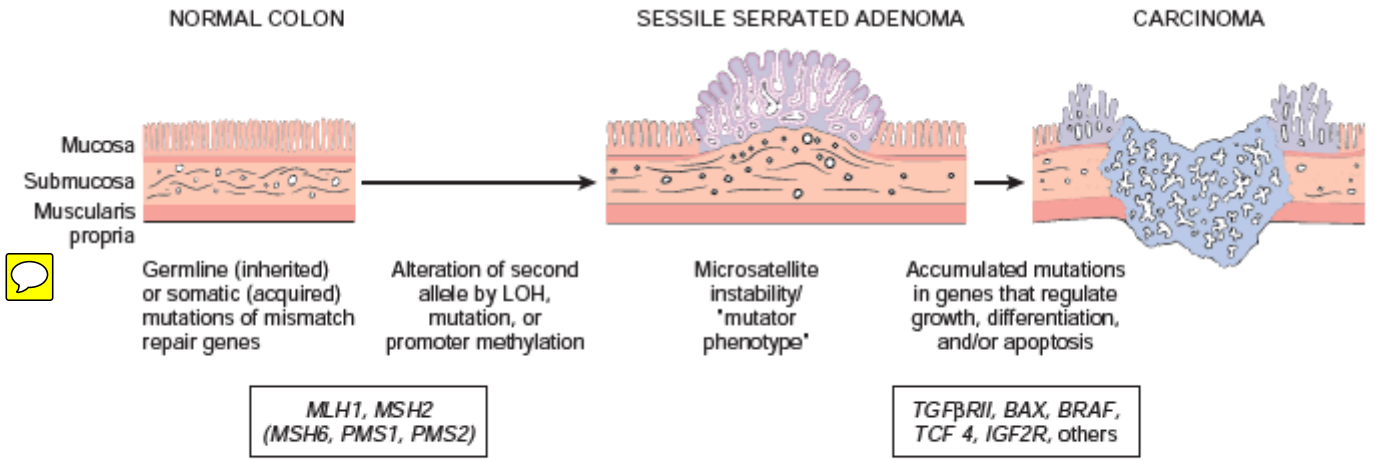

Figure 17-50 Morphologic and molecular changes in the mismatch repair pathway of colon carcinogenesis. Defects in mismatch repair genes result in microsatellite instability and permit accumulation of mutations in numerous genes. If these mutations affect genes involved in cell survival and proliferation, cancer may develop.

Sporadic colon cancer (70%-80%)	APC/WNT pathway	*APC*	None	Left side	Tubular, villous; typical adenocarcinoma
Sporadic colon cancer (10%-15%)	DNA mismatch repair	*MSH2, MLH1*	None	Right side	Sessile serrated adenoma; mucinous adenocarcinoma

- Clinical Features:

develop the most important clinical feature is anemia

Cecal and other right-sided colon cancers are most often called to clinical attention by the appearance of fatigue and weakness due to iron deficiency anemia--------**iron deficiency anemia in an older man or postmenopausal woman is GI cancer until proven otherwise**.

Left-sided colorectal adenocarcinomas may produce occult bleeding, changes in bowel habits, or cramping and left lower quadrant discomfort.

colorectum caner depend mainly on the stage

- the two most important prognostic factors are depth of invasion and the presence of lymph node metastases----- stage
- the liver is the most common site of metastatic lesions. The rectum does not drain via the portal circulation, hence carcinomas of the anal region that metastasize often circumvent the liver.

- Tumors of the Anal Canal:

Carcinomas of the anal canal may have typical glandular or squamous patterns of differentiation, recapitulating the normal epithelium of the upper and lower thirds, respectively.

appendix

- **Acute appendicitis**: is most common in children and adolescents. It is thought to be initiated by increased intraluminal pressure and compromised venous outflow, usually caused by a small stone-like mass of stool, or fecalith, or, less commonly, a gallstone, tumor, or mass of worms (oxyuriasis vermicularis). . which close the venous drainage

Typically, early acute appendicitis produces periumbilical pain that ultimately localizes to the right lower quadrant, followed by nausea, vomiting, low-grade fever, and a mildly elevated peripheral white

cell count. A classic physical finding is the **McBurney sign**, deep tenderness located two thirds of the distance from the umbilicus to the right anterior superior iliac spine (McBurney point). usually it comes with acute abdomin

- **Tumors of the Appendix**:

The most common tumor of the appendix is the welldifferentiated neuroendocrine (carcinoid) tumor. It is usually discovered incidentally at the time of surgery or examination of a resected appendix.

- Other tumors:

Conventional adenomas

non–mucin-producing adenocarcinomas

Mucinous cystadenoma

mucinous cystadenocarcinoma---- pseudomyxoma peritonei