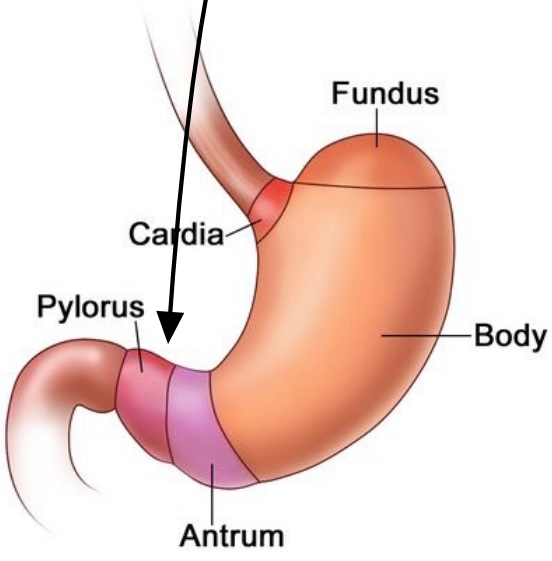
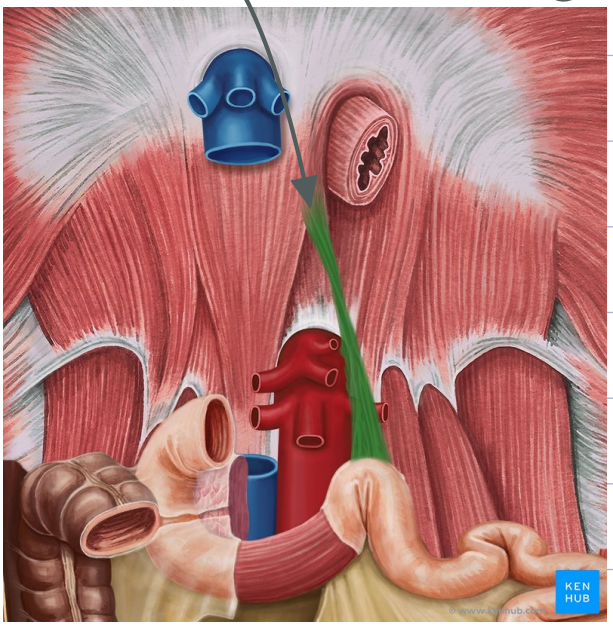
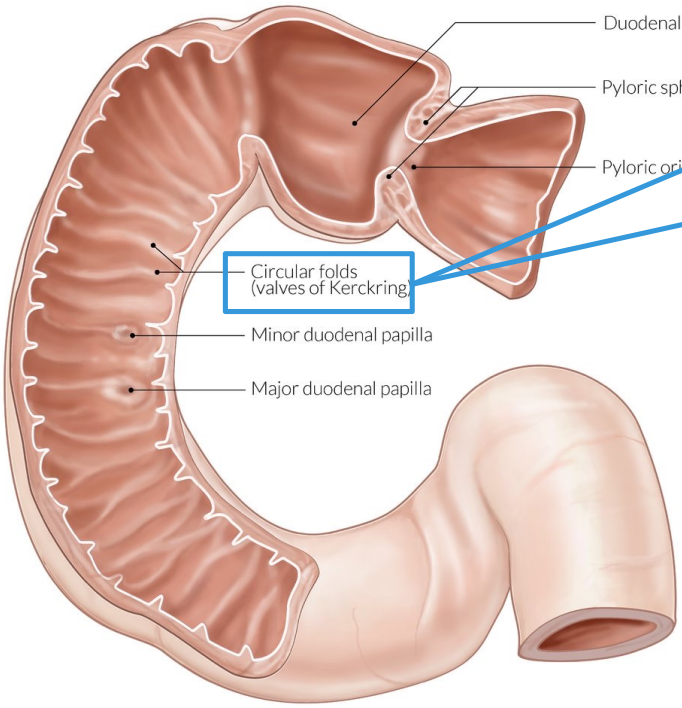
Anatomy of small intestine: 4-6 m

- Duodenum → most proximal
  → retroperitoneal
  → demarcated from stomach by pylorus
  → demarcated from jejunum by ligament of Treitz (suspensory lig. of duodenum)





- Jejunum → in peritoneal cavity
- Ileum → tethered to retroperitoneum by broad based mesentry
  → 40% of jejunoileal segment is defined as jejunum
  → 60% of jejunoileal segment is defined as ileum
  → ileum is demarcated from cecum by ileocecal valve

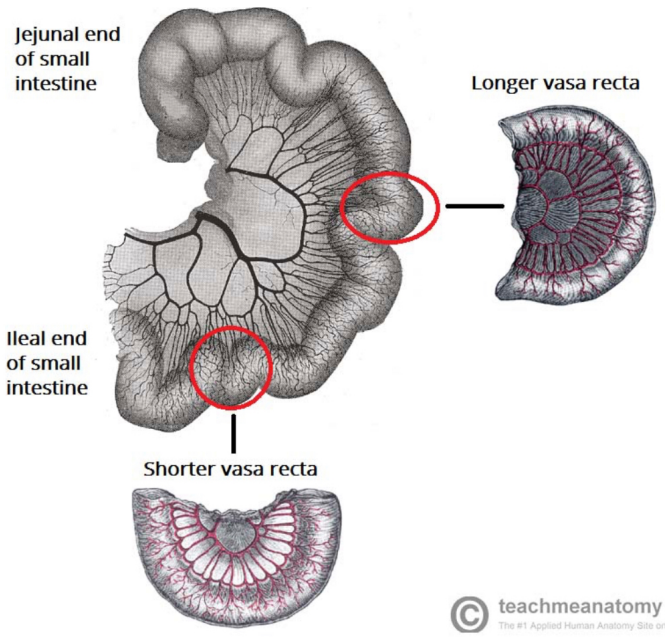


plicae circularis
valvulae conventes
these folds are visible radiographically
↳ not visible radiographically in Colon

Comparison of proximal & distal small intestine:

1) plicae circularis are more prominent in the proximal part
2) Proximal part has thicker walls & larger lumen
3) Proximal part has lesser fatty mesentry
4) Proximal part has longer vasa recta
5) The ileum contains larger aggregations of lymph nodes called Peyer's patches

The superior mesenteric artery arises from the aorta at the level of the L1 vertebrae, immediately inferior to the coeliac trunk. It moves in between layers of mesentery, splitting into approximately 20 branches. These branches anastomose to form loops, called arcades. From the arcades, long and straight arteries arise, called vasa recta.



Blood supply:-

- duodenum → proximal to major duodenal papillae
  - ↳ gastroduodenal → common hepatic → celiac
  - → distal to major duodenal papillae
    - ↳ inf. pancreaticoduodenal → Sup. mesentric
- jejunum
- ileum

→ Sup. mesentric artery

Venous drainage:

- duodenum
- jejunum
- ileum

→ Sup. mesentric vein

Lymph drainage:

through lymphatic vesseles coursing parallel to the arteries → draing into mesentric nodes → cisterna chyli → thoracic duct → left subclavian vein

Sympathetic innervation → Vagus nerve

Parasympathetic innervation → Splanchnic nerve

Physiology of small intestine:

- function → digestion of food
  → absorption of nutrients, water and electrolytes

- Carbohydrate and proteins → broken down in intestinal lumen
  final hydrolysis takes place ← by pancreatic enzymes
  at brush border of jejunum after which they are absorbed

- fats —(Pancreatic lipase / bile salts)→ fatty acid + monoglyceride
  ↳ seperates from bile salts in jejunum & absorbed for further processing

- absorption of bile salts & Vit. B12 only occurs at terminal ileum, where there are specific transporters
  ↳ if jejunum is resected
  ↳ ileum can do all the absorptive functions of jejunum

if ileum is resected ← terminal
- ↳ diminished bile salt pool
- ↳ Vit. B12 deficiency
- ↳ deficiency of fat soluble vitamins
  ↳ A, D, E, K

Inflammatory bowel disease:

- presence of idiopathic intestinal inflammation while infective & ischemic enteritis

- the only known (IBD) of small intestine
  - ↳ Crohn's disease (regional entritis)

- Crohn's disease → chronic full thickness inflammation that can affect → lips to the anal margin
  - → most common in north america & northern europe → 8/100 000 annually
  - → in last four decades the incidence has increased three folds → environmental factors
    - ↳ increased diagnostic modalities
  - → slightly more common in women than men
  - → most commonly diagnosed between 25 & 40 yrs
    - ↳ second peak incidence → 70 yrs
  - → in those countries of high prevalence
    - ↳ Caucasians → american whites & northern europeans
    - ↳ more common
    - less common in central europe, Asia, Africa, South America
  - → jews → 3-5 folds higher in Ashkenazi jews
    - ↳ less in jews of Israel than of Europe & USA → environmental factor

Aetiology:

- incompletely understood
- complex interplay of genetic & environmental factors
  - 10% have a first-degree relative
- smoking → increases the risk by 3 folds
  - → its exacerbating factor after diagnosis
- higher socioeconomic state → increases risk
- breast feeding → protective against development of CD

Pathogenesis:

As in UC there is increased gut mucosal permeability in early stages

→ increased passage of antigens

→ induce cell-mediated inflammatory response

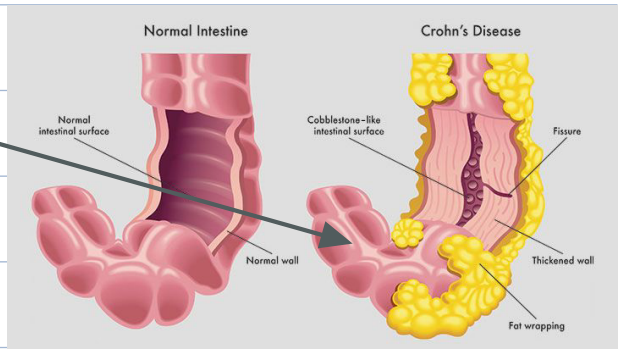
→ release of pro-inflammatory cytokines

→ IL-2 & TNF

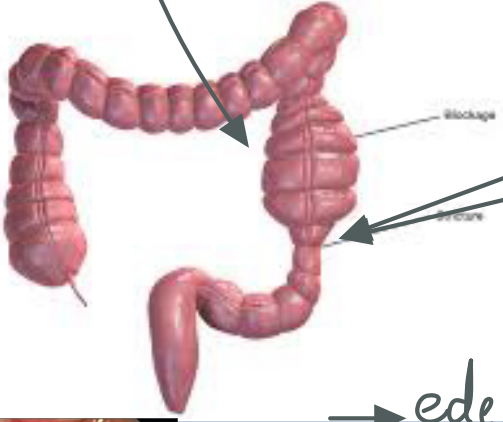
→ defect in Supressor T-cells (maybe)

- terminal ileum is most commonly involved (65%)
  - ↳ either in isolation or with colonic disease
- colitis alone occurs in 1/3 of cases & the remainder are patients with more proximal bowel involvement
- Stomach & duodenum are involved in 5%
- Perianal diseases occur in 25% of small bowel diseases BUT if occurs in 75% of CDs

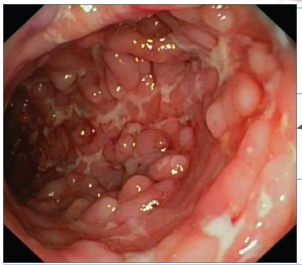
Macroscopically → fibrotic thickening of intestinal wall
→ lumen narrowing
→ fat wrapping (mesentric fat around bowel)



→ dilated bowel just proximal to the stricture with linear & snake like mucosal ulcerations



→ edema between the ulcers give rise to Cobblestone appearance of mucosa

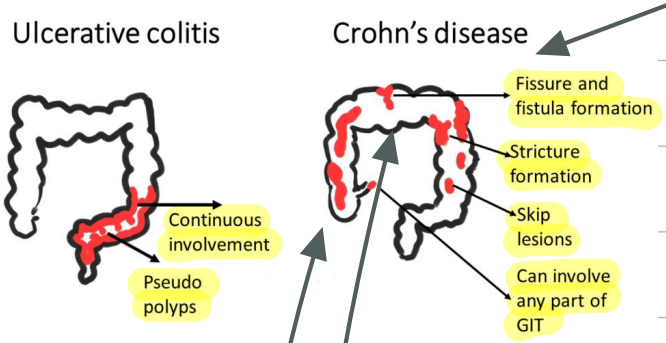


→ transmural inflammation (a charecteristic feature of CD) may lead to adhesion of bowel sigments to each other & to adjacent structures, inflammatory masses with mesentric abscesses & fistulae into adjacent organs

→ serosa is opaque & thickening of mesentry

→ enlarged mesentric lymph nodes

→ discontinuos or skip lesions



involving all layers

↳ lymphoid aggregates

→ focal areas of chronic inflammation

↳ Non-Caseating giant cell granuloma (66%)

→ multifocal arterial occlusions due to musculeris propria thickening

→ there maybe nerve cell hyperplasia & deep fissuring & ulceration within affected areas

→ there maybe normal areas immediately next to pathological areas

Differentiation between CD & UC

Crohn's disease	Ulcerative colitis
- can affect any part of the GIT, particularly small & large bowel	- affects the colon
- involves the full thickness of the bowel	- is a mucosal disease
- skip lesions	- continuous lesions in colon & rectum
- histologically → granulomas	- no granulomas
- often associated with perianal conditions	- not associated with perianal conditions
- if occurs in terminal ileum can mimic symptoms of appendicitis	- does not occur in UC
- resection is associated with recurrence	- resection of colon & rectum cures the patient
- more commonly causes strictures & fistulation	

Clinical features:

→ CD may present acutely with ileal inflammation & symptoms & signs resembling those of → acute appendicitis

→ can present with free perforation of small intestine
↳ resulting in local or diffuse peritonitis

→ may present with fulminant colitis → less common in UC
↳ most severe form of uncomplicated colitis

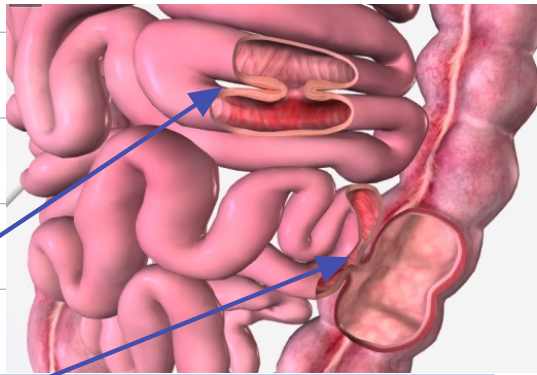
→ more commonly present with features of chronicity

→ small bowel CD → abdominal colicky pain
→ ↳ mostly postprandial
→ mild diarrhoea; extending over many months occuring in bouts
→ a tender mass palpable at right illiac fossa
→ intermittent fever
→ secondary anemia
→ weight loss

→ after many months of repeated attacks characterized by acute inflammation → the affected area of intestine begins to narrow with fibrosis
↳ more chronic obstructive symptoms

→ in children → might cause growth & sexual retardation

→ with progression
↳ adhesion
↳ transmural fissuring
↳ intra abdominal abscesses
↳ fistulae

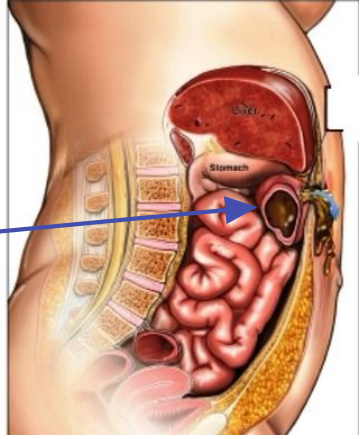


→ fistulation → enteroenteric

→ ileosigmoid

→ enterocutaneous: abd. wall

→ ileovesical: bladder or female genital tract



→ perianal CD: in the presence of active disease

superficial ulcers with undermined edges are relatively painless & can heal with bridging epithelium

perineal skin: bluish

deep cavitating ulcers are usually found in upper anal canal; painful; causes perineal abscesses & fistula

↳ associated with dense fibrous stricturing at anorectal junction

incontinence may develop as a result of destruction of anal sphincter musculature due to:

→ inflammation

→ abscess formation

→ fibrotic change

→ repeated surgical drainage

in severe cases the perineum may become densely fibrotic, rigid & covered with multiple discharging openings

watering - can perineum

Extraabdominal manifestations of CD:

a) related to disease activity
- → erythema nodosum
- → pyoderma gangrenosum
- → arthropathy
- → eye complications → iritis, Uveitis
- → aphthous ulcer
- → amyloidosis

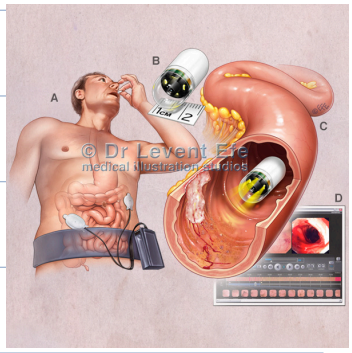
b) unrelated to disease activity
- → gallstones
- → renal calculi
- → Primary sclerosing cholangitis
- → Chronic active hepatitis
- → Sacroiliitis

Investigations laboratory
- → full blood count shows anemia
- → Vit. B_{12} defeciency might occur in terminal ileal disease or resection
- → Folate defeciency may occur as a result of diffuse small bowel disease or resection
- → active inflammatory disease → fall in Serum albumin, Magnesium, Selenium & Zinc
- → C-reactive protien and Esr may correlate disease activity

→ elevated concentration in the stools of calprotectin (a specific marker of inflammation → may support diagnosis
↳ moniter disease activity

Endoscopy:

- colonoscopy examination shows patchy inflammation
- normal areas in between areas of inflammation that are irregular and ulcerated with a mucopurulent exudate
- earliest findings are of aphthous ulcers surrounded by a rim of erythematous mucosa
- Stricturing is often present → malignancy must be excluded by multiple & often repeated mucosal biopsies
  ↳ an irregular CD stricture with polypoid mucosa may be almost macroscopically indistinguishable from malignancy
- terminal ileum may be ulcerated & strictured
- Upper GI symptoms may require upper GI endoscopy which may reveal deep longitudinal ulcers & cobblestoning of mucosa in the duodenum & stomach
- enteroscopy may reveal jejunal ulceration & stricturing
- capsule endoscopy is contraindicated due to possibility of the capsule becoming stuck in the narrow segment

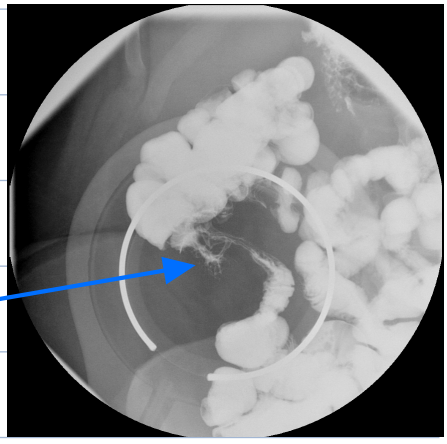


Imaging:

Ultrasound can demonstrate inflammed & thickened bowel loops, as well as fluid & abscess accumulation

small bowel enema is performed which is instilling contrast into the small bowel via nasoduodenal tube, and will show up areas of stricturing & prestenotic dilatation

the involved areas tend to be narrowed, irregular & sometimes when the length of terminal ileum is involved there may be the "string sign" of Kantor



incomplete filling of intestinal lumen

CT scans with oral contrast can demonstrate
- fistulae
- intra-abdominal abscesses
- bowel thickening
- dilatation

MRI is useful in assesing complex perianal diseases & excellent for assesing small bowel

labelled cell white scan is used to determine if an area is inflammed & guide decisions on medical treatment

→ in patients with enterocutaneous fistulae, fistulography is required to demonstrate the anatomy & complexity of the fistulae & allow adequate planning for future surgery

Treatment:

A) Medical treatment:

i) Steroids → traditional method for inducing remission in CD
→ remain important when rapid remission is required
→ they induce remission in 70-80% of cases with moderate to severe disease
→ they should be used in short courses only & tapered when a response has achieved
→ they reduce inflammation therefore they are ineffective in fibrostenotic diseases when the symptoms are related to obstruction
→ can be used as a topical agent in rectum
→ should not be used in maintenance therapy for CD & replaced with immuno-modulatory agents

ii) Aminosalicylates → colonic symptoms can be treated by 5-ASA agents (sulfasulazine, mesalamine) in a similar manner to that in UC
→ have limited efficacy in small bowel CD

iii) Antibiotics → metronidazole & ciprofloxacin is used
→ used for periods of a few weeks at a time
↳ especially in perianal disease

iv) Immunomodulatory agents → Azathioprine is used for its additive & steroid sparing effects & currently represents standard maintenance therapy
→ cyclosporin short-course intravenous cyclosporin is associated with 80% remission

v) 5- Monoclonal antibody therapy (Biologic agent): targeting tumor necrosis factor alpha & other key pro-inflammatory mediators

Infliximab → IV
→ given every 8 weeks for maintenance of remission

Adalimuab → Subcutaneous
→ every 1-2 weeks

→ they are widely used for induction & maintenance of remission
→ they are also effective for perianal treatment
→ disadvantages → expensive
→ risk of overwhelming bacterial infection
→ risk of specific malignancies over a long term
→ contraindications → active infection
→ TB
→ history of malignancy

B) Nutritional support: patients with moderate nutritional impairment will require nutritional supplementation & severly malnourished patients may require enteral tube or even intravenous feeding

C) Endoscopic treatment:
→ stricturing may be amendable to endoscopic treatment
→ dilatation of an inflammed or ulcerated stricture is contraindicated because of the risk of perforation

indications of surgery

- recurrent intestinal obstruction
- persistent, or less commonly massive acute bleeding
- free perforation of the bowel
- failure of medical therapy
- steroid dependent disease
- intestinal fistula
- Perianal disease → abscess, fistula, stenosis
- malignant change → notably in colon
  - → less commonly as a complicated of small bowel disease

D) Surgery for CD: Surgical resection will not cure CD, therefore it focuses on managing the complications of the disease

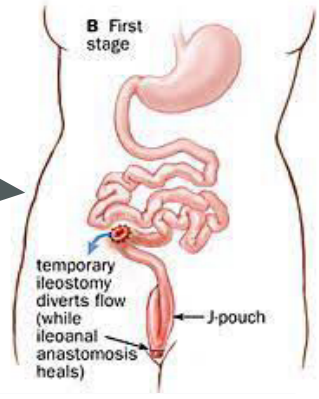
operations:

i) ileocecal resection: is the usual procedure for terminal ileal disease, with a primary anastamosis

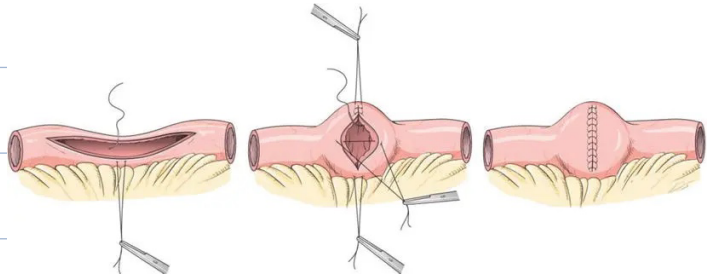
ii) segmental resections of short segments of small or large bowel strictures

iii) colectomy & ileorectal anastomosis

iv) temporary loop ileostomy



v) strictureplasty: a local widening procedure, to avoid small bowel resection & is thus is an important bowel sparing technique



vi) anal disease should be treated conservatively by simple drainage of abcesses & the use of setons through fistulae to avoid sphincter injury. Laying open fistulae (fistulotomy)