الجامعة السورية الخاصة كلية الطب البشري قسم الجراحة

الأمراض الجراحية الشائعة في الأمعاء الدقيقة و الغليظة Common small and large intestinal surgical diseases

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Topics

- Small bowel neoplasms.
 الآفات التنشؤية للأمعاء الدقيقة
- Meckele's diverticulum.
- متلازمة الكولون المتهيج IBD.
- أورام الكولون الخبيثة Colorectal cancer.

رتج میکل

Intestinal obstruction exists when blockage prevents the lacksquarenormal flow of intestinal contents through the intestinal tract.

يحدث الانسداد لأسباب تمنع الجريان الطبيعى ضمن الأنبوب الهضمي

Two types of processes can impede this flow. ۲

أسباب إعاقة الجريان

Mechanical. Functional.



Mechanical obstruction: پکي

An intraluminal obstruction (الانسداد عبر اللمعة) or a mural obstruction from pressure on the intestinal walls occurs. Examples are:

- intussusception
- polypoid tumors and neoplasms البوليبات السليمة و الخبيثة

الفتوق

- تضيقات الأمعاء Stenosis
- Adhesions الإلتصاقات
- Hernias
- abscesses.

الخراجات

• Functional obstruction:

الإنسداد الوظيفي

The intestinal musculature cannot propel the contents along the bowel.

عندما تعجز الحركات الحوية عن دفع محتوى الأمعاء باتجاه النهاية البعيدة

Examples are:

Amyloidosis الداء النشواني Muscular dystrophy الاعتلالات العضلية Endocrine disorders such as diabetes mellitus الأمراض الغدية و الاستقلابية كالداء السكري Neurologic disorders الاعتلالات العصبية

The obstruction can be partial or complete.

Its severity depends on:

يمكن للإنسداد أن يكون جزئياً أو كاملاً و تعتمد الخطورة على :

The region of bowel affected

المنطقة المصابة من السبيل الهضمي

The degree to which the lumen is occluded

درجة أو نسبة الانسداد

The degree to which the vascular supply to the bowel wall is disturbed.

Most bowel obstructions occur in the small intestine

غالبية الإنسدادات تحدث في الأمعاء الدقيقة

Adhesions الألتصاقات are the most common cause of small bowel obstruction, followed by hernias الفتوق and neoplasms . Other causes include intussusception الانفتال , volvulus الانفتال (ie, twisting of the bowel), and paralytic ileus الخذل المعدي المعوي. About 15% of intestinal obstructions occur in the large bowel; most of these are found in the sigmoid colon

ما يقارب 15% من انسدادات الأمعاء يحدث في الأمعاء الغليظة و غالبيتها في السين الكولوني

انسدادات الأمعاء الدقيقة

SMALL-BOWEL OBSTRUCTION

المراضية Epidemiology

The most frequently encountered surgical disorder.

≥75% is due to intra-abdominal adhesions.

تشكل الالتصاقات ضمن جوف البطن و بنسبة تصل لى 75% السبب الجراحي الغالب

Other: should be considered:

التشخيصات الأخرى تشتمل على : الفتوق Hernias Crohn's disease داء كرون Intestinal malrotation عدم دوران الأمعاء Mid-gut volvulus الأمعاء Mid-gut volvulus

انسدادات الأمعاء الدقيقة

Causes can be divided into three categories:

يمكن تصنيف الأسباب إلى :

Extraluminal causes such as adhesions, hernias, carcinomas, and abscesses

أسباب ضاغطة خارج اللمعة المعوية في حالات الالتصاقات و الفتوق و الأورام الخبيثة و الخراجات .

Intrinsic to the bowel wall (e.g., primary tumors)

أسباب ضمن الجدار المعوي كما في الأورام البدئية

Intraluminal obstruction (e.g., gallstones, enteroliths, foreign bodies, and bezoars)

إنسداد الأمعاء الدقيقة

SMALL-BOWEL OBSTRUCTION

PATHOPHYSIOLOGY:

الفيزيولوجيا الإمراضية

ألية حدوث الانسداد Obstruction onset

Gas and fluid accumulate within the intestinal lumen proximal to the site of obstruction.

يحدث تراكم في السوائل و الغازات ضمن لمعة الأمعاء الدقيقة في الجهة القريبة من منطقة الانسداد .

The bowel distends and intramural pressures rise.

توسع في العرى المعوية و ارتفاع في الضغط ضمن اللمعة Microvascular perfusion to the intestine is impaired, leading to intestinal ischemia, and, ultimately, necrosis. (strangulating bowel obstruction) من الممكن أن تؤدي هذه الحالة إلى إضطراب في التروية الدموية ، يعقبها نقص في تروية الأمعاء ، و يمكن أن يؤدى ذلك إلى حدوث نخر بنقص التروية (انسداد الأمعاء بسبب اختناق الأمعاء)

Progression to strangulation occurs quicker with complete bowel obstruction and more rapidly with closed loop obstruction which a segment of intestine is obstructed both proximally and distally (e.g., with volvulus).

FIGURE 38-6 Three causes of intestinal obstruction. (A) Intussusception invagination or shortening of the colon caused by the movement of one segment of bowel into another. (B) Volvulus of the sigmoid colon; the twist is counterclockwise in most cases. Note the edematous bowel. (C) Hernia (inguinal). The sac of the hernia is a continuation of the peritoneum of the abdomen. The hernial contents are intestine, omentum, or other abdominal contents that pass through the hernial opening into the hernial sac. 12/5/2016



BOWEL OBSTRUCTION

Clinical Presentation

Symptoms: الأعراض colicky abdominal pain غثيان

Vomiting

أقياء لمحتويات المعدة و الأمعاء تختلف طبيعتها حسب مستوى الانسداد انقطاع البراز بحسب مستوى الانسداد obstipation

Continued passage of flatus and/or stool beyond 6–12 h after onset of symptoms is characteristic of partial rather than complete obstruction.

في حال استمرار التبرز و طرح الغازات فهذا يرجح وجود اسداد تحت التام

BOWEL OBSTRUCTION



انسداد الأمعاء الدقيقة

Diagnosis



The diagnostic evaluation should focus on the following goals:

Distinguishing mechanical obstruction from ileus Determining the etiology of the obstruction Discriminating partial from complete obstruction Discriminating simple from strangulating obstruction. Determining the site of obstruction.

انسداد الأمعاء الدقيقة و الغليظة

- Diagnosis
 - Careful history taking:
 - prior Hx of abdominal operations \rightarrow ? presence of adhesions.
 - Hx of abdominal disorders (e.g., intraabdominal cancer or inflammatory bowel disease).
 - Careful examination:
 - a meticulous search for hernias (particularly in the inguinal and femoral regions) should be conducted.
 - The stool should be checked for gross or occult blood, the presence of which is suggestive of intestinal strangulation.

LARGE BOWEL OBSTRUCTION :Pathophysiology

- As in small bowel obstruction
 - large bowel obstruction results in an accumulation of intestinal contents, fluid, and gas proximal to the obstruction.
 - Obstruction in the large bowel can lead to severe distention and perforation unless some gas and fluid can flow back through the ileal valve.
 - Large bowel obstruction, even if complete, may be undramatic if the blood supply to the colon is not disturbed.

LARGE BOWEL OBSTRUCTION :Pathophysiology

- If the blood supply is cut off → intestinal strangulation and necrosis (ie, tissue death) occur; this condition is life threatening.
- dehydration occurs more slowly than in the small intestine because the colon can absorb its fluid contents and can distend to a size considerably beyond its normal full capacity.

LARGE BOWEL OBSTRUCTION :Clinical Manifestations

- Large bowel obstruction differs clinically from small bowel obstruction in that the symptoms develop and progress relatively slowly.
- In patients with obstruction in the sigmoid colon or the rectum, constipation may be the only symptom for days. loops of large bowel become visibly outlined through the abdominal wall, and the patient has crampy lower abdominal pain.
- Finally, fecal vomiting develops. Symptoms of shock may occur.

- X-RAY SERIES:
- Obstruction is usually confirmed with radiographic examination.
- Abdominal series consists of :
 - supine Abdominal X-ray
 - upright Abdominal X-ray
 - Upright Chest X-ray
 - The finding most specific for small-bowel obstruction is the triad of
 - dilated small-bowel loops (>3 cm in diameter)
 - air-fluid levels seen on upright films
 - a paucity of air in the colon.
- False negative :
 - Proximal obstruction
 - The bowel lumen is filled with fluid but no gas.

Assessment and Diagnostic Findings

- Diagnosis is based on symptoms and on x-ray studies.
- Abdominal x-ray studies (flat and upright) show a distended colon.
- Barium studies are contraindicated.







- CT Abdomen:
- Findings include:
 - A discrete transition zone with dilation of bowel proximally, decompression of bowel distally
 - intraluminal contrast that does not pass beyond the transition zone
 - Colon containing little gas or fluid.
 - Strangulation is suggested by:
 - Thickening of the bowel wall
 - Pneumatosis intestinalis (air in the bowel wall)
 - Portal venous gas
 - Mesenteric haziness
 - Poor uptake of intravenous contrast into the wall of the affected bowel.

CT scanning also offers a global evaluation of the abdomen and may
 2/5/2016 therefore reveal the etiology of obstruction.





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- SMALL-BOWEL SERIES (SMALL-BOWEL FOLLOW-THROUGH)
 - can be helpful
 - contrast is swallowed or instilled into the stomach through a nasogastric tube.
 - Barium or water-soluble contrast agents (Gastrografin)

ENTEROCLYSIS

- 200–250mLof barium followed by 1–2 L of a solution of methylcellulose in water is instilled into the proximal jejunum via a long nasoenteric catheter.
- Enteroclysis is rarely performed in the acute setting
- Offers greater sensitivity for lesions that may be causing partial small-bowel obstruction.







BOWEL OBSTRUCTION

• Therapy

- Fluid resuscitation.
- A nasogastric (NG) tube to evacuate air and fluid from stomach.
- An indwelling bladder catheter to monitor urine output.
- Central venous or pulmonary artery catheter monitoring may be necessary
- Broad-spectrum antibiotics
- The standard therapy for bowel obstruction is expeditious surgery with the exception of specific situations

MESENTERIC ISCHEMIA

- Therapy
- For embolus or thrombus-induced acute mesenteric ischemia, the standard treatment is surgical revascularization
 - embolectomy/thrombectomy/mesenteric bypass
 - Emergent laparotomy and resection, if signs of peritonitis develop
 - thrombolysis, using agents such as streptokinase, urokinase, or recombinant tissue plasminogen activator, is an alternative therapeutic option in sick, unstable pt.
- The standard treatment of NOMI is selective infusion of a vasodilator, most commonly papaverine hydrochloride, into the superior mesenteric artery.
- Anticoagulation:
 - Heparin administration is associated with reductions in mortality and recurrence rates, and should be initiated as soon as the diagnosis is made.
 - Most patients should be maintained on warfarin to achieve chronic anticoagulation for 6–12 months.

MESENTERIC ISCHEMIA

- Outcomes
 - Mortality rates among patients with acute **arterial** mesenteric
 - ischemia range from 59–93%.
 - Mortality rates among patients with acute mesenteric <u>venous</u> thrombosis range from 20–50%.
 - Perioperative mortality rates associated with surgical therapy for chronic mesenteric ischemia range from 0–16%

Take Home Points

- Careful history (pain, other GI symptoms)
- Remember DDx in **broad** categories
- Narrow DDx based on hx, exam, labs, imaging
- Always perform ABC, Resuscitate before Dx
- If patient's sick or "toxic", get to OR (surgical emergency)
 - Ideally, resuscitate patients before going to the OR
- Don't forget GYN/medical causes, special situations
- For acute abdomen, think of these commonly (below)

Perf DU	Appendicitis +/- perforation	Diverticulitis +/- perforation	Bowel obstruction
Cholecystitis	Ischemic or perf bowel	Ruptured aneurysm	Acute pancreatitis
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- Disorders: ulcerative colitis and Crohn's disease
- Incidence: 10,000 new cases per year in U.S.
- Age at onset: 15 to 40 years old
- Etiology: unknown, some familial
- Pathophysiology
 - Ulcerative colitis: inflammation of colon originates in rectum. Erosion, ulcers, abscesses, and necrosis result.
 - Crohn's disease: recurrent inflammatory process in any portion of GI tract. Bowel wall thickened and rigid, lumen narrows, fissures form.

Crohn's Disease
Definition

Chronic , transmural, inflammatory condition, which can involve the entire alimentary tract from mouth to anus (usually discontiously) with involvement of extraintestinal tissue.



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Historical Background

- 1932-Crohn, Ginzburg, Oppenheimer
 - "a disease of the terminal ileum, affecting mainly young adults, CHX by a subacute or chronic necrotizing & cicatrizing inflammation
 - They noted that the disease process lead to multiple fistulas.
 - They call it regional ileitis "believed that it involved only the terminal ileum".
- 1960-Lockhart, Mummery & Morson
 First to report C.D. of the large intestine.

Incidence and Prevalence of Crohn's Disease in Various Geographic Regions

Author(s)	Country	Annual Incidence/ 100,000	Prevalence/ 100,000
Garland et al. (1981)	Unites states (15 areas)	3.4 - 4.95	
Binder et al. (1982)	Denmark	2.70	34.0
Gollop et al. (1988)	Rochester, Minn., U.S.A.	4.00	
Haug et al. (1989)	Western Norway	5.30	
Stowe et al. (1990)	Rochester, N.Y., U.S.A	5.00	
Probert et al. (1993)	England		
	Europeans		75.8
	South Asians		33.2
Mate-Jimenz et al. (1994)	Spain	1.61	19.8
	Urban	1.87	
Odes et al. (1994)	Rural	0.86	
Oues et al. (1994)	Israel	4.20	50.6
	Asian-African born Jews	4.60	55.0
	European-American born Jews	3.90	58.7
The second at (100 A)	Bedouin Arabs		8.2
Islanos et al. (1994)	Greece	0.30	
Anseline (1995)	Australia	2.10	
Lindgren et al. (1996)	Sweden		
Manousos et al. (1996)	Crete, Greece	3.00	34.0
Moum et al. (1996)	Southeastern Norway	5.80	94.0
Tragnone et al. (1996)	Italy	2.30	
Hanauer and Meyers (1997)	United States A.Kubtan		39

Epidemiology

- Precise incidence is difficult to determine.
- Russell & Stockbrugger noted rapid increase in freq. of C.D. occurring between 1965 – 1980.
- World wide prevalence is estimated to be 10 –70 cases/100,000 population with incidence of 0.5 6.3 cases /100,000 pop./year.
- Highest rates are reported in Scandinavian countries and Scotland followed by England and north America. But are decreased in central and southern Europe.

Age & Gender

- Bimodal age distribution with a peak onset b/w 15-30 yrs. & second smaller peak b/w 55-80 yrs.
- Female in general 20%-30% greater risk of developing C.D.

Ethnicity

- Jewish people are afflicted 3-8 times > non Jews
- Jewish people from different countries are not equally affected with remarkable predominance of north American and south African Jewish
- In Israel a study of the Jewish population in Tel-Aviv revealed that Ashkenazi Jews born outside Israel increased 4 times incidence compared with Ashkenazi Jews born in Israel (16.69/100,000 Vs. 4.19/100,000)
- In England Hindus & Sikhs have a decrease incidence of C.D. Than the general population.
- American black & Indian populations are at low risk of IBD.

Epidemiology of Inflammatory Bowel Disease

1-10 (CD) 3-15 (UC)
20-100 (CD) 50-80 (UC)
Northern Countries > South Countries
Peak : 15-30 Second Peak 50-80 (CD)
M = F
Whites > Blacks
Jewish > Non-Jewish
Associated with CD : Protective in UC
May be protective in UC
Chromosome 16 (CD) Chromosome 3, 7, 12 (UC and CD) TNF- (CD); IL-1A (CD) HLA-A2; HLA-DR1; DQw5 (CD) HLA-DR2 (UC)

Etiology & Pathogenesis

UNKNOWN

We know neither its cause nor its cure

Etiology & Pathogenesis Cont'd

Sartor et al. reviewed 3 most recent theories of etiology of C.D.

- 1. Specific infectious agent
 - 1. mycobact. Para T.B.
 - 2. Paramoxyvirus & measles
 - 3. Listeria monocytogens
- 2. Defective mucosal barrier resulting in increase exposure to Ags.
- 3. Abnormal host response to a ubiquitous Ags

Other Factors

- Family history
 - 10%-20% increase incidence in close degree relatives
 - Study in Denmark showed 1st and 2nd generation relatives of Pts. With C.D. have 1-3 folds increase prevalence.
 - Probert et al. -1st degree relative of Pt. with C.D. increase risk up to 35 times.
 - Monozygotic twins > dizygotic twins
- Dietary factors
 - Increase intake of refined sugar and starch
 - Decrease intake of fresh fruits
- Smoking
 - Oxford study found R..R.. Of 3.4
 - Increase recurrence rate among smokers
- OCP
 - Use of OCP for 1-3 years has a R..R.. Of 2.5 & 4.3 > 3 years.
- 12/5/2016 Risk decrease after D/C OCP M.A. Kubtan

Pathology

- C.D. can affect any part of GIT
 - Ileocecal region(41%-55%) ,SI (30%-40%) ,colonic (14%-26%)
 - 2/3 of crohn's colitis pts. have total involvement
- Acute or active phase is marked by
 - Aphthous mucosal ulcers
 - Lymphoid aggregates
 - Granulomas (2/3 of pts.)
 - Transmural chronic inflammation with fissures & fistulas
- Heeling phase
 - CHX. By fibrosis with stricture formation & chronic ulcers

Gross





The wall of the ileum severely affected by Crohn's disease is inflamed and red. There are linear mucosal ulcerations present (yellow arrows), which would be focal if you could see the entire ileum. In addition, there is severe thickening 12/5/2016 of the bowel wall.

Gross features:

- Skip lesions
- Creeping fat
- Thickenedwall +/strctures
- Fissuring ulcers with a linear, serpentine appearance grossly; cobblestoning of mucosa

– Fistulae



This is another example of Crohn's disease involving the small intestine. Here, the mucosal surface demonstrates an irregular nodular appearance with hyperemia and focal superficial ulceration. The distribution of bowel involvement with Crohn's disease is irregular with more normal intervening "skip" areas.

Perforated terminal ileitis.

Crohn's disease may present in a similar manner to that of acute appendicitis. A 22 year old male has had a history of several bouts of episodic diarrhea associated with recurrent abdominal pain, lassitude and pyrexia. Although the terminal ileum has perforated in the patient, a much more common presentation is that of simple terminal ileitis. Consider a differential diagnosis of eosinophilic ileitis, other inflammatory bowel disease or lymphoma.



Crohn's Disease-Microscopic





- Transmural inflammation
 - Neutrophilic into glands
 - Crypt abscesses
 - +/- granulomas
- Signs of chronic injury: architectural distortion, atrophy, and metaplasia.
- Possible dysplasia late in disease





Granulomas are often in C.D. but not in UC.

Transmural inflammation in crohn's disease

Fissuring, linear ulcers in Crohn's Disease



Typical granuloma of Crohn's disease Light micrographs showing granulomatous lesion that is diagnostic of Crohn's disease. Low and high power views show a central giant cell surrounded by epitheliod cells and rimmed by lymphocytes. Courtesy of the American Gastroenterological Association©. This slide cannot be downloaded but may be purchased as part of a set from the AGA through Milner-Fenwick, Inc at 1-800-432-8433.

Clinical Features

Depend mainly on anatomical location of the disease

- Symptoms
 - Abd. Pain
 - Diarrhea
 - Wt. loss
 - Bleeding per rectum
 - Anorexia, fever, N ,V
 - Recurrent oral aphthous ulceration
 - Hx. Of perianal disease, extraintestinal manifestation
 - In children growth retardation & failure of 2nd sex Chx.



Perianal Crohn's disease Perianal fistulas and ulcers in a patient with Crohn's disease. There are bilateral posterior perianal fistulas (arrows) and two right posterior ulcers (arrowheads). Courtesy of Alain Bitton, MD, FRCPC.



Anal fistulas and abscess in Crohn's disease This picture shows anterior perianal fistulas involving the vulva and anterior perineum (arrows) in a woman with Crohn's disease being prepared for surgery. The surrounding skin is erythematous and indurated. An abscess (arrowhead) appears as a localized swelling. Hypertrophic skin tags (blue arrows) in the anal canal are commonly observed in perianal Crohn's disease and may be confused with external hemorrhoids. Courtesy of Alain Bitton, MD, FRCPC.



Perianal Crohn's disease Perineum in a woman with Crohn's disease shows a large posterior ulcer (arrow) witMahtebtor edematous fissure (arrowhead). Courtesy of Alain Bitton, MD, FRCPC.

Clinical Features-cont'd

- Signs
 - Pallor
 - Cachexia
 - Clubbing
 - Abdominal mass or tenderness
 - ? Evidence of obstruction
 - Anemia
 - Hypoproteinemia
- Exacerbating factors
 - Intercurrent infection (URTI or enteric)
 - Cigg. Smoking
 - NSAIDs.
 - ?Stress & psychological predisposition

Dx.

- The onset of C.D. is nearly always obscure & poorly defined thus, delayed diagnosis
- In only 30% of pts. Dx. is made within one year of onset of Sx.
- Dx. Is usually made during an acute exacerbation
- Hx. & ph.
- Radiology
 - Contrast radiography is essential for DDx. & to determine the severity of the disease
 - Indications include recurrence of Sx. After surgery & pts. With progressive disease requiring surgical Rx. or change in his medical plan.
 - Barium studies.
 - CT scan----- masses & abcesses.
 - ERCP ----- hepatobiliary involv.(sclerosing cholangitis).



Cobblestone appearance in Crohn's disease Small bowel follow through study demonstrates diffuse thickening of the small bowel mucosa in a patient with Crohn's disease. The cobblestone appearance is produced by barium being dispersed between the edematous inflamed mucosa. Courtesy of Norman Joffe, MD.



Crohn's colitis Double contrast barium enema in a patient with Crohn's disease shows extensive ulceration of the wall of the colon associated with mucosal thickening and inflammation. 12550006tesy of Jonathan Kruskal, MD, PhD.



Crohn's disease with abscess and fistulae Small bowel follow through study demonstrates an abscess cavity (white arrow) with fistulae connecting the cavity to the adjacent small bowel (black arrows). Note the marked thickening of the inflamed mucosal folds (small arrows). Courtesy of Jonathan Kruskal, MD, PhD.



Crohn's disease of the stomach This upper GI series, performed in a young man with known Crohn's disease of the terminal ileum shows numerous rounded filling defects in the stomach produced by edematous mucosa. In some of these areas, small central collections of barium are demonstrated (arrow) resulting from superficial erosions. These features are suggestive of Crohn's disease, but may features are suggestive of Crohn's disease, but may and in viral gastritis. Courtesy of Jonathan Kruskal, MD, PhD.



Right lower quadrant abscess in Crohn's disease Single axial CT scan of the lower abdomen demonstrates an abscess (arrowheads) extending from the markedly thickened and inflamed terminal ileum (arrow). The presence of contrast material within the abscess confirms a communication with the adjacent ileum. Courtesy of Norman Joffe, MD.



Ileocecal fistulae in Crohn's disease Small bowel follow through examination demonstrates nodular thickening of the terminal ileal mucosal folds in a patient with Crohn's disease (black 12/5/2@rcow). Several fistulae extend from the termine (ubtan ileum to the adjacent cecum (white arrows). Courtesy of Jonathan Kruskal, MD, PhD.



Chronic Crohn's colitis Barium enema demonstrates sacculations along the medial border of the ascending colon (arrows) produced by scarring and fibrosis in a patient with Crohn's disease. Courtesy of Jonathan Kruskal, MD, PhD.



Crohns disease Small bowel follow through examination demonstrates nodular filling defects arising on thickened folds in the terminal ileum (arrows). These features 59 are characteristic of Crohn's disease. Courtesy of Jonathan Kruskal, MD, PhD.



String sign in Crohn's disease Small bowel follow through study shows marked narrowing, irregularity and ulceration in the distal ileum (arrows) in a patient with Crohn's disease. Courtesy of Jonathan Kruskal, MD, PhD.



Cont'd

- Endoscopy.
 - Colonoscopy
 - Extent of the dis.
 - Confirm radiographic abnormalities.
 - Bx.
 - Shows aphthus ulcers, linear ulcers, ulcers in otherwise normal appearing mucosa, cobblestoning & asymmetric discontinous involvement.



Apthous ulcers in Crohn's disease Lower endoscopy demonstrates small discreet apthous ulcers that are characteristic of early lesions in Crohn's disease. Courtesy of James B McGee, MD.



Pseudomembranous colitis Endsocopy of pseudomembranous colitis reveals small groups of pseudomembranes (left panel) that may be confused grossly with the apthous ulcers of Crohn's disease (right panel). These lesions can be distinguished since pseudomembranes are present on top of the mucosa and do not result in ulceration of the underlying tissue. Courtesy of James B McGee, MD.



Asymmetric distribution of lesions in Crohn's disease Lower endoscopy in Crohn's disease demonstrates the characteristic patchy erythema (left panel) and ulceration (right panel) that occur next to areas of normal mucosa. Courtesy of James B McGee, MD.



Linear ulcerations in Crohn's disease Lower endoscopy shows linear ulcers that can course for several centimeters along the longitudinal axis of the colon in Crohn's disease. Courtesy of James B McGee, MD.



Endoscopic progression of Crohn's disease Ulcers are the dominant endoscopic feature in Crohn's disease. These tend to be linear and discontinuous, or "skip lesions". Early changes may be only patchy erythema (panel A) or aphthoid ulcers (panel B). Linear ulcers (panel C) are seen with more advanced disease, culminating in very deep and long serpiginous ulcers (panel D). Courtesy of James B McGee, MD.

12/5/2016

D.Dx.

- Chronic U.C.
 - The initial Dx. of U.C. Is confirmed in 80% of instances, is changed to crohn's colitis in 10% to15%, and remains indeterminant in 5% to 10%.
 - Acute appendicitis.
 - Other infections
 - Yersinia
 - Compylobacter
 - Shigella
 - Salmonella
 - E.coli
 - Т.В.
 - Lymphoma.
 - Ch. Mesenteric ischemia.

Extraintestinal manifestation

- Peptic ulcers.
- Arthritis.
- Skin lesions---(pyoderma gang.,Eryth.Nodosum)
- Cholelithiasis.
- Renal calculi.
- Eye lesions.

Extraintestinal Manifestations of IBD⁺

Common Extraintestinal Manifestations Musculoskeletal

Arthritis – colitic type, ankylosing spondylitis, isolated joint involvement Hypertrophic osteoarthropathy – clubbing, periostitis, metastatic Crohn's disease Miscellaneous – osteoporosis, aseptic necrosis, polymyositis

Skin and mouth

Reactive lesions – erythema nodosum, pyoderma gangrenosum, aphthous ulcers, vesiculopustular eruption, necrotizing vasculitis

Specific lesions - fissures and fistulas, or al Crohn's disease, drug rashes

Nutritional deficiency - acrodermatitis enteropathica (Zn), purpura (vitamin C & K), glossitis (vitamin B), hair loss and brittle nail (protein)

Associated diseases - vitiligo, psoriasis, amyloidosis, epidermolysis bullosa acquisita

Hepatobiliary

Specific complications – primary sclerosing cholangitis and bile duct carcinoma Associated inflammation – autoimmune chronic active hepatitis, pericholangitis, portal fibrosis and cirrhosis, granuloma in Crohn's disease Metabolic – fatty liver, gallstones associated with ileal Crohn's disease

Ocular

Uveitis (Initis), episolenitis, soleromalacia, conneal ulcers, retinal vascular disease

Metabolic

Growth/Fetal dation in children and adolescents, delayed set/dation

Less Common Extraintestinal Manifestations

Blood and vascular

Anemia due to iron, folate or B12 deficiency or autoimmune hemolytic anemia, thrombocytopenic purpura; leukocytosis, and thrombocytosis; Thrombophlebitis and thromboembolism, arteritis and arterial occlusion.

Renal and genitourinary tract

Uninary calculi (oxalate stones in ileal disease), local extension of Crohn's disease involving uneter or bladder, amyloidosis

Renal tubular damage with increased uninary excretion of various enzymes, eg, beta N-acetyI-D-glucosaminidase.

Neurological

Up to 3 percent of patients may have non-latrogenic various neurologic involvements including peripheral neuropathy, myelopathy, myasthenia gravis, and cerebro vascular disorders. Incidence equal in both UC & CD usually five to six years after the onset of IBD and frequently associated with other extraintestinal manifestations.

Broncho pulmonary

Pulmonary fibrosis, vasculitis, bronchitis, acute laryngotracheitis. Abnormal pulmonary function tests, without clinical symptoms, are common in up to 50 percent of cases.

Cardiac

Pericarditis, myocarditis and heart block - UC>CD. Pericarditis may also occur from sulfasalazine/SASA

Pancreas

Acute pancreatitis - CD>UC: risk factors include 6 mercaptopurine and 5 amino salicylate therapy, duodenal Crohn's disease

†Repትሪሲፈሪዸሪ⁶with permission from Das , KM , Dig Dis Sờiሳ ነቃቃንግ44 :8.



Erythema nodosum Patient with inflammatory bowel disease with red nodular areas on the shins which are characteristic of erythema nodosum. (Courtesy of the American Gastroenterological Association©. This slide cannot be downloaded but may be purchased as part of a set from the AGA through Milner-Fenwick, Inc at 1-800-432-8433.)



Episcleritis Patient with episcleritis associated with inflammatory bowel disease showing the characteristic injection of the ciliary vessels. (Courtesy of the American Gastroenterological Association©. This slide cannot be downloaded but may be purchased as part of a set from the AGA through Milner-Fenwick, Inc at 1-800-432-8433.)



Anterior uveitis Anterior uveitis in a patient with inflammatory bowel disease is characterized by injection of the conjunctiva and opacity in the anterior chamber. Courtesy of the American Gastroenterological Association©. This slide cannot be downloaded but may be purchased as part of a set from the AGA through Milner-Fenwick, Inc at 1-800-M.A.Kubta2432-8433

Comparison of Ulcerative Colitis and Crohn's Colitis

Manifestation	Ulcerative C	colitis Cro	hn's Colitis
Clinical Features			
Bleeding per rectum	3+		1+
Diarrhea	3+	3+	
Abdominal pain	1+	1+ 3+	
		Especially w	ith involvement of ileum
Vomiting	R		3+
Fever	R	R=rare, 0=not found,	2+
Palpable abdominal mass	R	1+=maybe present,	2+
Weight loss	+	2+=common,	3+
Clubbing	R	3+=usual finding,	1+
Rectal involvement	4+	4+=characteristic	1+
Small bowl involvement	0		4+
Anal and perianal involvement	R		4+
Risk of carcinoma	1+		1+
Clinical course	Relapses/remis	sion Slo	wly progressive
Radiologic			
Thumb printing sign on 127572016 barium enema	R M.A.Kuk	otan	1+ 69

Comparison of Ulcerative Colitis and Crohn's Colitis-cont'd

Manifestation	Ulcerative Colitis	Crohn's Colitis	
Endoscopic			
Distribution	Symmetric	Asymmetric	
Continuous involvement	4+	1+	
Rectal	4+	1+	
Vascular architecture	Absent	1+	
Friability	4+	1+	
Erythema	3+	1+	
Spontanous petechaise	2+	R	
Profuse bleeding	1+	R	
Aphthous ulcer	0	4+	
Serpiginous ulcer	R	4+	
Deep longitudinal ulcer	0	4+	
Cobblestoning	0	4+	
Mucosa surrounding ulcer	Abnormal	+-normal	
Pseudopolyps	2+	2+	
Bridging	R	1+	
Gross Appearance			
Thickened bowel wall	0	4+	
Shortening of bowel	2+	R	
Fat creeping onto serosa	0	4+	
Segmental involvement	0	4+	
Aphthous ulcer	RI A Kubtan	4+ 70	
Linear ulcer	0	4+	

Comparison of Ulcerative Colitis and Crohn's Colitis-cont'd

Manifestation	Ulcerative Colitis	ve Colitis Crohn's Colitis	
Microscopic Pictures			
Depth of involvement	Mucosa and submucosa	Full thickness	
Lymphoid aggregation	0	4+	
Sarcoid-type granuloma	0	4+	
Fissuring	0	2+	
Goblet cell mucin depletion	4+	1+	
Intramural sinuses	0	1+	
Operative Treatment			
Total proctolectomy	Excellent option in selected patients	Indicated in total large bowel involvement	
Segmental resection	R	Frequent	
Ileal pouch procedure	"Gold standard"	Contraindicated	
Prognosis			
Recurrence after total proctocolectomy	0	3+	
Complications			
Internal fistula	R	4+	
Intestinal obstruction	0	4+	
(stricture of infection)			
Hemorrhage	1+	1+	
Sclerosing cholangtitis	1+	R	
Cholelithiasis	0	2+	
Nephrolithiasis	0	2+	
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"Mr. Osborne, may I be excused? My brain is full."
Common small and large intestinal surgical diseases Part II

Khayal AlKhayal, MD, FRCSC Assistant Professor of Surgery Consultant Colorectal Surgeon 2010

Colorectal cancer

Outline

- Definitions
- Polyps
- Basics of colorectal cancer
- Surgery
- Staging

Perspective



Definitions

- Colon = large bowel = large intestine
- Rectum terminal portion of the colon
- Polyp benign growth; not invasive
- Adenoma type of polyp
- Cancer malignant growth; invasive
- Stage where the cancer is growing
- Primary the original tumour, where it started
- Metastases where the tumour has spread to

Cancer

A cancer cell :

- is immortal (lives forever)
- multiplies uncontrollably
- can live on its own without neighbors
- can live in other parts of the body

Colon and Rectum



Colorectal Cancer

- Most cancers are acquired some are inherited
- Almost all cancers begin as a benign polyp or adenoma
- Only a tiny percentage of adenomas become cancers

What is a polyp?



Fig. 22-7 Pedunculated polyp.



Polyp - Cancer Sequence

- The process from benign polyp to cancer takes from 7 10 years
- The transformation into cancer is based on
 - the type of polyp
 - Size of polyp

• Multiple polyps = greater risk of cancer



Chromosome 5-APC Tumor suppressor gene inactivation

> Chromosome 12 + K-ras Proto-oncogene activation

> > Chromosome 18 – DCC

Chromosome 17 – p53

Other alterations

The Effect of Age on the Incidence of Colorectal Cancer and Colorectal Polyps



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Removing polyps prevents cancer Colonoscopy

Colorectal Carcinoma

Classification

Adenocarcinoma 95% Carcinoid Lymphoma Sarcoma Squamous cell carcinoma

Epidemiology

- 3th most common malignancy worldwide.
- 1st most common in Saudi males.
- second to lung cancer as a cause of cancer death
- 21,500 new cases, 8900 will die (2008)
- risk of CRC women 1/16 , men 1/14
- peek incidence in 7th decade but it can occur at any age

Etiology of Colorectal Cancer



Risk Factors

- 1. Genetics, Family history
 - Personal history
 - One first degree family member doubles risk
 - Hereditary colorectal cancer syndomes
- 2. Polyps
- 3. Inflammatory bowel disease
- 4. Other
 - Diet, nutrients, smoking, ETOH

Colorectal Cancer Risk Based on Family History

•	General population		6%
•	One 1st degree CRC		2-3X* (12-18%)
•	Two 1st degree CRC		3-4X*
•	One 1st degree CRC < 50 y		3-4*
•	One 2nd or 3rd CRC		1.5X
•	2 2nd degree CRC	2-3X*	
•	1 first degree with polyp	2X*	

Clinical presentation

- 1. Bleeding gross, occult, anemia (37%)
- 2. Change in bowel habit pain, diarrhea, constipation, alternating pattern
- 3. Obstruction more common with left sided lesions most common cause of bowel obstruction in the elderly
- 4. Vague abdominal pains
- 5. Change in caliber of the stools
- 6. Weight loss
- 7. Abdominal mass
- 8. Asymptomatic

Investigations

- General:
 - Complete history and physical (DRE)
- Endoscopic (identify primary, synchronous lesions)
 - Flexible sigmoidoscopy
 - Colonoscopy
- Staging
 - Endorectal ultrasound (rectal cancer)
 - Chest x-ray (metastases)
 - Liver ultrasound (metastases)
 - Abdominal CT scan (metastases)
- Bloodwork
 - CBC electrolytes, CEA (tumour marker)



Surgical therapy

- Surgery is the most important variable in the treatment of colorectal cancer
- Radiation and chemotherapy alone cannot cure any stage of colorectal cancer
- The site of tumour dictates the basic procedure



Preoperative preparation

- Evaluation of medical problems
- Mechanical bowel preparation
 - Colyte , Oral fleet
- IV antibiotics
- DVT prevention (blood clots in the legs)
 - Heparin shots
 - Compression stockings
- Foley catheter
- Epidural catheter for pain

Principles of Surgery

- Examine the entire abdomen
- Remove the appropriate segment of the colon with adequate margins
- Remove the corresponding lymph nodes
- Open vs laparoscopic approach





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Ostomy

• The intestine is brought out through a hole in the abdominal wall

Colostomy (colon on the skin)

- Permanent when the rectum is removed
- Temporary when it is unsafe to make a join

lleostomy (ileum on the skin)

Temporary when the join needs time to heal









Recovery

- Surgery 2 to 4 hours
- Hospital stay 4 to 10 days
 - IV, urine catheter, compression stockings, intravenous pain killers, blood thinner
 - Discharge when ambulating, eating, bowel function, good pain control

• Recovery 4 weeks

Follow up

- Office visit every 3 months for two years then every 6 months for 3 years
- Regular blood work (CEA)
- Colonoscopy at year 1 and 4 and every 5 years
- CT scan yearly

Pathology of Colorectal Cancer

- Macroscopic:
- Microscopic (differentiation):
 - Well
 - Moderately
 - Poorly
- Lymph node involvement
Staging (Where is it Growing?)

1. How far into the wall has it grown? T stage

- Tis invasion of mucosa only
- T1 Invasion of submucosa
- T2 Invasion of muscularis propria
- T3 Full thickness/perirectal fat
- T4 Invasion into adjacent organs

Staging (Where is it Growing?)

- 2. Is it growing in other places? N stage, M stage
- N1 1-3 lymph nodes
- N2 >4 lymph nodes
- N3 distant lymph nodes
- M1 Distant organ (liver, lung)

TNM Staging

- Stage 0 Tis tumors
- Stage 1 T1 and T2 tumors
- Stage 2 T3 and T4 tumors
- Stage 3 Any lymph node involvement
- Stage 4 Distant metastases

Who Gets Additional Treatment?

- COLON
 - All stage 3 patients (positive nodes) chemotherapy
 - ?High risk stage 2 patients
- RECTUM
 - All stage 2 and stage 3 patients should get radiation and chemo

Survival and TNM Stage

 <u>STAGE</u> <u>5-Year Survival</u> 1 90%
 2 80%[^]
 3 27-69%*
 4 8%

^for T3N0 tumors
*depends on # of nodes involved

Summary

- 1. Common Cancer
- 2. Can be prevented through screening and resection of polyps
- 3. Surgery is the primary treatment
- 4. Slow but steady improvement in survival



"Mr. Osborne, may I be excused? My brain is full."