Diverticulosis, diverticulitis, Ischemic colitis. Irritable bowel syndrome

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Case Report

- The 55-year-old female patient (referred by a family doctor)
- History: serious illness is not known. Constipation since childhood.
- Complaints: left upper quadrant abdominal pain, bloody stool, nausea, dysuria
- Temperature: 38.3
- LAB:
  - WBC: 14
  - CRP: 15
  - Urine: normal

**UH:** thickened sigmoid colon
**Dg:** Diverticulitis
Colon cancer
Haemorrhoids, Colitis
Diverticulosis, diverticulitis

Diverticula are small, bulging pouches that can form in large bowel. They are found most often in the lower part of the large intestine (colon). Diverticula are common, especially after age 40, and seldom cause problems.

Sometimes, however, one or more of the pouches become inflamed or infected. That condition is known as diverticulitis.
Epidemiology

The incidence of diverticular disease has increased over the past century. Autopsy studies from the early part of the 20th century reported colonic diverticula rates of 2% to 10%. This has increased dramatically over the years. More recent data suggest that up to 50% of individuals older than 60 years of age have colonic diverticula, with 10% to 25% developing complications such as diverticulitis. Hospitalizations for diverticular disease have also been on the rise. According to an American study evaluating hospitalization rates between 1998 and 2005, rates of admission for diverticular disease increased by 26% during the eight-year study period. Similar trends have been observed in Canadian and European data over the same time period.

Patogenesis

Diverticula are small mucosal herniations protruding through the intestinal layers and the smooth muscle along the natural openings created by the vasa recta or nutrient vessels in the wall of the colon. True diverticulae contain all layers of the gastrointestinal wall (mucosa, muscularis propria, and adventitia) (eg, Meckel diverticulum). False diverticulae, or pseudo-diverticulae do not contain the muscular layers or adventitia, and they only involve the submucosa and mucosa. The sigmoid colon has the highest intraluminal pressures and is most commonly affected.
Patogenesis

• The reason for diverticula:
  
  • The pathophysiology of diverticular disease is complex and relates to abnormal colonic motility, changes in the colonic wall, chronic mucosal low-grade inflammation, imbalance in colonic microflora and visceral hypersensitivity. Moreover, there can be genetic factors involved in the development of colonic diverticula.
  
  • The weakening of the intestinal wall: (Ehlers-Danlos sy, Marfan sy, scleroderma)
  
  • Increased pressure of the bowel wall (manometry demonstrated).
  
  • Low fiber meal + lack of physical activity is crucial to the role.

• Whiteway J, Morson DC. Elastosis in diverticular disease of the colon. Gut
Clinical grounds

Complicated diverticulitis: Up to 25% of patients with acute diverticulitis develop complicated disease. This includes abscess formation, fistulas, strictures obstruction and perforation. Abscess occurs with the perforation of a diverticulum that is usually contained. Small abscesses (smaller than 3 cm) can often be treated with antibiotics alone. Larger abscesses (larger than 4 cm) may require computed tomography-guided percutaneous drainage followed by eventual surgery after resolution of the abscess. Perforating diverticular disease may also lead to fistula, with the most common locations being colovesicular and colovaginal. Fistula complications require surgical management.

Uncomplicated diverticulitis:
Diverticulitis without any significant complications accounts for more than 75% of cases. These patients typically present with left lower quadrant pain, fever and leukocytosis.

Serologic markers predict histological damage in acute diverticulitis J Clin Gastroenterol 2010: 44(10): 702-6 02 December 2010
The signs and symptoms of diverticulitis

- Pain is usually felt in the lower left side of the abdomen: 93–100%.
- Fever: > 50%.
- Leukocytosis: 75%.
- N/V, diarrhea, dysuria, haematochezia.
- Physical examination: local tenderness, or tactile resistance, peritoneal signs of excitement can be experienced.
- Rectal examination: tenderness, palpable resistance when the existence of an abscess.
- Differential diagnosis: IBS, CRC, IBD, intestinal obstruction, appendicitis.
- Gynaecological disorders - ovarian cyst rupture, ovarian torsion, ectopic pregnancy, pelvic inflammatory disease.
- Urological diseases.
- The elderly population: ischemic colitis.
Diagnosis

- The diagnosis of acute diverticulitis can usually be made on the basis of history and physical examination. Laboratory tests may be of help when the diagnosis is in question. A hemogram may reveal leukocytosis and a left shift, indicating infection. However, the absence of leukocytosis does not rule out diverticulitis, as 20-40% of patients have a normal white blood cell count. Liver tests and lipase, urine test, pregnancy test may help to exclude other causes of abdominal pain.

- Endoscopy is not recommended in the acute setting given the risk of worsening diverticulitis and bowel perforation. After the diverticulitis has subsided, colonoscopy can be used to evaluate the extent of diverticulosis or to rule out a malignancy masquerading as a benign postinflammatory stricture.

- Contrast enema is not the imaging modality of choice during an acute episode of abdominal pain and should only be considered in mild-to-moderate uncomplicated cases of diverticulitis when the diagnosis is in doubt.

- The diagnosis of diverticulitis can be made on clinical grounds, but a computed tomography (CT) scan of the abdomen is considered the best imaging method to confirm the diagnosis. The American College of Radiology (ACR) 2008 Appropriateness Criteria for left lower quadrant pain support this recommendation because of the specificity and sensitivity of CT scans, which allow for the diagnosis of causes of left lower quadrant pain that resembles diverticulitis.
Diagnosis
Medical treatments of diverticulosis

- **Uncomplicated (no symptoms):** It does not require treatment.
- **Recommend:** high fiber diet (cellulóz, hemicellulóz, pektinek). Avoid small seed food: (strawberry, raspberry, pappy seed)
- + it should be emphasized ample fluid intake as well.
- **Uncomplicated (with symptoms)** The use of non-absorbable antibiotics (**Rifamixin**) is the mainstay of therapy in patients with mild to moderate symptoms,


Medical treatment of diverticulosis

• Uncomplicated diverticulitis: (most patients (94 percent) can be treated on an outpatient) The decision to hospitalize a patient with uncomplicated diverticulitis depends on several factors, including the patient's ability to tolerate oral intake, severity of illness, comorbidities, and outpatient support systems.

• Recommended: liquid, fiber-free diet + antibiotics

• Elective resection of the involved bowel segment after 2-3 episodes of uncomplicated diverticulitis to prevent further attacks is generally recommended by consensus guidelines.

• In case of acute diverticulitis (immunocompromised patients) much greater possibility of complications, so after the first diverticulitis is recommended for elective surgery.


Uncomplicated diverticulitis

- Trimethoprim/sulfamethoxazole: DS, 160/800 mg orally every 12 hours
- Ciprofloxacin (Cipro), 750 mg orally every 12 hours,
- Levofloxacin (Levaquin), 750 mg orally every 24 hours,
  + Metronidazole (Flagyl), 500 mg orally every six hours
- Amoxicillin/clavulanate extended release (Augmentin XR), two 1,000/62.5-mg tablets orally every 12 hours
- Moxifloxacin (Avelox), 400 mg orally every 24 hours
Diverticulitis

Hospitalization should be considered if patients have signs of peritonitis or there is suspicion of complicated diverticulitis. Inpatient management includes no food or drink by mouth, intravenous fluid resuscitation (normal saline or lactated Ringer solution), and intravenous antibiotics. Clinical improvement is expected within two to four days and includes decreasing fever, leukocytosis, and pain. Fifteen to 30 percent of patients admitted with acute diverticulitis require surgical intervention during that admission.

Patients with a localized abscess

- Patients with a localized abscess may be candidates for CT-guided percutaneous drainage, a procedure that does not increase the risk of recurrent diverticulitis.

- Patients at increased risk of colonic perforation include immunocompromised patients and patients taking nonsteroidal anti-inflammatory drugs, corticosteroids, or chronic opioid analgesics.
Acute diverticulitis

Any of the following:
- High fever
- Leukocytosis
- Complicated disease on CT scan
- Immunosuppression
- Co-morbidities
- No home supports
- Unable to tolerate oral intake

YES

Admit

- Clear fluids or NPO
- IV antibiotics
- Frequent reassessment
- Expect recovery in 2-3 days
- Pain management

Ongoing fevers
- Worsening pain
- Free air on X-ray
- Fistula
- Obstruction

- Surgical consultation
- Repeat CT scan
- Percutaneous drainage of abscess if present

NO

Discharge
- Oral antibiotics
- Initial clear liquid diet
- Close follow up
- Return if not improving

Recovered

- High fiber diet
- Colonoscopy in 4-6 weeks (if 1st episode)

Consider surgical referral for elective resection in selected patients

Diverticular colitis

• Diverticular colitis is the term used to describe a particular pattern of active chronic inflammation in the sigmoid colon affected by diverticular disease.

• The pathogenesis remains uncertain but is almost certainly multifactorial.

• Symptoms and endoscopic findings are diverse.

• Histologically, the disease closely mimicking chronic inflammatory bowel disease, especially ulcerative colitis, but it is not IBD.

• Diverticular colitis may respond well to treatment similar to that used for chronic inflammatory bowel disease.

Ludemann L, Shepherd NA. What is diverticular colitis? Pathology 2002;34(6):568-72
Case report 2

- A 75-year-old woman comes to the office because of a 1-day history and abdominal pain.

Past Hx: not serious illness

Heavy smoker. She has constipation since childhood.

Complaints: suddenly developed umbilical crampy abdominal pain, nausea, haematochezia urgent.

- Fever: 37.9
- LAB:
  - Leukocytosis: 11, CRP: 10
  - LDH: 500, pH: 7.2

UH: normal ? Colono, angio
CT angio.
Dx: ??
ACG Clinical Guideline: Epidemiology, Risk Factors, Patterns of Presentation, Diagnosis, and Management of Colon Ischemia (CI)

DEFINITION

CI is the condition that results when blood flow to the colon is reduced to a level insufficient to maintain cellular metabolic function. The end result of this process is that colonocytes become acidotic, dysfunctional, lose their integrity and, ultimately, die. Although the etymologic root of the word ischemia is from the Greek iskhaimos, meaning a “stopping of the blood,” we now know that blood flow need not stop but only diminish significantly to cause ischemic damage. Moreover, ischemia may be followed by reperfusion injury and, for relatively brief periods of ischemia, this combined injury may produce more damage than just reduction of blood flow without reperfusion. The degree to which colonic blood flow must diminish before ischemia results varies with the acuteness of the event, the degree of preexisting vascular collateralization, and the length of time the low flow state persists.
Epidemiology

• The exact incidence of ischemic colitis is difficult to estimate, as many patients with mild ischemia may not seek medical attention. Ischemic colitis is responsible for about 1 in 2000 hospital admissions, and is seen on about 1 in 100 endoscopies.

• Men and women are affected equally; ischemic colitis is a disease of the elderly, with more than 90% of cases occurring in people over the age of 60.
Ischemic colitis

- Colonic blood supply.
- **Pink** - supply from superior mesenteric artery (SMA) and its branches: middle colic, right colic, ileocolic arteries.
- **Blue** - supply from inferior mesenteric artery (IMA) and its branches: left colic, sigmoid, superior rectal artery.
- 7 is for so-called Cannon-Böhm point (the border between the areas of SMA and IMA supplies), which lies at the splenic flexure.
Ischemic colitis

Classification of splanchnic, or gastrointestinal ischemia. ASS: Acute splanchnic syndrome; CSS: Chronic splanchnic syndrome; CACS, Celiac artery compression syndrome; NOMI: Non-occlusive mesenteric ischemia.

Diagnostic approach

• Clinicians must maintain a high index of suspicion for ischaemic bowel disease, because the signs and symptoms are relatively non-specific yet the condition has significant morbidity and mortality. Early recognition, appropriate diagnostic studies, and aggressive treatment are necessary to improve outcome.

• In the absence of highly specific or definitive signs and symptoms, a history and physical examination alone are generally not sufficient to make the diagnosis; usually some form of imaging is required. However, when fulminant ischaemic bowel disease is present, extensive diagnostic testing may not be appropriate in order that surgical intervention can proceed without delay.
Gross specimen of dead bowel.
<table>
<thead>
<tr>
<th></th>
<th>Acute mesenteric ischaemia</th>
<th>Chronic mesenteric ischaemia</th>
<th>Colonic ischaemia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Site</strong></td>
<td>Periumbilical pain. Focal pain if necrosis present.</td>
<td>Poorly localised.</td>
<td>Lateral abdomen or flanks. Focal pain if necrosis present.</td>
</tr>
<tr>
<td><strong>Onset</strong></td>
<td>Sudden.</td>
<td>Insidious.</td>
<td>Sudden. May become continuous and diffuse if it progresses.</td>
</tr>
<tr>
<td><strong>Character</strong></td>
<td>Sharp or colicky. Pain is out of proportion to the exam.</td>
<td>Repeated, transient episodes of pain, progressing with time.</td>
<td>Dull.</td>
</tr>
<tr>
<td><strong>Radiation</strong></td>
<td>No radiation.</td>
<td>No radiation.</td>
<td>Radiates to back.</td>
</tr>
<tr>
<td><strong>Associations</strong></td>
<td>Nausea, vomiting, diarrhoea. May have sudden forceful bloody bowel evacuation.</td>
<td>Nausea, vomiting.</td>
<td>Nausea, vomiting, diarrhoea. Passage of maroon stools.</td>
</tr>
<tr>
<td><strong>Timing, duration, frequency</strong></td>
<td>2-3 hours (arterial) or 5 to more than 30 days (venous).</td>
<td>Months.</td>
<td>Acute, subacute, or chronic.</td>
</tr>
<tr>
<td><strong>Exacerbating and relieving factors</strong></td>
<td>No association with meals, pain not relieved.</td>
<td>Worse after meals, resolving over hours.</td>
<td>None.</td>
</tr>
<tr>
<td><strong>Severity</strong></td>
<td>Severe.</td>
<td>Mild.</td>
<td>Mild-to-moderate.</td>
</tr>
<tr>
<td><strong>Abdominal examination</strong></td>
<td>Epigastric bruit and distention.</td>
<td></td>
<td>Abdominal distention and no bowel sounds as ischaemia progresses.</td>
</tr>
<tr>
<td><strong>Cardiovascular exam</strong></td>
<td>May have a fibrillation or other arrhythmia, evidence of peripheral vascular disease.</td>
<td>Atherosclerosis, peripheral vascular disease.</td>
<td>May have a fibrillation or other arrhythmia, atherosclerosis, evidence of peripheral vascular disease.</td>
</tr>
<tr>
<td><strong>Laboratory test results</strong></td>
<td>Leukocytosis, metabolic acidemia, and elevated serum amylase.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Imaging</strong></td>
<td>Thumbprinting on plain x-rays. Mesenteric occlusion on angiography. Subdiaphragmatic air if perforated. Pneumatosis intestinales, or air in portal vessels when bowel necrosis present. Use contrast-enhanced CT to diagnose mesenteric venous thrombosis.</td>
<td>Angiography demonstrates severe occlusion of at least 2 of the 3 splanchnic vessels.</td>
<td>Angiography has no role. Barium enema can be used if colonoscopy is not available (cobblestone appearance, thumbprinting, stricture).</td>
</tr>
</tbody>
</table>
Management

• Patients with chronic mesenteric ischemia have traditionally been treated with mesenteric vascular surgical revascularization.

• Overall, the operative mortality remains high (approximately 7.5%–10%).

• There are several surgical revascularization strategies, including visceral endarterectomy, antegrade supraceliac aorta to visceral bypass, and retrograde infrarenal aorta to visceral bypass.

• Before surgical revascularization, the patient may benefit from total parenteral nutrition (TPN).
Management

Angioplasty and stenting

- Angioplasty is technically difficult because of the anatomy of the SMA.
- Mesenteric angioplasty has a good technical success rate but a high rate of restenosis, (restenosis rates are 20-50%) and routine stenting is recommended.

Successful endovascular stenting of superior mesenteric artery stenosis for chronic mesenteric ischemia. (a) Pre-stenting; (b) Post-stenting. (Figure courtesy of Deepak L Bhatt, MD.)
Complete aortic occlusion (Leriche syndrome) with acute embolism of superior mesenteric artery
RISK FACTORS

Summary statements

1. Comorbid cardiovascular disease and diabetes mellitus should increase consideration of CI in patients with typical clinical features (10,14,15,20).
2. A history of irritable bowel syndrome (IBS) and constipation should be sought in patients suspected to have CI (8,13,15).
3. Selective cardiology consultation is justified in patients with CI, particularly if a cardiac source of embolism is suspected (23).
4. Chronic kidney disease and chronic obstructive pulmonary disease are associated with increased mortality from CI (7,10,24,25).
5. Evaluation for thrombophilia should be considered in young patients with CI and in all patients with recurrent CI (26–28).
6. Surgical procedures in which the inferior mesenteric artery (IMA) has been sacrificed, such as abdominal aortic aneurysm repair and other abdominal operations, should increase consideration of CI in patients with typical clinical features (14,29,30).
7. In patients suspected of having CI, a history of medication and drug use should be sought, especially constipation-inducing medications, immunomodulators, and illicit drugs (9,15,31).
### Table 2. Medical conditions and surgical history independently associated with colon ischemia in multivariate analyses of case–control studies

<table>
<thead>
<tr>
<th>Medical conditions</th>
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<tbody>
<tr>
<td>Cardiovascular/pulmonary</td>
</tr>
<tr>
<td>Atherosclerosis</td>
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<tr>
<td>Atrial fibrillation</td>
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<tr>
<td>Chronic obstructive pulmonary disease</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
</tr>
<tr>
<td>Congestive heart failure or ischemic heart disease</td>
</tr>
<tr>
<td>Congestive heart failure</td>
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<tr>
<td>Hypertension†</td>
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<tr>
<td>Hypotension</td>
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<tr>
<td>Ischemic heart disease</td>
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<tr>
<td>Peripheral vascular disease</td>
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<tr>
<td>Shock</td>
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<tr>
<td>Gastrointestinal</td>
</tr>
<tr>
<td>Constipation</td>
</tr>
<tr>
<td>Diarrhea²</td>
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<tr>
<td>Irritable bowel syndrome²</td>
</tr>
<tr>
<td>Miscellaneous</td>
</tr>
<tr>
<td>Deyo–Charlson Comorbidity Index Score</td>
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<tr>
<td>Diabetes</td>
</tr>
</tbody>
</table>

### Table 3. Drugs proposed to predispose to CI, estimate of evidence level, and postulated pathogenesis

<table>
<thead>
<tr>
<th>Moderate evidence</th>
<th>Immunomodulator drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constipation-inducing drugs</td>
<td></td>
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<tr>
<td>Decongestants</td>
<td>Chemotherapeutic drugs</td>
</tr>
<tr>
<td>Diuretics</td>
<td>Illlicit drugs</td>
</tr>
<tr>
<td>Ergot alcohols (often combined with caffeine)</td>
<td></td>
</tr>
<tr>
<td>Hormonal therapies</td>
<td></td>
</tr>
<tr>
<td>Laxatives</td>
<td>Low evidence</td>
</tr>
<tr>
<td>Appetite suppressants</td>
<td></td>
</tr>
<tr>
<td>Antibiotics</td>
<td>Surgical history</td>
</tr>
<tr>
<td>Cardiovascular surgery</td>
<td></td>
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<tr>
<td>Abdominal surgery</td>
<td></td>
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<tr>
<td>Aortic surgery</td>
<td></td>
</tr>
<tr>
<td>Systemic rheumatologic disorders²</td>
<td></td>
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<tr>
<td>Dyslipidemia</td>
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<tr>
<td>Rheumatoid arthritis²</td>
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<tr>
<td>Shock</td>
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</tbody>
</table>

*Am J Gastroenterol 2015; 110:18–44; doi:10.1038/aig.2014.395; published online 23 December 2014*
### Table 1. Recommendations and summary statements

#### Colon Ischemia Recommendations and Best Practice Summary Statements

<table>
<thead>
<tr>
<th>Recommendation and Best Practice Statements</th>
<th></th>
</tr>
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<tbody>
<tr>
<td><strong>Clinical Presentation</strong></td>
<td></td>
</tr>
<tr>
<td>1. The diagnosis of CI is usually established in the presence of symptoms including sudden cramping, mild, abdominal pain; an urgent desire to defecate; and passage within 24 h of bright red or maroon blood or bloody diarrhea. (Strong recommendation, very low level of evidence) (7,9,17)</td>
<td></td>
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<tr>
<td>2. A diagnosis of non-isolated right colon ischemia (non-IRCI) should be considered when patients present with hematochezia. (Strong recommendation, very low level of evidence) (7,9,17)</td>
<td></td>
</tr>
<tr>
<td><strong>Imaging of CI</strong></td>
<td></td>
</tr>
<tr>
<td>1. CT with intravenous and oral contrast should be the first imaging modality of choice for patients with suspected CI to assess the distribution and phase of colitis. (Strong recommendation, moderate level of evidence) (111–113)</td>
<td></td>
</tr>
<tr>
<td>2. The diagnosis of CI can be suggested based on CT findings (e.g., bowel wall thickening, edema, thumbprinting). (Strong recommendation, moderate evidence) (111–113)</td>
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<tr>
<td>3. Multiphasic CTA should be performed on any patient with suspected IRCI or in any patient in whom the possibility of AMI cannot be excluded. (Strong recommendation, moderate level of evidence) (113,114)</td>
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</tr>
<tr>
<td>4. CT or MRI findings of colonic pneumatosis and porto-mesenteric venous gas can be used to predict the presence of transmural colonic infarction. (Strong recommendation, moderate level of evidence) (115)</td>
<td></td>
</tr>
<tr>
<td>5. In a patient in whom the presentation of CI may be a heralding sign of AMI (e.g., IRCI, severe pain without bleeding, atrial fibrillation), and the multiphasic CT is negative for vascular occlusive disease, traditional splanchnic angiography should be considered for further assessment. (Conditional recommendation, low level of evidence) (114)</td>
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</tbody>
</table>
### Colonoscopy in the Diagnosis of CI

1. Early colonoscopy (within 48h of presentation) should be performed in suspected CI to confirm the diagnosis. (Strong recommendation, low level of evidence) (17)

2. When performing colonoscopy on a patient with suspected CI, the colon should be insufflated minimally. (Conditional recommendation, very low level of evidence) (69,135)

3. In patients with severe CI, CT should be used to evaluate the distribution of disease. Limited colonoscopy is appropriate to confirm the nature of the CT abnormality. Colonoscopy should be halted at the distalmost extent of the disease. (Strong recommendation, low level of evidence)

4. Biopsies of the colonic mucosa should be obtained except in cases of gangrene. (Strong recommendation, very low level of evidence)

5. Colonoscopy should not be performed in patients who have signs of acute peritonitis or evidence of irreversible ischemic damage (i.e., gangrene and pneumatisos). (Strong recommendation, very low level of evidence)

### Severity and Treatment of CI

1. Most cases of CI resolve spontaneously and do not require specific therapy. (Strong recommendation, low quality of evidence) (107,108,139)

2. Surgical intervention should be considered in the presence of CI accompanied by hypotension, tachycardia, and abdominal pain without rectal bleeding; for IRCI and pan-colonic CI; and in the presence of gangrene. (Strong recommendation, moderate level of evidence) (17,107,108)

3. Antimicrobial therapy should be considered for patients with moderate or severe disease. (Strong recommendation, very low level of evidence) (107,108,140)
Colon Ischemia Recommendations and Best Practice Summary Statements

**Laboratory Tests in CI**

1. Laboratory testing should be considered to help predict CI severity (17,94,107)
2. Decreased hemoglobin levels, low serum albumin, and the presence of metabolic acidosis can be used to predict severity of CI (141,142)

**Severity and Treatment of CI**

1. When considering mortality risk for patients undergoing surgical intervention for acute CI, the Ischemic Colitis Mortality Risk (ICMR) factors should be utilized (141,142)

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**Table 5. Recommended Initial serology and stool studies for suspected colon Ischemia (CI)**

<table>
<thead>
<tr>
<th>Blood tests</th>
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<tbody>
<tr>
<td>Albumin</td>
<td></td>
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<tr>
<td>Amylase</td>
<td></td>
</tr>
<tr>
<td>Complete blood count</td>
<td></td>
</tr>
<tr>
<td>Comprehensive electrolyte panel</td>
<td></td>
</tr>
<tr>
<td>Creatine kinase (CK)</td>
<td></td>
</tr>
<tr>
<td>Lactate</td>
<td></td>
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<tr>
<td>Lactate dehydrogenase (LDH)</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Stool tests</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Clostridium difficile toxin assay</td>
<td></td>
</tr>
<tr>
<td>Culture</td>
<td></td>
</tr>
<tr>
<td>Ova and parasite</td>
<td></td>
</tr>
</tbody>
</table>

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**IMAGING OF CI**

**Recommendations**

1. CT with intravenous and oral contrast should be ordered as the imaging modality of choice for patients with suspected CI, to assess the distribution and phase of colitis (strong recommendation, moderate level of evidence) (111–113).
2. The diagnosis of CI can be suggested based on CT findings (e.g., bowel wall thickening, edema, and thumbprinting) (strong recommendation, moderate evidence) (111–113).
3. Multiphasic CT angiography (CTA) should be performed on any patient with suspected IRCI or in any patient in whom the possibility of AMI cannot be excluded (strong recommendation, moderate level of evidence) (113,114).
4. CT or magnetic resonance imaging (MRI) findings of colonic pneumatosis and portomesenteric venous gas can be used to predict the presence of transmural colonic infarction (strong recommendation, moderate level of evidence) (115).
5. In a patient in whom the presentation of CI may be a heralding sign of acute mesenteric ischemia (AMI; e.g., IRCI, severe pain without bleeding, and atrial fibrillation), and the multiphasic CT is negative for vascular occlusive disease, traditional splanchnic angiography should be considered for further assessment (conditional recommendation, low level of evidence) (114).
<table>
<thead>
<tr>
<th>Disease severity</th>
<th>Criteria</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| Mild             | Typical symptoms of CI with a segmental colitis not isolated to the right colon and with none of the commonly associated risk factors for poorer outcome that are seen in moderate disease | Observation  
Supportive care |
| Moderate         | Any patient with CI and up to three of the following factors:            | Correction of cardiovascular abnormalities (e.g., volume replacement)  
Broad-spectrum antibiotic therapy  
Surgical consultation |
|                  |   - Male gender                                                          |                                                                           |
|                  |   - Hypotension (systolic blood pressure < 90 mm Hg)                     |                                                                           |
|                  |   - Tachycardia (heart rate > 100 beats/min)                             |                                                                           |
|                  |   - Abdominal pain without rectal bleeding                               |                                                                           |
|                  |   - BUN > 20 mg/dl                                                       |                                                                           |
|                  |   - Hgb > 12 g/dl                                                        |                                                                           |
|                  |   - LDH > 350 IU                                                         |                                                                           |
|                  |   - Serum sodium < 136 mEq/l (mmol/l)                                   |                                                                           |
|                  |   - WBC > 15 cells/cmm (x 10^9/l)                                       |                                                                           |
|                  |   - Colonic mucosal ulceration identified colonoscopically               |                                                                           |
| Severe           | Any patient with CI and more than three of the criteria for moderate disease or any of the following: | Emergent surgical consultation (treatment is likely to be surgical)  
Transfer to intensive care unit  
Correction of cardiovascular abnormalities (e.g., volume replacement)  
Broad-spectrum antibiotic therapy |
|                  |   - Peritoneal signs on physical examination                            |                                                                           |
|                  |   - Pneumatosis or portal venous gas on radiologic imaging              |                                                                           |
|                  |   - Gangrene on colonoscopic examination                                |                                                                           |
|                  |   - Pancolonic distribution or IRCI on imaging or colonoscopy           |                                                                           |

BUN, blood urea nitrogen; CI, colon ischemia; Hgb, hemoglobin; IRCI, isolated right-colon ischemia; LDH, lactate dehydrogenase; WBC, white blood cell count.
Prognosis

• Most patients with ischemic colitis recover fully, although the prognosis depends on the severity of the ischemia. Patients with pre-existing peripheral vascular disease or ischemia of the ascending (right) colon may be at increased risk for complications or death.

• Non-gangrenous ischemic colitis, which comprises the vast majority of cases, is associated with a mortality rate of approximately 6%. However, the minority of patients who develop gangrene as a result of colonic ischemia have a mortality rate of 50-75% with surgical treatment; the mortality rate is almost 100% without surgical intervention.
Case Report 3

- 35-year-old female patient,
- History: illness was not serious. (Smokers), Constipation and abdominal pain often since childhood.
- Complaints: sudden umbilical crampy abdominal pain (after eating), nausea. She will be better after a bowel movement.
- Fever: 36.3
- LAB:
  - Leukocytosis: 8 , CRP: 3
  - LDH: 15, pH: 7.4

UH: normal, colono: neg, Gynecologist: neg, Stool culture: neg
Dx: ??
Irritable bowel syndrome (IBS)
Irritable bowel syndrome
(Classification)

• IBS is a chronic functional disorder of the gastrointestinal system. Patients experience abdominal pain and altered bowel habit, with either predominantly:
  • Symptoms:
  • (IBS-D) diarrhea Mild
  • (IBS-C) constipation Moderate
  • (IBS-M) or both Serious

• There is no definitive investigation as no biomarker has been found, so IBS is diagnosed clinically.

• Monoclonal Anti-Vinculin antibody A Serologic Test for Irritable Bowel Syndrome??
Epidemiology

• IBS disease of primarily young women. (Female / male = 2/1, 4/1)
• The visit to the doctor on female predominance of the disease the real one is even more significant - and often easier for women to seek medical advice, (female / male ratio = 2/1).
• Studies show that women are 20% of men at some point in life has 12% of the symptoms caused by irritable syndrome.
Worldwide prevalence of irritable bowel syndrome, as reported by country
Irritable bowel syndrome frequency in different countries
The causes of IBS
The causes of IBS

• While the causes of IBS are still unknown, it is believed that the entire gut–brain axis is affected.

• Post-infectious: The risk of developing IBS increases six-fold after acute gastrointestinal infection. (IBS-PI) „postinfectious IBS„, (In some individuals, IBS may have an acute onset and develop after an infectious illness characterized by two or more of: fever, vomiting, diarrhea, or positive stool culture.)

• Stress: childhood physical and psychological abuse is often associated with the development of IBS

• Bacteria, Fungus, Protozoa Small intestinal bacterial overgrowth occurs with greater frequency in patients who have been diagnosed with IBS compared to healthy controls. (SIBO)

• Genetic, environmental, and psychological factors seem to be important in the development of IBS.
Measurement of anti-vinculin antibodies in serum demonstrated higher levels of these autoantibodies in IBS patients, compared with those without the disorder, with positive predictive values between 90% and 100% depending on the cutoff values used, reported Mark Pimentel, MD, of Cedars-Sinai Medical Center in Los Angeles, and colleagues.

**IBS, possibly of the post-infectious type**

Vinculin is a cell migration and adherence protein found predominantly on nerves and epithelium. A series of studies leading up to this result suggests that acute gastroenteritis causes antibodies to be formed to vinculin and is associated with IBS development.

**higher levels of plasma IL-6**

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**Monoclonal Anti-Vinculin antibody A Serologic Test for Irritable Bowel Syndrome**
Could IBS be an autoimmune condition?

One cause of IBS is having a previous gut infection which often triggers small intestinal bacterial overgrowth (SIBO). The idea behind this study is that a bacterial toxin called cytolethal distending toxin B (CdtB) produced by gram negative Proteobacteria (such as Shigella sp. and E. coli), triggers gastroenteritis by molecular mimicry which stimulates auto-antibodies to vinculin. This process may be involved eventual development of IBS, though the details of how this happens are far from clear. Studying antibody titers to vinculin in IBS patients can establish the role of gastroenteritis in IBS and help determine the extent to which autoimmune processes are involved.

It is believed that the entire gut–brain axis is affected
Altered sensation

Abnormal CNS motor control

Abnormal GI smooth muscle activity

Abnormal GI mechanoreceptor sensitivity

Abnormal CNS sensory processing
Altered motility
The pressure conditions of the colon in healthy humans/ IBS patients
The Gastrocolic Reflex & Irritable Bowel Syndrome

Contractions of Sigmoid Colon After a Meal (Normal Human)

Contractions of Sigmoid Colon After a Meal (Spastic Colon Syndrome)
Diagnosis of IBS: Rome III (2006)

Irritable bowel syndrome has been a diagnosis of exclusion, to help this process, researchers have developed diagnostic criteria for IBS.

(Rome diagnostic criteria):

At least 3 days per month in past 12 weeks of continuous or recurrent abdominal pain or discomfort.

With at least 2 of the following:

- Relief with defecation
- Altered stool frequency
- Altered stool form

Onset of symptoms more than 6 months before diagnosis.
Diagnosis of IBS

• (Rome diagnostic criteria)

• **Except**

• Some red flag signs and symptoms that suggest need for additional tests:

  • **New onset, after age 50, weight loss, rectal bleeding, fever, nausea or recurrent vomiting, abdominal pain, especially if it's not completely relieved by a bowel movement, or occurs at night, diarrhea that is persistent or awakens you from sleep, anemia related to low iron.**
Differential diagnosis

DIFFERENTIAL DIAGNOSIS OF CHRONIC/RECURRENT BOWEL DYSFUNCTION

1. Irritable bowel syndrome
2. Lactase deficiency
3. Drugs
   a. Laxative cathartics
   b. Magnesium containing antacids
4. Bacterial infection
   a. Salmonella species
   b. Campylobacter jejuni
   c. Yersinia enterocolitica
   d. Clostridium difficile
5. Parasitic infection
   a. Giardia Lamblia
   b. Entameba histolytica
6. Inflammatory bowel disease
   a. Crohn’s disease
   b. Ulcerative colitis
   c. Microscopic (e.g., collagenous
7. Malabsorption
   a. Chronic pancreatitis
   b. Celiac sprue
   c. Post-gastrectomy syndromes
8. Metabolic disorders
   a. Diabetes mellitus
   b. Thyrotoxicosis
9. Endocrine-producing tumors
   a. Gastrinoma
   b. Carcinoid
   c. VIPoma
10. Psychiatric disorders
    a. Depression/Panic Disorder
    b. Somatization disorders
11. Intestinal pseudoobstruction
    a. Primary Visceral Myopathy, Neuropathy
    b. Secondary myopathy/neuropathy (e.g., scleroderma, diabetes)
12. Other colonic diseases
    a. Mast-cell disease
    b. Villous Adenoma
13. Opportunistic infections in immunocompromised host
Treatments and drugs
Because it's not clear what causes irritable bowel syndrome, treatment focuses on the relief of symptoms. *(Symptom-oriented treatment)*

In most cases, you can successfully control mild signs and symptoms of irritable bowel syndrome by learning to manage stress and making changes in your diet and lifestyle. Try to avoid foods that trigger your symptoms. Also try to get enough exercise, drink plenty of fluids and get enough sleep.
Diet

- **Dietary changes:**
  - Eliminating high-gas foods: especially cabbage, broccoli and cauliflower — and raw fruits.
  - Eliminating gluten:
  - Eliminating FODMAPs: (fermentable oligo-, di-, and monosaccharides and polyols). FODMAPs are found in certain grains, vegetables, fruits and dairy products.
Symptom-oriented treatment
<table>
<thead>
<tr>
<th>Pharmacologic Agent</th>
<th>Usual Adult Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diarrhea-Predominant IBS</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Opioid µ-Receptor Agonists</strong></td>
<td></td>
</tr>
<tr>
<td>Loperamide (Imodium)</td>
<td>2-4 mg, up to qid prn</td>
</tr>
<tr>
<td>Diphenoxylate (Lomotil)</td>
<td>5 mg qid prn</td>
</tr>
<tr>
<td><strong>Smooth-Muscle Relaxants</strong></td>
<td></td>
</tr>
<tr>
<td>Dicyclomine (Bentyl)</td>
<td>20 mg qid initially, then up to 40 mg qid</td>
</tr>
<tr>
<td>Hyoscyamine (Levsin, NuLev, Levbid)</td>
<td>0.125 mg sublingually tid prn or 0.375 mg bid PO</td>
</tr>
<tr>
<td><strong>Tricyclic Antidepressants</strong></td>
<td></td>
</tr>
<tr>
<td>Amitriptyline (Elavil, Endep)</td>
<td>10-25 mg bid or 25-50 mg qhs</td>
</tr>
<tr>
<td>Desipramine (Norpramin)</td>
<td>50 mg tid</td>
</tr>
<tr>
<td><strong>Selective 5-HT₃ Receptor Antagonist</strong></td>
<td></td>
</tr>
<tr>
<td>Alosetron (Lotronex)</td>
<td>1 mg qd × 4 wk, may increase to 1 mg bid × 4 wk*</td>
</tr>
<tr>
<td><strong>Constipation-Predominant IBS</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Bulking Agents</strong></td>
<td></td>
</tr>
<tr>
<td>Psyllium (Metamucil)</td>
<td>20 g/day, divided, with &gt;250 mL water</td>
</tr>
<tr>
<td>Polycarbophil (Konsyl Fiber)</td>
<td>1-6 g/day, divided, with &gt;250 mL water</td>
</tr>
<tr>
<td>Methylcellulose (Citrucel)</td>
<td>3-6 g/day, divided, with &gt;250 mL water</td>
</tr>
<tr>
<td><strong>Osmotic Laxatives</strong></td>
<td></td>
</tr>
<tr>
<td>Polyethylene glycol (MiraLax)</td>
<td>1 dose (17 g in glass of water) qd or bid</td>
</tr>
<tr>
<td>Lactulose (Kristalose, Cephulac, Chronulac, Constulose, Duphalac, Enulose, R O Lactulose)</td>
<td>15-60 mL/day, divided</td>
</tr>
<tr>
<td>Sorbitol (Ora-Sweet)</td>
<td>120 mL of 25% solution</td>
</tr>
<tr>
<td>Pharmacologic Agent</td>
<td>Usual Adult Dosage</td>
</tr>
<tr>
<td>--------------------------------------</td>
<td>--------------------------------------------------------</td>
</tr>
<tr>
<td>Pain</td>
<td></td>
</tr>
<tr>
<td><strong>Smooth-muscle relaxants</strong></td>
<td></td>
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<td>50 mg tid</td>
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Treatment of IBS

Psychological factors
• You have to explain that the complaint does not depend on the quality of food consumed, but also causes the meal itself.

• The symptoms due to non-normal gastrocolic reflex occurred.
The hypnotic effect of the colon pressure
Thank you for your attention!