Supportive module 2 "Basics of diagnosis, treatment and prevention of major gastroenterological diseases"

<table>
<thead>
<tr>
<th></th>
<th>Chronic disease of the small intestine</th>
<th></th>
<th>15/11</th>
</tr>
</thead>
<tbody>
<tr>
<td>11.</td>
<td>Chronic disease of the colon: IBS and nonspecific colitis</td>
<td>2</td>
<td>22/11</td>
</tr>
<tr>
<td>12.</td>
<td>Chronic pancreatitis</td>
<td>2</td>
<td>29/11</td>
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<tr>
<td>13.</td>
<td>Chronic hepatitis</td>
<td>2</td>
<td>06/12</td>
</tr>
<tr>
<td>14.</td>
<td>Cirrhosis of the liver</td>
<td>2</td>
<td>13/12</td>
</tr>
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</table>
Supportive module 2: Basics of diagnosis, treatment and prevention of major gastroenterological diseases

Chronic Disease of the Small Intestine: Crohn's disease, Celiac disease

LECTURE IN INTERNAL MEDICINE FOR IV COURSE STUDENTS

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V.N. Karazin National University Medical School’ Internal Medicine Dept.
Plan of the Lecture

- Definition
- Epidemiology
- Mechanisms
- Classification
- Clinical presentation
- Diagnosis
- Treatment
- Prognosis
- Prophylaxis
- Abbreviations
- Diagnostic guidelines

Inflammatory infiltrate in the mesentery
Definition
Crohn's disease, Celiac disease

• Crohn’s disease encompasses a multisystem group of autoimmune inflammatory disorders with specific clinical and pathological features characterized by focal, asymmetric, transmural, and, occasionally, granulomatous inflammation primarily affecting the gastrointestinal tract.

• Celiac disease (gluten-sensitive enteropathy) is an autoimmune inflammatory disorder of the small intestine that is precipitated by the ingestion of gluten, a component of wheat protein, in genetically susceptible persons and it is the result of the interaction between genetic and environmental factors.

Epidemiology
Crohn's disease, Celiac disease

- Crohn’s disease with potential for systemic and extraintestinal complications can affect any age group, but the onset (diagnosis) is most common in the second and third decades; the incidence and prevalence in developed counties estimated to be 5/100,000 and 50/100,000, respectively.

- The prevalence of celiac disease is approximately one case per 250 persons.
Epidemiology
Geographic Distribution of Crohn's disease
Epidemiology
Aged distribution of Crohn's disease

[Graph showing incidence of Crohn's disease by age and gender]
Epidemiology
The Celiac Disease Iceberg

Symptomatic CD

Mucosal damage

Silent CD

Latent CD

Serologic markers positive
Genetic predisposition (DQ2 or DQ8)

Normal mucosa
Risk Factors & Etiology
Crohn's disease, Celiac disease

• The causes of Crohn’s disease are not known, and a malfunctioning immune system, genetics, and environment may all play a part; risk factors may include (young) age, ethnicity (whites and Jews), family history (1 in 5 people with Crohn's disease has a family member with the disease), cigarette smoking, nonsteroidal anti-inflammatory medications, life in an urban area or in an industrialized country;

• The cause for celiac disease is unknown, and people of European descent and those with other autoimmune disorders are at increased risk for its developing; among genetic risk factors, the strongest association is with the HLA class II DQ region; nevertheless at least 39 non-HLA loci are associated with disease; gluten is the main trigger of the disease; a role for infectious agents and microbiota composition in disease development has also been proposed.
Mechanism
Crohn's disease

Key Players

• Genetics (Crohn’s disease is genetically linked to celiac disease)
• Environmental factors (the increased incidence of Crohn's in the industrialized world indicates an environmental component)
• Immunobiology:
  • Microbiota (a causal role for Mycobacterium avium subspecies paratuberculosis)
  • Intestinal barrier
  • Microbial sensing, innate immunity, and autophagy
  • Adaptive immunity (Crohn's disease is a primary T cell autoimmune disorder and results from an impaired innate immunity) and leucocyte migration.
Mechanism
Crohn's disease 2

- Chronic inflammation from T-cell activation leading to tissue injury is implicated in the pathogenesis of Crohn disease
- The initial lesion starts as a focal inflammatory infiltrate around the crypts, followed by ulceration of superficial mucosa
- Inflammatory cells invade the deep mucosal layers and, in that process, begin to organize into noncaseating granulomas
- The granulomas extend through all layers of the intestinal wall and into the mesentery and the regional lymph nodes
- Neutrophil infiltration into the crypts forms crypt abscesses, leading to destruction of the crypt and atrophy of the colon
- Transmural inflammation results in thickening of the bowel wall and narrowing of the lumen
Mechanism
Crohn's disease 3

- Ulcerations are common and are often seen on a background of normal mucosa
- As disease progresses, it is complicated by obstruction or deep ulceration leading to fistulization by way of the sinus tracts penetrating the serosa, microperforation, abscess formation, and malabsorption
- Obstruction is intermittent and can often be reversed by means of conservative measures and anti-inflammatory agents but with further disease progression becomes chronic because of fibrotic scarring, luminal narrowing, and stricture formation
- Serosal inflammation causes adhesions; thus, free perforations are less common in Crohn disease than in other inflammatory bowel conditions.
Mechanism
Factors contributing to Crohn's disease

[Diagram showing molecular mechanisms and factors contributing to Crohn's disease]

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4447044/figure/fig001/
Mechanism
Celiac disease 1

- Celiac disease results from genetic abnormal immune response to gluten that leads to local activation of immune system.
- As a result of immunological reactions with significant role of T lymphocytes (non proliferative activation of lamina propria CD4+ lymphocytes and proliferative activation of intra-epithelial TcR alpha/beta CD8+ and TcR gamma/delta lymphocytes) the inflammatory process with typical histopathological lesions develops.
- In immunological reaction to gluten besides T lymphocytes other cells are involved (lymphocytes B, natural killer cells (NK), neutrophils, eosinophils, macrophages, mastocytes).
- This intense local inflammatory reaction produces the villous flattening characteristic of gluten-sensitive enteropathy.

Mechanism
Celiac disease 2

- Malabsorption of micronutrients (e.g., vitamins and minerals) and macronutrients (e.g., protein, carbohydrate, fat) follows
- Small-bowel involvement is most prominent proximally and may be “patchy,” especially in patients with “silent” celiac disease (i.e., minimal or no symptoms) and those with dermatitis herpetiformis
- About 95% of patients with celiac disease exhibit specific Human Leukocyte Antigen (HLA) class II alleles DQA1*0501 and DQB1*0201
- Patients with type 1 diabetes, autoimmune thyroid disease, Sjögren's syndrome, primary biliary cirrhosis, Addison's disease, systemic lupus erythematosus, selective IgA deficiency, and alopecia areata may also exhibit similar genotypes and are at risk for gluten-sensitive enteropathy.

Mechanism
Genetic Pathways of Celiac disease

Mechanisms
1. Innate immune detection
2. T-cell development
3. T-cell and B-cell co-stimulation
4. Cytokines, chemokines and their receptors

Intestinal inflammation
XI Diseases of the digestive
K50-K52 Noninfective enteritis and colitis
K50.0 Crohn disease of small intestine
K90-K93 Other diseases of the digestive system
K90.0 Celiac disease
## Classification

Vienna and Montreal classification for Crohn's disease

<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td><strong>Age at diagnosis</strong></td>
<td>A1 below 40 y</td>
<td>A1 below 16 y</td>
</tr>
<tr>
<td></td>
<td>A2 above 40 y</td>
<td>A2 between 17 and 40 y</td>
</tr>
<tr>
<td></td>
<td>A3 above 40 y</td>
<td>A3 above 40 y</td>
</tr>
<tr>
<td><strong>Location</strong></td>
<td>L1 ileal</td>
<td>L1 ileal</td>
</tr>
<tr>
<td></td>
<td>L2 colonic</td>
<td>L2 colonic</td>
</tr>
<tr>
<td></td>
<td>L3 ileocolonic</td>
<td>L3 ileocolonic</td>
</tr>
<tr>
<td></td>
<td>L4 upper</td>
<td>L4 isolated upper disease</td>
</tr>
<tr>
<td><strong>Behaviour</strong></td>
<td>B1 non-stricturing, non-penetrating</td>
<td>B1 non-stricturing, non-penetrating</td>
</tr>
<tr>
<td></td>
<td>B2 stricturing</td>
<td>B2 structuring</td>
</tr>
<tr>
<td></td>
<td>B3 penetrating</td>
<td>B3 penetrating</td>
</tr>
<tr>
<td></td>
<td>p perianal disease modifier</td>
<td>p perianal disease modifier</td>
</tr>
</tbody>
</table>
Classification
Phenotype of Crohn's disease

Montreal classification

L1 Terminal ileum
L2 Colon
L3 Ileocolon
L4 Upper GI tract
L4+L3 Upper GI tract and distal disease

Montreal B-category

B1 Without stricture formation non-penetrating
B2 Strictureing
B3 Penetrating
B3p Perianally penetrating

Major extraintestinal manifestations and associated autoimmune disorders (blue)

- Multiple sclerosis
- Iritis, uveitis
- Sensorineural hearing loss
- Aphthous ulcers
- Autoimmune thyroiditis
- Primary sclerosing cholangitis
- Autoimmune cholangitis, Overlap syndrome
- Psoriasis
- Asthma
- Vasculitis
- Myocarditis, pericarditis
- Autoimmune hepatitis
- Immune thrombocytopenia
- Coeliac disease
- Autoimmune pancreatitis, Type 1 diabetes
- Nephritis, amyloidosis
- Urolithiasis
- Axial arthropathy (spondylitis and sacroiliitis)
- Polyarticular arthritis
- Osteoporosis
- Pauciarticular arthritis
- Pyoderma gangrenosum
- Erythema nodosum

## Classification

The Crohn's Disease Activity

<table>
<thead>
<tr>
<th>Classification</th>
<th>Patient Activity and Common Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild-to-moderate</td>
<td>Patient tolerates oral alimentation without dehydration, abdominal pain, obstruction, toxicity, or weight loss &gt;10%</td>
</tr>
<tr>
<td>Moderate-to-severe</td>
<td>Patient nonresponsive to treatment of mild-to-moderate disease; has fever, weight loss, abdominal pain, nausea and vomiting (without obstructive findings), or significant anemia</td>
</tr>
<tr>
<td>Severe-fulminant</td>
<td>Patient receiving steroids and experiencing persistent symptoms; presents with high fever, significant weight loss, persistent vomiting, intestinal obstruction, rebound tenderness, cachexia, or abscess formation</td>
</tr>
<tr>
<td>Remission</td>
<td>Patient asymptomatic, no inflammatory complications, or response to acute medical intervention (CDAI &lt;150).</td>
</tr>
</tbody>
</table>

*CDAI: Crohn's Disease Activity Index. Source: References 19, 20.*
# Classification

## Marsh Grading of Celiac Disease

<table>
<thead>
<tr>
<th>Marsh grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal mucosa</td>
</tr>
<tr>
<td>1</td>
<td>Increased number of intraepithelial lymphocytes, usually exceeding 20 per 100 enterocytes</td>
</tr>
<tr>
<td>2</td>
<td>Proliferation of the crypts of liberkuhn</td>
</tr>
<tr>
<td>3</td>
<td>Variable villous atrophy</td>
</tr>
<tr>
<td>3a</td>
<td>Partial villous atrophy</td>
</tr>
<tr>
<td>3b</td>
<td>Subtotal villous atrophy</td>
</tr>
<tr>
<td>3c</td>
<td>Total villous atrophy</td>
</tr>
<tr>
<td>4</td>
<td>Hypoplasia of the small bowel architecture</td>
</tr>
</tbody>
</table>

![Images of different grades of celiac disease](http://2.bp.blogspot.com/_zIDF7N81bbQ/S9Dte7QMstI/AAAAAAAAASI/P6Yt6D_wA6w/s1600/MARSH.jpg)
Signs and Symptoms
Crohn's disease

• The characteristic presentation is abdominal pain and diarrhea, which may be complicated by intestinal fistulization or obstruction
• Other signs and symptoms of Crohn disease may include rectal bleeding; fever; weight loss, anorexia; nausea, vomiting; malnutrition, vitamin deficiencies; generalized fatigability; bone loss
• Psychosocial issues (e.g., depression, anxiety, and coping difficulty); pediatric patients may also experience psychological issues regarding quality of life and body image
• Growth failure in pediatric patients: may precede gastrointestinal symptoms by years.
Signs and Symptoms
Celiac Disease

• In infancy disease manifests as failure to thrive, diarrhea, abdominal distention, and developmental delay
• In adults, gastrointestinal tract involvement may manifest as diarrhea, constipation, or other symptoms of malabsorption (bloating, flatus, or belching)
• Fatigue, depression, fibromyalgia-like symptoms, aphthous stomatitis, bone pain, dyspepsia, gastroesophageal reflux, etc.
• Women comprise approximately 75% of newly diagnosed disease cases and tend to have more clinically conspicuous disease.

History
Crohn's disease

• Patients with suspected Crohn disease should be evaluated initially by their primary care team, and symptoms should be elicited in detail.
• Obtain a complete medical, surgical, social, and family history, and perform a detailed review of systems.
• Preliminary laboratory data (e.g., inflammatory and anemia markers) may be helpful.
• If Crohn disease is suspected, the patient should be promptly referred to a gastroenterologist for consultation.
History
Celiac Disease

- The manifestations of untreated celiac disease can be divided into gastrointestinal symptoms and extraintestinal symptoms.
- Gastrointestinal symptoms include diarrhea due to maldigestion and malabsorption of nutrients (watery or semiformal stools, steatorrhea, flatulence, borborygmus, weight loss, weakness and fatigue, severe abdominal pain).
- Extraintestinal symptoms include anemia, a bleeding diathesis, osteopenia and osteoporosis, neurologic symptoms (motor weakness, paresthesias with sensory loss, and ataxia), skin disorders (including dermatitis herpetiformis), hormonal disorders (amenorrhea, delayed menarche, and infertility in women and impotence and infertility in men).
Physical Exam
Crohn's disease

- Vital signs are usually normal, though tachycardia may be present in anemic or dehydrated patients
- Chronic intermittent fever is a common presenting sign
- Abdominal findings may vary from normal to those of an acute abdomen
- In addition to local complications, various extraintestinal manifestations may be associated with Crohn disease, usually involving the skin, joints, mouth, eyes, liver, or bile ducts.

http://emedicine.medscape.com/article/172940-clinical
Physical Exam
Celiac Disease

- A protuberant and tympanic abdomen due to distention of intestinal loops with fluids and gas
- Weight loss, including muscle wasting or loose skin folds
- Orthostatic hypotension
- Peripheral edema
- Ecchymoses
- Hyperkeratosis or dermatitis herpetiformis
- Cheilosis and glossitis
- Peripheral neuropathy
- Chvostek sign or Trousseau sign.
Complications
Crohn's disease

- The major significant complications include intestine obstruction, abscesses, free perforation and hemorrhage, which in rare cases may be fatal.
- Obstruction occurs from strictures or adhesions that narrow the lumen, blocking the passage of the intestinal contents.
- A fistulae develop between two loops of bowel, between the bowel and bladder, between the bowel and vagina, and between the bowel and skin.
- Abscesses are walled off concentrations of infection, which can occur in the abdomen or in the perianal area.
- Crohn's disease also increases the risk of cancer in the area of inflammation.

https://en.wikipedia.org/wiki/Crohn%27s_disease
Complications
Celiac Disease

- Iron deficiency
- Lower prevalence of sexual satisfaction
- Osteoporosis
- Malignancy (lymphomas, carcinomas)
Diagnosis
Examination for Crohn’s disease

• Vital signs: normal, but possible presence of tachycardia in anemic or dehydrated patients; possible chronic intermittent fever

• Gastrointestinal: may vary from normal to those of an acute abdomen

• Genitourinary: may include presence of skin tags, fistulae, ulcers, abscesses, and scarring in the perianal region; nephrolithiasis, hydronephrosis, and enterovesical fistulae

• Musculoskeletal: possible arthritis and arthralgia

• Dermatologic: may show pallor or jaundice, mucocutaneous or aphthous ulcers, erythema nodosum, and pyoderma gangrenosum

• Ophthalmologic: may reveal episcleritis; possible uveitis

• Growth delay: decreased growth velocity (eg, height), pubertal delay

• Hematologic: hypercoagulable state.

Diagnosis
Laboratory Tests, Imaging studies and Procedures in Crohn’s disease

• Routine laboratory studies include CBC count, chemistry panel, liver function tests, inflammatory markers, stool studies, serologic tests; they may be used as surrogate markers for inflammation and nutritional status and to screen for deficiencies of vitamins and minerals.

• Imaging studies include plain abdominal radiography, barium contrast studies, computed tomography (CT) and magnetic resonance imaging (MRI), nuclear imaging, fluorine-18-2-fluoro-2-deoxy-D-glucose scanning combined with positron emission tomography, etc.

• Procedures include endoscopic visualization and biopsy, colonoscopy, ileocolonoscopy, small bowel enteroscopy, interventional radiology.

Diagnosis
Blood Tests in Crohn’s disease

• CBC may reveal anemia, which is caused by blood loss leading to iron deficiency or by vitamin B12 deficiency, caused by ileal disease impairing vitamin B12 absorption

• Serum iron, total iron binding capacity and transferrin saturation may be more easily interpreted in inflammation

• Erythrocyte sedimentation rate (ESR) and C-reactive protein help assess the degree of inflammation

• Testing for Saccharomyces cerevisiae antibodies (ASCA) and antineutrophil cytoplasmic antibodies (ANCA) help differentiate Crohn's disease from ulcerative colitis

• Low serum levels of vitamin D are associated with Crohn's disease

• Increasing levels of antilaminaribioside, antichitobioside, etc. may aid in the prognosis of Crohn's disease.

## Diagnosis

### Ordinary Findings in Crohn’s disease

<table>
<thead>
<tr>
<th>Condition</th>
<th>Frequency</th>
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</thead>
<tbody>
<tr>
<td>Terminal ileum involvement</td>
<td>Commonly</td>
</tr>
<tr>
<td>Colon involvement</td>
<td>Usually</td>
</tr>
<tr>
<td>Rectum involvement</td>
<td>Seldom</td>
</tr>
<tr>
<td>Bile duct involvement</td>
<td>No increase in rate of primary sclerosing cholangitis</td>
</tr>
<tr>
<td>Distribution of disease</td>
<td>Patchy areas of inflammation (skip lesions)</td>
</tr>
<tr>
<td>Endoscopy</td>
<td>Deep geographic and serpiginous (snake-like) ulcers</td>
</tr>
<tr>
<td>Depth of inflammation</td>
<td>May be transmural, deep into tissues</td>
</tr>
<tr>
<td>Stenosis</td>
<td>Common</td>
</tr>
<tr>
<td>Granulomas on biopsy</td>
<td>May have non-necrotizing non-peri-intestinal cryptgranulomas</td>
</tr>
</tbody>
</table>

Diagnosis

Stenosis in Crohn's disease

(A) MR enterography of Crohn's disease restricted to the terminal ileum (Montreal category L1) with inflammatory stenosis. (B) Ultrasound image of an intestinal stenosis in Crohn's disease.

Diagnosis
Ileal Crohn's disease

Resected ileum for a person with Crohn's disease.
# Diagnosis
## Serologic Tests in Celiac Disease

<table>
<thead>
<tr>
<th>ANTIBODY TEST</th>
<th>SENSITIVITY (%)</th>
<th>SPECIFICITY (%)</th>
<th>TIME COURSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>IgA antiendomysial antibody</td>
<td>85 to 100</td>
<td>96 to 100</td>
<td>Antibody disappears within several months after institution of gluten-free diet.</td>
</tr>
<tr>
<td>IgA antitransglutaminase antibody</td>
<td>95</td>
<td>90</td>
<td>Limited data; correlated with IgA antiendomyosal antibody in studies</td>
</tr>
<tr>
<td>IgA antigliadin antibody</td>
<td>53 to 100</td>
<td>65 to 100</td>
<td>More persistent than IgA antiendomysial antibody; may persist for 6 months or longer</td>
</tr>
<tr>
<td>IgG antigliadin antibody</td>
<td>57 to 100</td>
<td>42 to 98</td>
<td>Most persistent; may be detectable up to 12 months after institution of gluten-free diet</td>
</tr>
</tbody>
</table>

False-positive tests reported in patients with Crohn’s disease, wheat-protein allergy, and postdiarrhea states.
# Diagnosis

Abnormal Laboratory Findings in Celiac Disease

<table>
<thead>
<tr>
<th>Laboratory Finding</th>
<th>Pathophysiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anemia</td>
<td>Iron deficiency; vitamin B&lt;sub&gt;12&lt;/sub&gt; and/or folate deficiency</td>
</tr>
<tr>
<td>Elevated alkaline phosphatase level</td>
<td>Osteoporosis, osteomalacia</td>
</tr>
<tr>
<td>Elevated aspartate transaminase and alanine transaminase levels</td>
<td>Minimal elevation common in celiac disease; presumably autoimmune</td>
</tr>
<tr>
<td>Decreased albumin level</td>
<td>Malnutrition</td>
</tr>
<tr>
<td>Elevated calcium level, decreased phosphate level</td>
<td>Vitamin D deficiency, secondary hyperparathyroidism</td>
</tr>
<tr>
<td>Thrombocytosis, leukocytosis</td>
<td>General inflammatory reaction</td>
</tr>
<tr>
<td>Coagulopathy</td>
<td>Decreased vitamin K absorption</td>
</tr>
<tr>
<td>Low high-density and low-density lipoprotein cholesterol levels</td>
<td>Decreased fat absorption, decreased hepatic lipoprotein production</td>
</tr>
</tbody>
</table>

Diagnosis
Algorithm for the Diagnosis of Celiac disease

Clinical suspicion: patient with symptoms or risk factors suggestive of gluten-sensitive enteropathy (celiac disease)

Serologic testing:
- IgA antigliadin antibody
- IgG antigliadin antibody
- IgA antiendomysial antibody or IgA antitransglutaminase antibody

All tests negative:
- Low probability of celiac disease
  - If clinical suspicion is high, consider gluten challenge or EGD.

IgA-negative tests, IgG-positive test:
- Quantitative IgA measurements
  - IgA normal
    - Intermediate probability of celiac disease
      - Plan EGD, with or without gluten challenge.
  - IgA deficient
    - High probability of celiac disease
      - EGD to rule out celiac disease

Any IgA-positive test:
- High probability of celiac disease
Diagnosis

Other Conditions with Similar Symptoms as Crohn's disease and Celiac disease

- Intestinal tuberculosis
- Behçet’s disease
- Ulcerative colitis
- Nonsteroidal anti-inflammatory drug enteropathy
- Irritable bowel syndrome
- Crohn's disease (for celiac disease)
- Celiac disease (for Crohn's disease)

https://en.wikipedia.org/wiki/Crohn%27s_disease
Management
Lifestyle modification in Crohn's disease

- Lifestyle changes reduce symptoms, including dietary adjustments, elemental diet, proper hydration, and smoking cessation
- Diets that include higher levels of fiber and fruit are associated with reduced risk, while diets rich in total fats, polyunsaturated fatty acids, meat, and omega-6 fatty acids may increase risk
- Eating small meals frequently instead of big meals
- A food diary may help with identifying foods that trigger symptoms
- Some people should follow a low dietary fiber diet
- Some find relief in eliminating casein and gluten from their diets
- Fatigue can be helped with regular exercise, a healthy diet, and enough sleep.

https://en.wikipedia.org/wiki/Crohn%27s_disease
Management
Patient Education

Education of patients and their families is encouraged and is extremely important in the treatment process.
Management
Pharmacotherapy of Crohn's disease

• 5-Aminosalicylic acid derivative agents (e.g., mesalamine rectal, mesalamine, sulfasalazine, balsalazide)
• Corticosteroids (e.g., prednisone, methylprednisolone, budesonide, hydrocortisone, prednisolone)
• Immunosuppressive agents (e.g., mercaptopurine, methotrexate, tacrolimus)
• Monoclonal antibodies (e.g., infliximab, adalimumab, certolizumab pegol, natalizumab, vedolizumab)
• Antibiotics (e.g., metronidazole, ciprofloxacin)
• Antidiarrheal agents (e.g., loperamide, diphenoxylate-atropine)
• Bile acid sequestrants (e.g., cholestyramine, colestipol)
• Anticholinergic agents (e.g., dicyclomine, hyoscyamine, propantheline).

Management
Surgery of Crohn's disease

- Crohn disease has no surgical cure
- Surgical management of the terminal ileum, ileocolon, and/or upper gastrointestinal tract may include resection of the affected bowel, ileocolostomy or proximal loop ileostomy, drainage of any septic foci with later definitive resection, strictureplasty, bypass endoscopic dilatation of symptomatic strictures.
Management
Celiac disease 1

- At present, the only effective treatment is a lifelong gluten-free diet
- No medication exists that will prevent damage or prevent the body from attacking the gut when gluten is present
- Strict adherence to the diet allows the intestines to heal, leading to resolution of all symptoms in most cases and, depending on how soon the diet is begun, can also eliminate the heightened risk of osteoporosis and intestinal cancer and in some cases sterility
- The diet can be cumbersome; failure to comply with the diet may cause relapse
- Up to 5% of people have refractory disease, which means they do not improve on a gluten-free diet, and if alternative causes have been eliminated, steroids or immunosuppressants (such as azathioprine) may be considered in this scenario

https://en.wikipedia.org/wiki/Coeliac_disease#Treatment
Management
Celiac disease 2

• In many countries, gluten-free products are available on prescription and may be reimbursed by health insurance plans.
• Gluten-free products are usually more expensive and harder to find than common gluten-containing foods.
• The term gluten-free is generally used to indicate a supposed harmless level of gluten rather than a complete absence.
• The European Commission issued regulations in 2009 limiting the use of "gluten-free" labels for food products to those with less than 20 mg/kg of gluten, and "very low gluten" labels for those with less than 100 mg/kg.
• In the United States, the FDA issued regulations in 2013 limiting the use of "gluten-free" labels for food products to those with less than 20 ppm (one part per million = one part per 1,000,000 parts, one part in $10^6$) of gluten.

https://en.wikipedia.org/wiki/Coeliac_disease#Treatment
Prognosis
Crohn's disease, Celiac disease

• Appropriate medical therapy helps patients with Crohn’s disease to have a reasonable quality of life, with an overall good prognosis and an extremely low risk of a fatal outcome; most patients develop complications that require surgery, and postoperative clinical relapse occurs in a significant proportion.

• Most patients who have celiac disease begin to feel better soon after starting the *gluten-free* diet; patients who begin a strict, *gluten-free* diet immediately after diagnosis have the best chance of living a healthy and active life; full recovery can take a few months to several years.
Prophylaxis
Crohn's disease, Celiac disease

• Crohn's disease can not be prevented
• Celiac disease can not be prevented; in the last years, several studies suggested a protective role of breast-feeding.
Abbreviations

ANCA - antineutrophil cytoplasmic antibodies
ASCA - Saccharomyces cerevisiae antibodies
CT – computed tomography
ESR - Erythrocyte sedimentation rate
IBD - inflammatory bowel disease
MRI - magnetic resonance imaging
NK - natural killer cells
ppm - one part per million
Diagnostic and treatment guidelines

- Gluten-Sensitive Enteropathy (Celiac Disease)
- ESPEN guidelines on chronic intestinal failure in adults
- Inflammatory Bowel Disease
- Management of Crohn’s Disease in Adults
- Guidelines for the investigation of chronic diarrhea
- Radiation-induced small bowel disease: latest developments and clinical guidance
- Guidelines for the initial biopsy diagnosis of suspected chronic idiopathic inflammatory bowel disease