Inflammatory Bowel Disease; Common Dermatoses

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NOTE FROM THE PUBLISHER:
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I. INTRODUCTION

Inflammatory bowel disease (IBD), sometimes called idiopathic IBD because of its unknown etiology, comprises 2 similar yet distinct diseases: Crohn’s disease and ulcerative colitis (UC). UC is characterized by inflammation of the colonic mucosa only. This inflammation always involves the rectum and proceeds proximally in a continuous fashion to involve variable lengths of colon. UC does not involve other parts of the gastrointestinal (GI) tract besides the colon, except for occasional involvement of the terminal ileum known as backwash ileitis. In contrast, Crohn’s disease manifests as inflammation throughout the entire thickness of the bowel wall, and many complications of the disorder develop from this transmural involvement. Crohn’s disease may involve any part of the GI tract, from mouth to anus, but spares the rectum in most cases. One can sometimes find normal bowel, referred to as skip lesions, in between areas of diseased bowel.

In some patients, IBD can run an indolent course; others, however, may have substantial morbidity, with sequelae including chronic abdominal pain, bowel obstruction, multiple abdominal surgeries, liver disease, and colorectal cancer. Often, the primary care physician must make a diagnosis of IBD and, with the gastroenterologist, plays an important role in managing patients with chronic IBD symptoms. This article will review the presentation, course, treatment, and complications of IBD. Five case patients are presented to highlight features of the management of IBD.

II. CASE PATIENT 1

PRESENTATION

Patient 1 is a 21-year-old female college student who presents to her primary care physician because of bloody diarrhea, which began a few weeks ago. Since symptom onset, her bowel movements have become more frequent and increasingly loose, such that she now has 3 to 5 bowel movements each day. During the past week, she has noted small amounts of blood and mucus in her stool. She has been having tenesmus and crampy, left lower quadrant abdominal pain that is relieved with bowel movements. Also, she has painful, red bumps on her shins as well as pain in her ankles and knees. She denies nausea, vomiting, and weight loss. Her medical and surgical histories are both unremarkable. Currently, she is not taking any medication. Recently, she returned from summer break, which she spent volunteering at a clinic in southern Mexico and traveling throughout Central America.

On physical examination, patient 1’s pulse and blood pressure are normal and do not change with postural positioning. Her abdominal examination shows tenderness in the left lower quadrant, without guarding or rebound. Perianal skin is reddened but otherwise unremarkable. Rectal examination reveals a scant amount of brown stool mixed with blood and mucus. Several red, tender nodules are noted on the anterior surface of her shins bilaterally. Examination of her joints is unremarkable.

CUTANEOUS MANIFESTATIONS OF INFLAMMATORY BOWEL DISEASE

• What is the most likely diagnosis for patient 1’s skin lesions?
  A) Erythema nodosum
  B) Erythema migrans
  C) Erythema multiforme
  D) Erythema infectiosum
  E) Erythema marginatum

Discussion

The correct answer is A. The description of the rash suggests erythema nodosum (Figure 1). Erythema nodosum (EN) is the most common dermatologic
condition associated with IBD. Affecting as many as 15% of patients with Crohn’s disease and 4% of patients with UC, EN may precede IBD development but more often parallels the disease course. EN may occur with various other diseases, including sarcoidosis and many infectious diseases.

Another cutaneous but less common manifestation of IBD is pyoderma gangrenosum (Figure 2), which is seen in approximately 2% of patients with UC and less frequently in patients with Crohn’s disease. Initially, pyoderma gangrenosum presents with purple papules that enlarge and coalesce to form necrotic ulcers. Usually, this lesion occurs on the lower limbs, at sites of previous trauma, or on surgical scars. Treatment involves immunosuppression (ie, systemic corticosteroids) and treating the underlying intestinal inflammation because this can assist with wound healing. Occasionally, total colectomy is required to control extensive pyoderma. In addition to medical therapy, treatment must include gentle débridement of devitalized tissue, which may be accomplished by daily whirlpool baths. A rare cutaneous manifestation is known as metastatic Crohn’s disease. Patients present with noncaseating papules on the face, extremities, or vulva; this condition tends to flare when Crohn’s disease is active.

DIFFERENTIAL DIAGNOSIS

At this point, it important to test for the various causes of diarrhea as well as to assess the degree of and reason for patient 1’s blood loss. Laboratory studies are performed for patient 1, including complete blood count, levels of electrolytes and serum iron, prothrombin time, and partial thromboplastin time. Results are significant only for mild anemia, with a plasma hemoglobin of 11.1 g/dL. To investigate infectious etiologies, stool is sent for culture and examined for ova and parasites.

- Which of the following could NOT be the cause of patient 1’s symptoms?
  A) Shigella species
  B) Ulcerative colitis
  C) Crohn’s disease
  D) Giardiasis
  E) Systemic lupus erythematosus

Discussion

The correct answer is D. Patient 1’s age and symptoms are fairly classic for a first presentation of IBD. In particular, the pattern of her pain, along with tenesmus and relatively normal appearance of the perianal skin, make the diagnosis of UC more likely than Crohn’s disease. However, in the differential diagnosis of IBD, various potential causes (including infections and vasculitis) must be considered (Table 1). Given patient 1’s history of recent travel, one must investigate infectious etiologies such as Salmonella species, Shigella species, Campylobacter species, and amebiasis. If she had reported eating...
undercooked hamburger, *Escherichia coli* O157:H7 should be suspected, particularly if she developed the hemolytic uremic syndrome. In addition, because patient 1 is a young woman, vasculitis involving the bowel from such causes as systemic lupus erythematosus (SLE) should be considered. Although *Giardia* (which typically presents with steatorrhea and foul-smelling stools) is a relatively common cause of diarrhea, this parasite does not usually cause hematochezia or tenesmus.

### EPIDEMIOLOGY

- In which of the following ethnic groups would a diagnosis of IBD be most likely?
  - A) Ashkenazi Jews from the United States
  - B) Sephardic Jews from Israel
  - C) Americans of Asian descent
  - D) African-Americans

**Discussion**

The correct answer is A. Although the etiology of IBD remains elusive, epidemiologic data show geographic and ethnic variance and indicate that environmental factors may also play a role. Rates for both UC and Crohn’s disease are highest among white persons in northern Europe and North America. In these geographic areas, the incidence and prevalence of UC are approximately 2 to 10 per 100,000 persons and 35 to 100 per 100,000 persons, respectively. Crohn’s disease is somewhat less common, with an incidence of 1 to 6 per 100,000 and a prevalence of 10 to 100 per 100,000.

These rates are somewhat lower in central and southern Europe; they are much lower in Africa, Asia, and Central and South America.

In any given region, IBD is more common among Caucasians than other races. The disease is 2 to 8 times more common among Jews than non-Jews and is more common in Jewish persons living in the United States or Europe than in those living in Israel. Furthermore, IBD is more common among Ashkenazi Jews than Sephardic or Asian Jews.

Environmental factors have been shown to play a role in the development of IBD as well. Interestingly, cigarette smoking seems to protect against UC but not Crohn’s disease. UC is more common among non-smokers and former smokers than among smokers. Conversely, patients with Crohn’s disease smoke at a similar or greater rate than that of the general population. Evidence suggests that smokers with Crohn’s disease may have a poor response to medical therapy. In addition, Crohn’s disease (but not UC) is more common in urban than in rural areas. Migrant studies have shown that when a person moves from an area of low prevalence to an area of higher prevalence, the incidence of the disease increases.

### GENETIC FACTORS

- An individual would have the greatest risk of developing IBD if which of the following first-degree relatives had the disease?
  - A) A child
  - B) A sister
  - C) A parent
  - D) An identical twin

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**Table 1.** Differential Diagnosis of Inflammatory Bowel Disease

<table>
<thead>
<tr>
<th>Infectious causes</th>
<th>Vascular causes</th>
<th>Drug-induced colitis</th>
<th>Miscellaneous disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bacteria</strong></td>
<td>Polyarteritis nodosa</td>
<td>NSAIDs</td>
<td>Microscopic colitis (lymphocytic and collagenous)</td>
</tr>
<tr>
<td><em>Campylobacter</em> species</td>
<td>Systemic lupus erythematosus</td>
<td>Gold</td>
<td>Radiation colitis or proctitis</td>
</tr>
<tr>
<td><em>Salmonella</em> species</td>
<td>Other vasculitides</td>
<td>Methylodopa</td>
<td>Solitary rectal ulcer syndrome</td>
</tr>
<tr>
<td><em>Shigella</em> species</td>
<td>Ischemic colitis</td>
<td>Flucytosine</td>
<td>Neutropenic cecitis</td>
</tr>
<tr>
<td><em>Yersina</em> species</td>
<td></td>
<td>Isotretinoin</td>
<td>Diverticulitis</td>
</tr>
<tr>
<td><em>Escherichia coli</em> O157:H7</td>
<td></td>
<td>Allopurinol</td>
<td>Eosinophilic gastroenteritis</td>
</tr>
<tr>
<td><em>Clostridium difficile</em></td>
<td></td>
<td></td>
<td>Cathartic colon</td>
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<tr>
<td>Gonorrhea</td>
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<tr>
<td><em>Chlamydia</em> species</td>
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<tr>
<td>Spirochetes</td>
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<td>Syphilis</td>
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<td>Viruses</td>
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<tr>
<td>Herpes simplex virus</td>
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<tr>
<td>Cytomegalovirus</td>
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<tr>
<td>Parasites</td>
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<tr>
<td>Amebiasis</td>
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<tr>
<td>Schistosomiasis</td>
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<tr>
<td><em>Balantidium coli</em></td>
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<td><em>Endolimax nana</em></td>
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<td>Fungi</td>
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<tr>
<td><em>Histoplasma capsulatum</em></td>
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<tr>
<td><strong>Malignancies</strong></td>
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<tr>
<td>Lymphoma</td>
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<tr>
<td>Ileoceleal carcinoma</td>
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</tbody>
</table>

NSAIDs = nonsteroidal anti-inflammatory drugs.

Discussion

The correct answer is D. The greatest risk factor for developing IBD is having a relative with the disease, which confers about a 15% lifetime risk.2 A first-degree relative with IBD is associated with the highest risk for a patient. Offspring and siblings of those with IBD have an approximate 9% risk of developing the disease; parents of affected children have a 3.5% risk. Monozygotic twins of affected individuals have the greatest risk at approximately 25%. Dizygotic twins have about the same risk as siblings who are not twins.

FURTHER TESTING FOR PATIENT 1

Patient 1 undergoes a colonoscopy, which shows diffuse inflammation with some superficial ulceration of the distal colon, extending from the rectum to the splenic flexure where the inflammation abruptly stops. No evidence of disease is noted in the remainder of the colon or in the terminal ileum. Biopsies reveal distortion of crypt architecture, inflammation in the lamina propria, a villous mucosa, and crypt atrophy. These histologic findings along with the fact that the colitis does not involve the entire colon, confirm a diagnosis of UC in patient 1. Unfortunately, there are no criteria for diagnosing IBD such as exist for endocarditis or SLE. In most cases, colonoscopy is necessary.

MEDICAL THERAPY FOR PATIENT 1

• On questioning about drug allergies, patient 1 states that she developed a rash with trimethoprim-sulfamethoxazole, which she received for a urinary tract infection 1 year ago. What is the most appropriate pharmacologic treatment for patient 1?
  A) Oral methotrexate, 15 mg weekly
  B) Azathioprine, 2.0 to 2.5 mg/kg of body weight
  C) Oral sulfasalazine, 500 mg 4 times daily
  D) Mesalamine enema, 4 g every night

Discussion

The correct answer is D. Many therapies are available for the treatment of IBD. The 5-aminosalicylic acid (5-ASA) group of drugs is first-line therapy for mild-to-moderate IBD (Table 2).3 Originally, sulfasalazine was used in IBD treatment; however, patients with a sulfia allergy (eg, patient 1) cannot take this agent because of adverse reactions to the sulfapyridine component.4 Studies have shown that the active component of sulfasalazine is the 5-ASA moiety.5 For patients with a sulfia allergy, 5-ASA drugs without sulfia are available.

The best drug for any given patient delivers the largest amount of the agent to the affected area. Several formulations of 5-ASA drugs exist that are designed to deliver medication to specific parts of the GI tract (Table 2). Of the oral formulations (mesalamine, olsalazine, and sulfasalazine), only mesalamine works in the small bowel. Mesalamine suppositories are appropriate for proctitis, although mesalamine enemas will treat splenic flexure disease and would thus be most appropriate for patient 1. Methotrexate, at doses of 25 mg or less per week, has been shown to induce remission in Crohn’s disease and may provide benefits in UC as well6–10 but would not be considered first-line treatment for patient 1.

FURTHER PRESENTATION OF PATIENT 1

Patient 1 returns to the office after 6 weeks because of continued abdominal pain, diarrhea, bloody stools, and tenesmus.

• What is the most appropriate drug to add to patient 1’s regimen?
  A) Metronidazole, 250 mg orally 3 times a day
  B) Prednisone, 60 mg orally once a day
  C) Ciprofloxacin, 500 mg orally twice a day
  D) 6-Mercaptopurine, 1.5 mg/kg of body weight

Discussion

The correct answer is B. A number of agents are used to treat IBD (Table 3). Short-term use of corticosteroids is indicated for mild-to-moderate flares of IBD

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Table 2. 5-Aminosalicylic Acid Drugs and Sites of Action*

<table>
<thead>
<tr>
<th>Agents</th>
<th>Daily Dose, g</th>
<th>Site of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Oral agents</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mesalamine (Pentasa)</td>
<td>2–4</td>
<td>Duodenum through colon</td>
</tr>
<tr>
<td>Mesalamine (Asacol)</td>
<td>1.6–2.4</td>
<td>Distal ileum, colon</td>
</tr>
<tr>
<td>Olsalazine (Dipentum)</td>
<td>0.75–3.0</td>
<td>Colon</td>
</tr>
<tr>
<td>Sulfasalazine (Azulfidine)</td>
<td>2–6</td>
<td>Colon</td>
</tr>
<tr>
<td><strong>Topical agents</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mesalamine enema (Rowasa)</td>
<td>4</td>
<td>Rectum, sigmoid, left colon</td>
</tr>
<tr>
<td>Mesalamine suppositories (Rowasa)</td>
<td>0.5–1.0</td>
<td>Rectum</td>
</tr>
</tbody>
</table>

*The 5-aminosalicylic acid group of drugs is first-line therapy for mild-to-moderate inflammatory bowel disease. The best drug for any given patient delivers the largest amount of the agent to the affected area.

of adherence, chemotaxis, and phagocytosis. For mild proinflammatory cytokines and the leukocyte functions as well. Metronidazole has been shown in patients with Crohn’s disease and in UC for patients who are either resistant to or dependent on corticosteroids; these agents exert an anti-inflammatory effect and inhibit cytotoxic T-cell and natural killer cell function. The most serious side effect of these medications is bone marrow toxicity (eg, leukopenia and thrombocytopenia). Methotrexate is a folic acid antagonist that exerts its immunomodulatory effect by inhibiting production of thymidylate, purines, and methionine, leading to accumulation of adenosine (which is an anti-inflammatory substance). Methotrexate has been shown in randomized, double-blind, placebo-controlled trials to induce remission in patients with Crohn's disease. Methotrexate has several side effects that limit its usefulness, including hepatotoxicity, myelosuppression, pulmonary toxicity, decreased fertility, and enteritis-colitis. Cyclosporine is a cyclic peptide that inhibits production of several pro-inflammatory cytokines such as interleukin 2 (IL-2), interferon γ, tumor necrosis factor–α (TNF-α), GM-CSF (granulocyte-macrophage colony-stimulating factor), and IL-4. Cyclosporine can be used to treat acute flares of both Crohn’s disease and UC when disease is refractory to other therapies. The side effects of cyclosporine are serious and include decreased creatinine clearance, hypertension, seizures, and hepatotoxicity. Because of these problems, use of cyclosporine is limited to patients whose symptoms are refractory to other treatments.

**Infliximab**, an agent newly approved by the U.S. Food and Drug Administration (FDA) for IBD, is the first biologic response modifier to be used in the management of IBD. A chimeric monoclonal IgG1 antibody directed against TNF, infliximab has been approved for use in patients with moderate-to-severe disease who are not responding to conventional therapy. A randomized, single-blinded study demonstrated that infliximab was significantly better than placebo at inducing and maintaining remission. Another study has shown that infliximab is beneficial for healing fistulas. Infliximab appears to be safe for short-term use; however, long-term data regarding its safety are lacking. This agent is approved for single-dose treatment in nonfistulous disease and 3-dose treatment in patients with fistulas.

**COLORECTAL CANCER IN INFLAMMATORY BOWEL DISEASE**

Patient 1 has been researching UC on the internet. She has learned that because of her UC, she is at greater than average risk for developing colon cancer.

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**Table 3. Medications Used to Treat Inflammatory Bowel Disease**

<table>
<thead>
<tr>
<th>5-ASA drugs</th>
<th>Corticosteroids</th>
<th>Immunomodulators</th>
<th>Biologic response modifier</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfasalazine</td>
<td></td>
<td></td>
<td>Infliximab</td>
</tr>
<tr>
<td>Mesalamine</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Olsalazine</td>
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<tr>
<td>Balsalazide</td>
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<tr>
<td><strong>Antibiotics</strong></td>
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<td></td>
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</tr>
<tr>
<td>Metronidazole</td>
<td></td>
<td></td>
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<tr>
<td>Ciprofloxacin</td>
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</tbody>
</table>

5-ASA = 5-aminosalicylic acid.

• Which of the following colon cancer screening strategies do you recommend?
   A) Flexible sigmoidoscopy every 3 to 5 years beginning at age 50 years
   B) Fecal occult blood testing yearly, followed by colonoscopy if the test is positive
   C) Colonoscopy with random biopsies looking for dysplasia every 1 to 2 years, beginning 8 years after the initial onset of UC
   D) Colonoscopy with random biopsies looking for dysplasia every 1 to 2 years, beginning 15 years after the initial onset of UC
   E) Immediate total colectomy with ileoanal pull-through anastomosis, followed by endoscopic surveillance of the anastomosis every 1 to 2 years

Discussion
The correct answer is D. Patients with UC are at substantially increased risk of developing colon cancer, with a lifetime risk of approximately 3% to 5%. This risk is greater in patients with pancolitis than in those with predominantly left-sided disease; however, patients who develop UC at an age younger than 15 years may be at increased risk for cancer. In patients with pancolitis, risk begins to increase after about 8 years and increases steadily thereafter. Published estimates of lifetime colon cancer risk for these patients vary, from as high as 34% after 25 years of disease in Sweden to as low as 1.4% after 18 years of disease in Denmark. Individuals with predominantly left-sided colitis who develop cancer tend to do so later. Because of these statistics, patients with UC should undergo colonoscopy every 1 to 2 years, beginning 8 years after an initial diagnosis of pancolitis and 15 years after an initial diagnosis of left-sided disease. Cancer surveillance is performed using colonoscopy, with random biopsies of colonic mucosa in order to look for evidence of dysplasia.

Despite surveillance, patients with UC may still develop occult colorectal cancer. Some patients, wishing to minimize their colon cancer risk, may choose to undergo a total colectomy. However, this procedure is not currently recommended for all patients with UC. Periodic sigmoidoscopy beginning at age 50 years would be appropriate screening for an individual at average risk for colorectal cancer but would be inappropriate for this high-risk patient. Similarly, testing stool annually for occult blood would be appropriate for routine screening of average-risk patients age 50 years and older.

Patients with Crohn’s disease develop cancer less often than patients with UC but more often than the general population. As in UC, colon cancer risk in Crohn’s disease increases with the length of time after the initial diagnosis. Patients with Crohn’s disease tend to develop cancer at a younger age than the general population. Surveillance is more difficult in these patients because of strictures and mucosal abnormalities. Despite the increased colon cancer risk, no studies have shown a benefit in screening Crohn’s disease patients for cancer; therefore, screening is not recommended.

III. CASE PATIENT 2

PRESENTATION
Patient 2 is a 23-year-old, previously healthy man who is brought to the emergency department by a family member because of mental status changes. He had been well until a couple of weeks ago, when he began to have loose stools. Since then, his stools have become more frequent and looser. For the past 2 days, he has been having 8 to 10 bloody bowel movements per day. He began taking loperamide 2 days before admission, and his diarrhea has improved since beginning this medication. However, on the night before presentation, he developed a temperature of 39.5°C. In the morning, he became confused and was then brought to the emergency department.

On presentation, patient 2’s temperature is 38.8°C and his pulse is 130 bpm. Blood pressure is 100/60 mm Hg but decreases to 60/– mm Hg when the patient sits up. His oral mucosa is dry. His abdomen appears distended and is diffusely tender without rebound tenderness, and his bowel sounds are hypoactive. Rectal examination reveals a scant amount of blood and mucus. He is disoriented to time and location. His neurologic examination is otherwise nonfocal.

Laboratory studies reveal a leukocyte count of 13,200 cells/µL, plasma hemoglobin of 11.7 g/dL, hematocrit of 36%, and normal platelet count. Serum levels of sodium, chloride, and bicarbonate are low. Abdominal films show a dilated transverse colon with an 8-cm diameter.

• Which of the following medications are contraindicated in patient 2?
   A) Intravenous hydrocortisone
   B) Intravenous morphine
   C) Intravenous ampicillin/sulbactam
   D) Sulfasalazine

Discussion
The correct answer is B. Patient 2 has toxic megacolon resulting from inflammatory colitis, a rare but serious
complication of IBD. Twenty years ago, the lifetime risk of developing toxic megacolon in IBD was between 1% and 5% but is believed to be lower at this time, owing to earlier recognition and better management of severe colitis.17 Toxic megacolon can occur as a complication of other disorders besides IBD, including infectious causes (such as Clostridium difficile pseudomembranous colitis) and non-infectious causes. Toxic megacolon results from pancolitis, not segmental disease, and may occur at any age. Usually, this condition afflicts patients early in the course of their IBD. Predisposing factors include hypokalemia and certain drugs that slow intestinal motility (ie, opiates, antidiarrheals, and anticholinergics) and are thus contraindicated. In patient 2, the loperamide may have triggered his toxic megacolon. Presentation may be preceded by symptoms of colitis for a week or more, and bloody diarrhea is often seen. Paradoxically, improving diarrhea may signal the onset of toxic megacolon. Clinically, the patient with this disorder has dehydration, tachycardia, and orthostatic hypotension. The abdomen will be distended and tympanitic, with or without peritoneal signs. One can make a diagnosis of toxic megacolon if the patient meets the diagnostic criteria (Table 4) and has an abdominal radiograph showing a colonic diameter of 6 cm in any segment. Typically, dilation occurs in the transverse or right colon. The radiograph may also demonstrate multiple air-fluid levels in the colon, loss of haustral markings, and mucosal ulcerations, which appear as air-filled crevices between large pseudopolypoid projections into the colonic lumen.17 Treatment involves bowel rest, decompression with a nasogastric tube, and management of the patient’s dehydration and electrolyte abnormalities (particularly hypokalemia, which may exacerbate the condition).17 Consultation with an experienced surgeon should be sought as soon as this diagnosis is entertained. Serial abdominal examinations and plain radiographs should be performed to monitor the colon’s diameter. Intravenous steroids are used to decrease inflammation, despite a lack of evidence demonstrating their benefit. Intravenous antibiotics are often used in anticipation of peritonitis. 5-ASA drugs have no role in the treatment of toxic megacolon.

Complications of toxic megacolon include massive hemorrhage and perforation resulting in either abscess formation or frank peritonitis. Any of these complications, as well as progression of toxicity and of colonic dilatation, are indications for surgery. Subtotal colectomy with ileostomy is the usual emergent treatment because it causes less morbidity and mortality than does total proctocolectomy. Mortality of patients undergoing colectomy is 2% to 8% in those without perforation and 40% in those with perforation.17 Thus, it is important to identify those patients who are not responding to conservative management and to operate on them before a colonic perforation occurs.

### DIFFERENTIATING CROHN’S DISEASE FROM ULCEARTIVE COLITIS

At 48 hours, patient 2 remains febrile and becomes hypotensive. His blood pressure does not respond to intravenous fluids, and he is placed on a dopamine drip. His abdomen is becoming more distended and tympanic. A surgeon, who had been consulted when the patient was admitted, performs an exploratory laparotomy and subtotal colectomy with end-ileostomy. The surgical specimen is sent for pathologic analysis.

**• Which of the following pathologic features would indicate that patient 2’s underlying disease process is UC rather than Crohn’s disease?**

A) Transmural inflammation  
B) Noncaseating granulomata  
C) Lymphoid aggregates  
D) Aphthous ulcers  
E) None of the above

**Discussion**

The correct answer is E. All of these pathologic and histologic findings are seen in Crohn’s disease and are not usually found in UC. Although many clinical, radiographic, endoscopic, and histologic features enable practitioners to distinguish Crohn’s disease from UC, approximately 25% of colitis patients are classified with
indeterminate disease because of physician inability to categorize the problem definitively as Crohn’s disease or UC. The distinction is not simply one of nomenclature but has important clinical consequences. For example, patients with known UC should receive a total colectomy with an ileo-anal pull-through anastomosis. In contrast, Crohn’s disease is likely to recur and spare the rectum; thus, the appropriate procedure is a subtotal colectomy with rectal sparing.

Many clinical features can help the physician to differentiate UC from Crohn’s disease (Table 5). Typically, UC is characterized by passage of blood and mucus via the rectum as well as by urgency and tenesmus. UC tends to cause crampy, lower abdominal pain relieved with bowel movements. In contrast, Crohn’s disease tends to produce right-sided and postprandial pain. Perianal skin findings are much more common in Crohn’s disease, occurring in as many as 90% of patients. These findings include violaceous color of the anal margin, perianal skin tags, fissures, abscesses, sinuses, and fistulae.

HEPATO-BILIARY COMPLICATIONS OF INFLAMMATORY BOWEL DISEASE

Patient 2

The surgical specimen from patient 2 is examined and determined to be most consistent with UC. There is diffuse colitis without evidence of skip lesions. No evidence of aphthous ulcers or transmural involvement is seen. The terminal ileum appears normal.

Patient 2 recovers from the surgery uneventfully and is discharged to home. He eventually undergoes restorative surgery with creation of an ileal reservoir and ileo-anal anastomosis. After the second procedure, he does well for several years, until he presents to his primary care physician with fatigue, pruritus, and jaundice. The patient has had a 10-pound weight loss and his skin is jaundiced and his sclerae are icteric. The liver is percussed to 16 cm in the mid-clavicular line. The spleen is palpable on deep inspiration.

- Which of the following is the best test to confirm a diagnosis of primary sclerosing cholangitis (PSC)?
  A) Endoscopic retrograde cholangiopancreatography (ERCP)
  B) Percutaneous transhepatic cholangiography
  C) Liver biopsy
  D) Ultrasound of the abdomen
  E) Magnetic resonance cholangiography

Discussion

The correct answer is A. PSC is a chronic inflammatory disease affecting the entire biliary tree and causing fibrosis, resulting in beading and strictures of the bile ducts. This disorder occurs in approximately 7.5% of patients with UC. PSC may be asymptomatic for several years or may be discovered incidentally by an isolated elevation in alkaline phosphatase levels. Common findings on presentation include fatigue, jaundice, pruritus, acholic stools, bacterial cholangitis, hepatosplenomegaly, and portal hypertension.

ERCP is the test of choice for diagnosing PSC. Percutaneous transhepatic cholangiography can also be used but is associated with high failure rates secondary to narrowed hepatic ducts. Liver biopsy is considered an adjunctive test. Magnetic resonance cholangiography may emerge as the noninvasive test of choice but is not a standard test at this time. When cholangiography or ERCP is performed, PSC appears as irregular, diffusely narrowed, tortuous and ectatic areas within or outside of the liver.

PSC is a progressive and fatal disease, with 5-year survival rates ranging from 50% to 70%. Death occurs as the result of liver failure or complications thereof. Several factors are associated with a poor outcome, including advanced age, high bilirubin level, anemia, presence of IBD, and advanced histologic disease.

Various medical treatments (eg, corticosteroids, ursodiol, methotrexate, and bile acid sequestrants) have been tested without success. Colectomy does not cure the disease either, as demonstrated in patient 2. Liver transplantation is the only effective cure for PSC and should be considered in patients with bilirubin levels greater than 5 mg/dL, portal hypertension, ascites, or varices. Interestingly, UC may develop a more progressive course after liver transplantation.

In addition to PSC, several hepatobiliary diseases can occur in patients with IBD. These diseases include fatty liver, cholangiocarcinoma, gallstones, cirrhosis, chronic hepatitis, granulomatous hepatitis, and amyloidosis. The hepatobiliary diseases are important sequelae of IBD, potentially resulting in significant morbidity and mortality. Therefore, the primary care physician should be aware of these conditions and should monitor his patients with IBD for evidence of disease onset.

The more common hepatobiliary diseases associated with IBD are fatty liver, cholangiocarcinoma, and gallstones. Of these 3 conditions, fatty liver is the perhaps the most common, with an incidence of 5% to 80%. Mild elevations in alkaline phosphatase (less than twice that of normal levels) or hepatomegaly suggests a diagnosis of fatty liver, a benign condition that does not progress to cirrhosis. Treatment involves identifying and treating the underlying cause, which may include infection, medication side effects, malnutrition, and protein malabsorption.
Bile duct carcinoma may occur either in Crohn’s disease or UC but usually does so in patients with preexisting PSC. This disease may present with obstructive jaundice, weakness, right upper quadrant pain, and fever. Radiographically, it may be hard to differentiate from PSC. Recurrence is common in liver transplant recipients, and the prognosis for cholangiocarcinoma is poor, with a mean survival of approximately 6 months. Gallstones are commonly seen in Crohn’s ileitis, with a prevalence of 13% to 33%21; however, they are no more common in UC than in the general population. Stone formation increases with duration of disease and length of the affected ileum. The pathologic process is believed to involve malabsorption of bile acids in the diseased or absent terminal ileum, resulting in lithogenic bile.

The remaining hepatobiliary diseases—cirrhosis, chronic active hepatitis, granulomatous hepatitis, and amyloidosis—are relatively uncommon, occurring in 1% to 5% of patients with IBD.

### IV. CASE PATIENT 3

#### PRESENTATION

Patient 3 is a 62-year-old man who presents to the emergency department because of severe right lower quadrant abdominal pain and vomiting. He has not moved his bowels in 2 days. Normally, he has 2 to 4 loosely formed bowel movements per day, which he reports has been his pattern for the past few years. During the past year, he has lost 25 lb. He denies melena and hematochezia.

On physical examination, patient 3 appears uncomfortable. He is afebrile, his blood pressure is 120/80 mm Hg, and his pulse is 105 bpm supine. When he stands up, his blood pressure drops to 90/50 mm Hg and his pulse increases to 130 bpm. His mucus membranes are dry. Abdominal examination reveals high-pitched, tinkling bowel sounds and fullness in the right lower quadrant. Multiple skin tags are seen around his anus. Rectal examination reveals dark, heme-positive stool. Laboratory studies show a leukocyte count of 15,600 cells/µL, plasma hemoglobin of 10.4 g/dL, and increased platelet count. Electrolyte levels show a contraction alkalosis. Results of liver function tests are normal except for a serum albumin of 2.8 g/dL. Erythrocyte sedimentation rate is 85 mm/hr.

- **What is the most appropriate next step in patient 3’s evaluation?**
  
  A) Barium study of the small bowel  
  B) Computed tomography (CT) scans of the abdomen  
  C) Colonoscopy  
  D) Exploratory laparotomy  
  E) Abdominal ultrasound

#### Discussion

The correct answer is A. A small-bowel barium enema study is usually the first-line test in a patient with suspected Crohn’s disease. A typical radiograph demonstrates segmental terminal ileal narrowing, irregularity, and ulceration as well as skip lesions (sections of normal bowel interspersed between sections of diseased bowel). The mucosa often have a characteristic cobblestone appearance. A double-contrast radiologic study using both barium and air, such as an enteroclysis or an oral pneumocolon, may provide greater visualization of the...
diseased areas. CT scans and abdominal ultrasounds can provide noninvasive visualization of the abdomen and may show thickened loops of bowel; however, these tests are relatively nonspecific. In the absence of colonic disease, colonoscopy is of relatively little use because inflammation and narrowing of the terminal ileum may prevent intubation and visualization of the lumen. Occasionally, a patient having his first Crohn’s disease flare is misdiagnosed as having appendicitis and is taken to the operating room for a laparotomy.

Patient 3 illustrates the fact that Crohn’s disease as well as UC can present at any age. Multiple studies have indicated a bimodal distribution of the age of onset of these 2 diseases (Figure 3). The first mode occurs between ages 15 and 25 years, and the second appears between ages 50 and 80 years (most often near 70 years). An IBD diagnosis can be delayed in older patients because of several factors, including a blunted response to pain, the elderly patient’s fear of the medical system, and an overemphasis on cancer as the underlying cause of symptoms. Earlier studies suggesting that elderly patients had a poorer prognosis than younger ones were flawed by selection bias. More recent studies have shown that elderly persons have a more favorable prognosis and that their mortality from IBD may actually be lower.

**COMPLICATIONS OF CROHN’S DISEASE: OBSTRUCTION AND ABSCESS FORMATION**

Patient 3 undergoes a small-bowel barium enema study, which shows dilated loops of small bowel and near-complete obstruction of the terminal ileum. He is treated with intravenous fluids, and a nasogastric tube is placed to decompress the small bowel. He is started on mesalamine, intravenous methylprednisolone, and intravenous morphine sulfate for pain. On hospital day 3, he begins vomiting feculent material. His temperature increases to 39.2°C. Physical examination reveals clear lungs and a distended, diffusely tender abdomen. His abdominal pain is increased with flexion of his right hip against resistance. Urinalysis is unremarkable.

• What is the most likely cause of patient 3’s fever?
  A) Exacerbation of his Crohn’s disease
  B) Urinary tract infection
  C) Psoas abscess
  D) Aspiration pneumonia

**Diagnosis**

The correct answer is C. Patient 3 has a positive psoas (Cope’s) sign and fever indicative of a right psoas abscess. Abscesses in Crohn’s disease can be classified into 3 distinct categories: pericolic, psoas, and liver abscesses. Pericolic abscesses can occur anywhere in the abdomen but do so most often in the region of the sigmoid colon. They often present as a localized fluid collection because of transmural bowel inflammation and localized perforation. Typical symptoms include fever and abdominal pain, although corticosteroid therapy can often mask these symptoms. A psoas abscess usually arises on the right side as a result of ileitis from primary ileocolic disease or of recurrent disease at the site of an ileocolic anastomosis. Liver abscesses, which present with fever and right upper quadrant pain, are usually multifocal within the liver. A fluid collection demonstrated by either CT scan or abdominal ultrasound makes the diagnosis. Usually, CT scan is more...
useful in defining the extent of the fluid collection. Abdominal ultrasound is more sensitive for detecting liver abscesses than it is for psoas or pericolic abscesses.

A CT scan performed on patient 3 demonstrates dilated loops of small bowel proximal to the terminal ileum, thickened walls in the terminal ileum, and a fluid collection in the right psoas muscle.

- In addition to broad-spectrum antibiotics, what is the most appropriate intervention at this point?
  A) Surgical consultation for laparotomy
  B) CT-guided percutaneous drainage
  C) A magnetic resonance imaging (MRI) scan of the abdomen and retroperitoneum
  D) Colonoscopy

**Treatment**

The correct answer is A. Treatment of intra-abdominal abscesses depends on abscess location. Small pericolic fluid collections may respond to antibiotics and percutaneous drainage, but open surgical drainage with resection of the diseased bowel is often needed, especially for larger collections. Treatment of psoas abscesses usually involves ileocolic resection with end-to-side or end-to-end anastomosis and with open drainage of the abscess. Liver abscesses are usually treated with ultrasound-guided drainage of fluid collection and with intravenous antibiotics.

Patient 3’s small-bowel obstruction will also be cured by the surgery. Obstruction in Crohn’s disease usually develops because of transmural inflammation with submucosal fibrosis but can occasionally be precipitated by a large food bolus, as when a nut or pit becomes lodged in a severely narrowed section of small bowel. Symptoms of small-bowel obstruction from Crohn’s disease are similar to those seen in obstruction from other causes such as adhesions. Colicky pain and vomiting are typically the presenting symptoms; the vomitus may be feculent. Radiographic studies show the characteristic dilated loops of small bowel with air-fluid levels. A small-bowel barium study may show the “string sign” of Kantor, a term given to the characteristic narrowing of the terminal ileum. Because the obstruction is caused by fibrosis, surgery is often required to resolve symptoms but may be delayed until the acute attack is over.

**FOLLOW-UP OF PATIENT 3**

Patient 3 does well for 2 years, until he develops some purulent drainage from his laparotomy scar. A barium radiograph of his small bowel confirms the presence of a fistula from his neoterminal ileum—near the site of his anastomosis—to the skin.

**Fistula Formation**

A fistula is a communication between the bowel and any other epithelial surface and commonly occurs as a complication of Crohn’s disease. The prevalence of fistulae in Crohn’s disease is between 20% and 40%. Enterocutaneous fistulae, which tend to develop at the site of a previous surgery because of recurrent disease at the anastomosis, often present with pus or foul-smelling intestinal discharge. This drainage may collect under the skin and form an abscess. Enterocutaneous or enterocolic fistulae may also occur; these may be asymptomatic and found incidentally on barium studies. In other cases, colonic contents may track through a fistula to the small bowel, contaminating it and causing bacterial overgrowth that can cause malabsorption with weight loss and diarrhea. In patients with enterocutaneous fistulae, pain and diarrhea are usually caused by the underlying Crohn’s disease and not by the fistulae themselves. In women, colovaginal and rectovaginal fistulae can occur and present with foul-smelling vaginal discharge or sometimes with flatus and feces through the vagina if the fistula is large enough. Colovesicular or enterovesicular fistulae may occur and present with pneumaturia as well as recurrent urinary tract infections.

**Treatment**

Treatment of fistulous disease depends on the type of fistula and the symptoms experienced by the patient. The occurrence of enterocutaneous fistula at the site of a previous surgery is not an absolute indication to reoperate. However, many patients find the continual drainage of fluid through their abdominal wall to be intolerable and wish to have the fistula surgically repaired. Medical therapy is effective for enteroenteric fistula. Specifically, immunomodulatory agents and bowel rest, with supplemental total parenteral nutrition (TPN), have been shown to be effective in the short term. Fistulae tend to recur; however, after the TPN or immunomodulatory agent is stopped, ultimately requiring the patient to undergo surgery to achieve a more definitive cure. Surgery involves resecting the diseased section of the bowel. For small, minimally symptomatic rectovaginal fistulae, no treatment may be necessary. Although metronidazole may be useful for small rectovaginal fistulae, definitive treatment with surgery involving either primary closure, diverting colostomy, or abdominal perineal resection is usually required. Similarly, rectovesical or colovesical fistulae usually require surgical therapy to prevent kidney damage from urinary tract infections.
**V. CASE PATIENT 4**

**PRESENTATION**

Patient 4 is a 43-year-old woman with long-standing Crohn’s disease who presents to her primary care physician with profuse, watery diarrhea that has been ongoing for the past several months; weight loss; generalized fatigue; and lethargy. She has been using loperamide and limiting the amount of fruits and vegetables she eats to decrease her diarrhea.

Patient 4 has a 25-year history of Crohn’s disease of the small bowel. She has undergone multiple resections of her small bowel, secondary to Crohn’s disease complications. In the past, she has been treated with steroids, azathioprine, and mesalamine, with varying results. Currently, she is taking no medications other than the loperamide.

- **Patient 4 is at risk for which of the following nutritional deficiencies?**
  - A) Protein deficiency
  - B) Vitamin B12 deficiency
  - C) Folic acid deficiency
  - D) Zinc deficiency
  - E) All of the above

**Nutritional Deficiencies**

The correct answer is E. Malnutrition is a common finding in Crohn’s disease, with a prevalence as high as 80% for certain nutritional deficiencies (Table 6) but is somewhat less common in UC. Malnutrition results from several causes such as inadequate oral intake, malabsorption, and elevated nutritional requirements. Patients may reduce their intake secondary to nausea, vomiting, or in the belief that the reduction may improve their Crohn’s disease. Malabsorption may develop from loss of brush-border enzymes, impaired enterohepatic circulation of bile salts secondary to disease in the terminal ileum, or loss of bowel surface area as a result of Crohn’s disease itself or as a result of small-bowel resections. Crohn included the physical findings of malnutrition (namely muscle wasting and weight loss) in his original description of regional enteritis, and indeed, these clinical features are recognized today as a common presenting signs in Crohn’s disease. Important clinical consequences may result from malnutrition, including growth retardation in childhood, impaired wound healing, and depressed cellular immunity.

**Iron deficiency** is very common in both Crohn’s disease and UC. This condition results from gastrointestinal bleeding rather than from decreased iron intake, but those who have decreased oral iron intake (eg, vegetarians) and those with increased blood losses (eg, menstruating women) are at greater risk. Clinical signs include anemia, angular cheilitis, brittle and occasionally spoon-shaped nails, and a smooth tongue due to loss of papillae. Treatment may be difficult because many patients find that iron supplements exacerbate their abdominal pain or diarrhea.

**Vitamin deficiencies** are also common and include the fat-soluble vitamins pyridoxine, thiamine, riboflavin, and folic acid. Studies of patients with Crohn’s disease have shown normal absorption of the fat-soluble vitamins A and E, however, deficiencies of both of these vitamins as well as vitamin D have been observed. Decreased levels of vitamin A are believed to be associated with increased protein catabolism, resulting in decreased serum levels of retinol-binding protein. Inadequate intake of vitamin A may also play a role. Vitamin D deficiency is seen in undernourished patients with Crohn’s disease. Osteomalacia and even osteoporosis caused by vitamin D deficiency are fairly common findings in these patients and are often exacerbated by corticosteroids. Thus, fat-soluble vitamin deficiencies represent an important aspect of nutritional deficiency in Crohn’s disease.

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**Table 6. Prevalence of Deficiencies in Inflammatory Bowel Disease**

<table>
<thead>
<tr>
<th>Sign</th>
<th>Crohn’s Disease</th>
<th>Ulcerative Colitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight loss</td>
<td>65–76</td>
<td>18–62</td>
</tr>
<tr>
<td>Growth retardation</td>
<td>40</td>
<td>—</td>
</tr>
<tr>
<td>Hypoalbuminemia</td>
<td>25–80</td>
<td>25–50</td>
</tr>
<tr>
<td>Anemia</td>
<td>60–80</td>
<td>—</td>
</tr>
<tr>
<td>Iron</td>
<td>39</td>
<td>81</td>
</tr>
<tr>
<td>Folate</td>
<td>54</td>
<td>36</td>
</tr>
<tr>
<td>Vitamin B₁₂</td>
<td>48</td>
<td>5</td>
</tr>
<tr>
<td>Calcium</td>
<td>13</td>
<td>—</td>
</tr>
<tr>
<td>Magnesium</td>
<td>14–88</td>
<td>—</td>
</tr>
<tr>
<td>Potassium</td>
<td>6–20</td>
<td>—</td>
</tr>
<tr>
<td>Zinc</td>
<td>40–50</td>
<td>—</td>
</tr>
</tbody>
</table>

Water-soluble vitamins and minerals are often deficient in Crohn’s disease patients as well. Vitamin B₁₂ (cyanocobalamin) deficiency arises because of malabsorption in terminal ileum disease or in those who have had terminal ileum resection. Bacterial overgrowth because of advanced age or fistulae can also cause vitamin B₁₂ deficiency. Folate deficiency is usually secondary to decreased intake of fruits and vegetables but may also be caused by medications such as sulfasalazine, which can cause folic acid malabsorption. Megaloblastic anemia and glossitis may result from lack of either vitamin B₁₂ or folate. The glossitis of B₁₂ deficiency is characterized by fissuring, whereas the glossitis of folate deficiency is characterized by nodularity. After iron, zinc is the second most common mineral deficiency seen in Crohn’s disease, resulting from malabsorption in patients with massive diarrhea, short bowel secondary to surgery, or enteric fistulae. Clinical features of zinc deficiency are bullous dermatitis, impaired wound healing, neurosensory disorders, and diarrhea.²⁹ Magnesium deficiency is also common in patients with IBD. Urinary levels of magnesium are a more reliable indicator of deficiency than are serum levels. Treatment of vitamin and mineral deficiencies involves treating the underlying cause of the deficiency as well as replacing the deficient nutrient.

**FURTHER PRESENTATION OF PATIENT 4**

Patient 4 is quite thin. She is 66 inches (168 cm) tall and weighs 90 pounds (41 kg). Her body mass index is 14.6. Her conjunctiva are pale; mild cheilitis is seen around her mouth. Her tongue is depapillated and erythematous, with some nodularity. An abdominal examination reveals multiple surgical scars but is otherwise unremarkable. Her extremities are notable for muscle wasting.

• Which of the following initial interventions would NOT be appropriate for patient 4?
  A) Referral to a nutritionist for diet instruction
  B) Elemental diet administered orally or by nasogastric tube
  C) Total parenteral nutrition
  D) Treatment with high-dose mesalamine and either corticosteroids or azathioprine

**Treatment**

The correct answer is C. Patient 4 has protein and calorie malnutrition as well as vitamin B₁₂ and folate deficiency, as evidenced by her glossitis, cheilitis, and pale conjunctiva. Her condition is likely associated with Crohn’s disease in the small bowel. She may also have short-bowel syndrome from her multiple surgeries. In the past, many people had large sections of bowel removed in the mistaken belief that these procedures would be curative. Today, surgeons try to preserve as much bowel as possible. One way to do so is to perform a stricturoplasty on short strictures rather than resecting them, thereby relieving the stricture while preserving the bowel surface area. However, some patients have such severe small-bowel disease, necessitating removal of much of the small bowel, that they develop malabsorption from short-bowel syndrome.

Patient 4 may benefit from dietary instruction by a licensed nutritionist. Some patients avoid certain foods because they believe it will make their symptoms worse. They may have been told by well-meaning friends, family members, or health care professionals to do so. In the past, bowel rest was a treatment for both UC and Crohn’s disease. However, randomized trials have not shown that bowel rest provides a benefit in Crohn’s disease.²⁹

In the late 1960s, elemental diets consisting of fat-free, residue-free, amino acid-based formulas were introduced. These solutions, devoid of intact proteins, are easily absorbed in the jejunum, an area that is typically spared by Crohn’s disease. In randomized clinical trials, elemental diets have been shown to be equivalent to corticosteroids in treating active Crohn’s disease.³⁴,³⁵ In a separate trial, elemental diets have demonstrated a higher level of improvement in activity index, body weight, serum albumin, and inflammatory signs (ie, erythrocyte sedimentation rate) when compared with steroids.³⁶ One drawback to elemental diets is their unpleasant taste, which can be overcome by giving the formula via nasogastric tube. Total parenteral nutrition (TPN) is also beneficial in IBD but has the drawbacks of high cost and a high rate of complications. Therefore, TPN is reserved for patients who either fail or cannot tolerate enteral feeding.

**Nephrolithiasis**

Patient 4 is treated with mesalamine and an elemental diet. She begins to feel better and gains weight. After 4 months, her serum albumin level is normal. She is instructed to stop the elemental diet and resume a normal diet.

Two months later, patient 4 develops an influenza-like illness with fevers, nausea, and vomiting. Two days after symptom onset, she develops severe left-sided flank pain and hematuria. She goes to the emergency department, where she undergoes a CT scan of abdomen and pelvis, which demonstrates a calcified stone at the left ureterovesicular junction and hydronephrosis on the left side.
What is the most likely cause of patient 4’s nephrolithiasis?

A) Primary hyperparathyroidism secondary to hypercalcemia
B) Hyperoxaluria
C) Hyperuricemia
D) Idiopathic hypercalciuria

Discussion

The correct answer is B. Patients with Crohn’s disease are prone to develop calcium oxalate kidney stones. Rapid intestinal transit in these patients results in increased absorption of dietary oxalate followed by hyperoxaluria. Predisposing factors include terminal ileal resection, dehydration, fever, and loss of alkaline fluid from the gut, which causes urinary acidification. Unlike Crohn’s disease, UC is not an independent risk factor for developing nephrolithiasis.

VI. CASE PATIENT 5

PRESENTATION

Patient 5 is a 35-year-old man with a long-standing history of Crohn’s colitis who presents to his primary care physician with abdominal pain, diarrhea, and an open sore near his anus that drains pus and stool. He is taking sulfasalazine and ibuprofen (400 mg, 4 times per day) for bilateral knee pain.

On physical examination, his vital signs are normal; his pulse and blood pressure do not change substantially with position. His abdomen is nontender, without rebound or guarding. His perianal skin appears to have a fistulous opening with a small amount of discharge. His stool is brown, and guaiac testing of the stool reveals occult blood. His joint examination is remarkable for some mild swelling of the knees and ankles; however, no warmth or effusions are apparent. A radiograph of the knees is unremarkable.

What is the most likely cause of patient 5’s knee pain?

A) Rheumatoid arthritis
B) Osteoarthritis
C) Septic arthritis
D) Colitic arthritis

Discussion

The correct answer is D. Rheumatoid arthritis (RA) typically affects the metacarpal phalangeal joints, although it may also affect the knees. Patient 5’s examination, however, does not show an inflammatory arthritis. In the absence of joint effusion and fever, both RA and septic arthritis would be unlikely. In addition, bilateral knee involvement would be unusual for septic arthritis. Osteoarthritis is a possibility, but the patient is rather young to develop this disease. Also, osteoarthritis of the knee would be expected to show crepitus and osteophytes on radiograph. The fact that patient 5 has known IBD and lacks any findings for other common forms of arthritis makes the diagnosis of colitic arthritis most likely. Anecdotal evidence further suggests that nonsteroidal anti-inflammatory drugs exacerbate Crohn’s disease.

Joint symptoms are the most common extraintestinal manifestation of IBD. Two types of arthritis are associated with the disorder: colitic arthritis and ankylosing spondylitis. Colitic arthritis is a noninflammatory migratory arthritis, usually affecting 6 or fewer joints such as the ankles, knees, hips, wrists, and elbows. This condition is more common in Crohn’s colitis than in UC and is uncommon in Crohn’s disease of the small bowel. The course of colitic arthritis tends to parallel that of IBD; treatment of the underlying IBD with corticosteroids often improves the arthritis.

In contrast, ankylosing spondylitis develops independently of the course of IBD. Ankylosing spondylitis, which presents with morning stiffness, low back pain, and stooped posture, is 30 times more common in patients with UC than in the general population. The association between ankylosing spondylitis and human leukocyte antigen–B27 (HLA-B27) is well known. Although no increased incidence of HLA-B27 is seen in patients with IBD, most patients with both UC and ankylosing spondylitis are HLA-B27 positive. Management of ankylosing spondylitis is difficult because it does not respond to treatment of underlying IBD. Physical therapy can be a useful modality.

Sacroiliitis is also a common finding in patients with UC and can be seen in conjunction with ankylosing spondylitis or by itself. Sacroiliitis, which occurs in approximately 12% to 15% of UC patients, may be asymptomatic or associated with low back pain.

PERIANAL COMPLICATIONS OF CROHN’S DISEASE

Patient 5 has a perianal fistula. Approximately 33% of patients with Crohn’s disease will develop anal or perianal complications in their lifetime. These complications include perianal skin tags, anal fissures, perianal abscesses, pararectal abscesses, and rectal-cutaneous fistulae. Perianal skin tags, the most common finding in Crohn’s disease, tend to be bluish or violaceous and more rigid than hemorrhoids. The differential diagnosis of perianal skin tags includes sexually transmitted
disease, tuberculosis, and malignancy. Most skin tags are asymptomatic and do not require treatment.

Also commonly found in Crohn’s disease are anal fissures, which resemble common idiopathic fissures but tend to be less painful. Asymptomatic fissures will usually heal with medical management, whereas more painful ulcers may require surgical treatment. However, surgical management is controversial because it may result in fecal incontinence and a nonhealing wound.

Perianal abscesses are yet another frequently occurring complication. These abscesses may be complicated and have tracts that penetrate deep into the pararectal area. MRI or transrectal ultrasound can be used to define the extent of the abscess. Treatment of acute disease involves urgent incision and drainage under general anesthesia; alternatively, chronic perianal abscesses with low-grade sepsis may respond to metronidazole therapy.

Perianal fistulae, perhaps some of the most unpleasant manifestations of Crohn’s disease, can occur as a single discharging sinus (as in patient 5) or as multiple fistulous openings in the perianal skin. Further, fistulae can track deeply within the perineum and form openings in the labia and scrotum. As in perianal abscesses, MRI or transrectal ultrasound may be used to define the extent of the fistulae. Treatment involves both medical and surgical therapy, aiming more at alleviating symptoms rather than at completely eradicating the fistulae. The ultimate goal is to maintain anal sphincter function.

Medical therapy for perianal fistulae involves treating the underlying IBD as well as using adjunctive antibiotic agents such as metronidazole or ciprofloxacin. Surgical treatment involves curettage of tracks and deployment of plastic setons to keep fistulae open so that they may drain. Open surgery is a last resort, used only when medical and local surgery fail. Radical surgery may be necessary when the anal sphincter mechanism is compromised.

VII. SUMMARY POINTS

- The disorders that comprise inflammatory bowel disease (IBD) are Crohn’s disease and ulcerative colitis (UC).
- An inflammatory disease of the mucosa only, UC always involves the rectum and proceeds proximally in a continuous fashion.
- Crohn’s disease causes transmural inflammation and can affect any portion of the gastrointestinal tract from the mouth to the anus.
- Although the diseases may present at any age, their incidence has a bimodal age distribution, with an initial peak between ages 15 and 25 years and a later peak between ages 50 and 80 years.
- UC typically presents with bloody diarrhea, tenesmus, and cramping abdominal pain.
- Crohn’s disease presents with diarrhea (usually non-bloody), weight loss, fatigue, and muscle wasting.
- Extraintestinal manifestations of IBD include oral, cutaneous, ocular, and rheumatologic problems.
- IBD treatment involves a combination of medical and surgical therapies. Drugs used to treat IBD include 5-aminosalicylate drugs, immunosuppressive agents, antibiotics, and biologic response modifiers.
- Complications of UC include toxic megacolon, colorectal cancer, and primary sclerosing cholangitis.
- Complications of Crohn’s disease include malnutrition, malabsorption, bowel obstruction, fistula formation, abscess formation, cholelithiasis, and nephrolithiasis.

REFERENCES


Chapter 2—Common Dermatoses: Case Studies

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1. PSORIASIS

INTRODUCTION

Psoriasis is a chronic inflammatory skin disease that commonly presents as red, well-defined plaques with silvery-white scaling, which are most often located on the elbows, knees, and scalp. The surface area affected by psoriasis may be small or may involve almost the entire body. In the United States, 1% to 2% of the population has psoriasis, with an equal incidence in men and women. Variants of psoriasis are shown in Table 7, including psoriasis vulgaris or plaque psoriasis (which is the most common form), pustular psoriasis, guttate psoriasis, erythrodermic psoriasis, palmar-plantar psoriasis, inverse psoriasis, and psoriatic arthritis. The etiology of the disorder is unknown but could be related to increased epidermal proliferation associated with immune system activation. Patients with psoriasis often have itching. For some patients, the disease has a major adverse effect on their quality of life.

CASE PATIENT 6 PRESENTATION

Patient 6 is a healthy 24-year-old man who presents to his primary care physician with a 4-month history of rash on his scalp, elbows, knees, and upper gluteal cleft. The mildly pruritic rash was first noticed during the past winter and has gotten worse despite the use of over-the-counter (OTC) topical treatment. No exacerbating factors are noted. Patient 6 reports that he can tolerate the itching, but he is especially concerned about his appearance and wonders if he can spread the rash to others. He reports no family history of a similar condition and is a graduate student at a state college.

- What are the typical presenting features of psoriasis?

Discussion

The history for patient 6 is remarkable only for a somewhat itchy rash of gradual onset. There was no family history of a similar condition and no exacerbating factors were noted. Psoriatic lesions are often indolent and may be present months before a patient seeks medical care. Occasionally, the onset of lesions can be sudden (eg, in acute guttate psoriasis and generalized pustular psoriasis). Pruritus is common, particularly in the scalp. Constitutional symptoms are usually not present except in psoriatic arthritis, acute pustular psoriasis, and erythrodermic psoriasis. A positive family history is present in only approximately 33% of cases. Sometimes physical trauma (Koebner’s phenomenon) can induce lesions at new sites or adjacent to current sites. Streptococcal infection has been associated with guttate psoriasis, and physical or emotional stress may be a factor in psoriasis flares. HIV has been diagnosed after new-onset psoriasis. The mnemonic “PLAN B” can be used to remember drugs that have been associated with psoriasis flares: Prednisone (systemic), Lithium carbonate, Antimalarials, Nonsteroidal anti-inflammatory agents, and Beta-blockers.

PHYSICAL EXAMINATION

On physical examination, patient 6 is noted to have well-defined red plaques with silvery, thick scales on his elbows, knees, upper gluteal cleft, and postauricular scalp. He also has small pits involving the nail plates. No joint tenderness is observed. These skin findings are consistent with plaque psoriasis.

- What are common signs and symptoms of psoriasis?
- What is included in the differential diagnosis?

Discussion

Psoriatic plaques are often round or oval but can be linear or coalesce into polycyclic forms. Distribution can involve a single lesion or, more commonly, areas on the elbows, knees, and scalp. Removal of scales on the lesion may demonstrate punctate bleeding from capillary loops in the dermal papillae (Auspitz sign). Often, torso lesions are observed (Figure 4), and in rare cases whole-body or erythrodermic psoriasis occurs. Nail findings
are common; pitting is most frequently seen, as in patient 6’s case. Other nail changes include onycholysis (separation of the nail plate from the underlying nail bed), “oil drop” lesions (yellow areas in the nail bed), and subungual debris (Figure 5). Rarely, psoriasis presents only in the nails.

Diagnosis can often be made clinically by the appearance of a typical psoriasis lesion in common areas of distribution. Although usually not necessary, a skin biopsy may help confirm the diagnosis because other dermatoses can have a psoriasiform pattern. Differential diagnosis may also include other papulosquamous inflammatory skin diseases such as nummular dermatitis, pityriasis rosea, lichen planus, pityriasis rubra pilaris, and secondary syphilis.

**TREATMENT**

Psoriasis is a chronic condition, and eradication of all lesions (ie, total clearance) is unrealistic for most patients. Thus, the goal for treatment should be to control the psoriatic process as much as possible using a combination of topical agents, ultraviolet light, and/or systemic agents. Because many therapies exist, it may be most effective to use a step-ladder approach, in which the most simple, inexpensive, and least toxic agents are tried before more effective but often more costly and potentially more toxic agents are administered. OTC emollients, especially ointments like petroleum jelly, can be used after showers or baths to help soften and remove plaques, whereas OTC tar preparations can reduce pruritus and help to thin plaques.

Topical prescription corticosteroids are effective, particularly the medium-potency (triamcinolone) to high-potency (clobetasol) preparations. Total body application of triamcinolone ointment using “wet wraps” may be needed for erythrodermic psoriasis. Ointment-based (greasy) preparations of corticosteroids are usually more effective, but creams are often more cosmetically acceptable. Twice-daily application is optimal for most preparations. The most common side effect from topical corticosteroids is skin atrophy, which can be minimized by applying agents only to psoriatic areas and by using such therapy only during flares. A reasonably safe, moderately effective but very expensive topical therapy that can be used on a chronic basis is the vitamin D3 analogue calcipotriene (Dovonex), which is approved by the US Food and Drug Administration (FDA) for this indication. Like other preparations, twice-daily application produces the best results, but some patients respond adequately to daily application. Older preparations containing anthralin are occasionally used, although irritation can occur. Tazarotene, a topical retinoid, is occasionally used in conjunction with topical corticosteroids to reduce irritation and improve results. Rated as Pregnancy Category X, tazarotene should not be used in women of childbearing age.

Ultraviolet (UV) light often improves psoriasis; many patients note improvement in their condition during the summer. Use of light units in tanning salons or home units are beneficial for some patients. Office-based phototherapy with UVB or UVA light with psoralen (PUVA) can be effective but inconvenient. The physician must remain aware of increased risk of cutaneous malignancy with chronic phototherapy.

In addition to these treatments, systemic medications can be effective and are indicated for more severe forms of psoriasis and psoriatic arthritis. Most
agents have potentially significant toxicities and require routine monitoring. Methotrexate is the “gold standard” and is given orally on a weekly basis; it is FDA approved for psoriasis. However, biopsies are needed to detect liver toxicity, which can cause permanent fibrosis and liver failure. A baseline liver biopsy and then additional biopsies each time a total dose of 1.5 g of methotrexate has been given should be obtained. Cyclosporine is approved by the FDA for severe psoriasis and is usually administered for several months to induce a remission. Close monitoring is required to prevent the development of complications such as hypertension and renal toxicity. Acitretin (Soriatane), a systemic retinoid, has been most effective in pustular psoriasis and psoriasis with palm and sole involvement. Acitretin is FDA approved for severe psoriasis, including erythrodermic and generalized pustular types. Side effects are common and include dry skin, cracked lips, and hypertriglyceridemia. Because of teratogenic effects, pregnancy must be avoided in women of childbearing age who are taking this medication.

II. DERMATOPHYTE INFECTIONS

INTRODUCTION

Dermatophytes are parasitic fungi that feed on skin keratin and can cause infections that are sometimes referred to as tinea or “ringworm.” Sources of dermatophytes include human (anthrophilic), animal (zoophilic), or soil (geophilic). The groin, feet, and toenails are preferred areas for the growth of these organisms, although infection can occur on most skin surfaces (including hair and nails). Infections by Trichophyton species are most common, and Epidermophyton and Microsporum species are seen with some frequency. In general, these infections appear clinically as one or more scaly red patches that are often annular or serpiginous and are associated with itching, burning, or both. Maceration and vesiculation can occur, especially in occluded areas.

CASE PATIENT 7

Patient 7 is an elderly man with chronic lung disease who has been taking prednisone, 10 mg daily, for several years. He presents to his internist with large, confluent, red scaly patches on his buttocks and lower mid-back. On physical examination, well-defined borders of the rash are evident as well as scale and central clearing in some areas, as if the rash were moving outward and healing in the center. A similar rash with well-defined borders is discovered in the patient’s groin bilaterally (Figure 6). The scrotum is uninvolved, and the toenails are dystrophic with extensive subungual debris. Although his condition is not causing serious health problems, patient 7 is nevertheless concerned about the rash and associated itching.
DISCUSSION
• What should be included in the differential diagnosis of patient 7?

Diagnosis
Classic lesions are not always seen in dermatophyte infections; thus, the differential diagnosis should include allergic or irritant contact dermatitis, atopic dermatitis, granuloma annulare, discoid lupus, erythema migrans, and other dermatoses. Scrotal involvement rarely occurs in dermatophyte infections as it does in Candida, which usually causes a beefy red rash with satellite papules. A diagnosis can easily be made by scraping the rash border with a #15 blade to remove scale containing the dermatophyte. Potassium hydroxide (KOH) is added to the slide to help dissolve the keratinocytes and to accentuate translucent septate-branching, thread-like hyphae characteristic of dermatophytes, which are easily visualized by a microscope under low power. This test is performed on patient 7 and confirms a diagnosis of dermatophyte infection.

• What is the best way to treat patient 7’s rash?

Treatment
Although topical antifungals may clear dermatophytes from the skin and improve the rash, infected toenails and feet may cause reinfection. A 3-month course of oral terbinafine (250 mg daily) will rapidly clear the skin and over 6 to 12 months will usually clear the toenails as new nails grow in; this indication has been approved by the FDA. Terbinafine, an allylamine antifungal that is fungicidal, disrupts the fungal cell membrane by inhibiting squalene epoxidation. Although adverse hematologic and hepatic effects are rare, laboratory monitoring should be performed, including a complete blood count and liver function tests at baseline and after 4 weeks of beginning treatment.

Success has also been achieved with itraconazole, administered in three 1-week cycles of 200-mg doses twice daily every month for 3 months. However, itraconazole has only been approved by the FDA for a dose of 200 mg daily for 12 weeks. Itraconazole, a triazole antifungal that is fungistatic, works by inhibiting fungal cytochrome p450 isoenzymes. Substantial drug-drug interactions can occur with this treatment, especially between itraconazole and medications metabolized by cytochrome p450 enzymes. Therefore physicians should carefully review other medications that the patient is taking to avoid drug interactions.

Chronic prednisone and other forms of immunosuppression may predispose patient 7 to recurrent tinea infections of the skin or nails after antifungal therapy is completed. Corticosteroid treatment may necessitate repeated courses of antifungal therapy. Prophylactic use of antifungal creams or sprays about once a week may help to prevent recurrences in all patients who have had dermatophyte infections.

CASE PATIENT 8
Patient 8 is a 48-year-old woman with diabetes who presents with a red, edematous, tender left lower leg and foot. She is then quickly admitted for her third hospitalization in the past 2 years for intravenous cefazolin to treat cellulitis of this limb. No evidence of diabetic ulcers or peripheral neuropathy is observed, and patient 8 has no history of deep venous thrombosis in either leg. Physical examination reveals fissuring and maceration between the fourth and fifth digit on her left foot.

DISCUSSION
• How does patient 8’s medical history affect her diagnosis?

Diagnosis
Patient 8’s history is pertinent for recurrent cellulitis. Searching for a portal of entry for bacteria may be useful in preventing future infection. The fissuring and maceration between the toes caused by tinea provided a recurrent entry point for infection. A KOH preparation confirms patient 8 has tinea.

• What is the most appropriate treatment for patient 8’s infection?

Treatment
Interdigital tinea will usually respond to topical antifungal agents. Terbinafine cream is available OTC and is fungicidal. The azole antifungals, which are fungistatic but clinically effective, are reasonable alternatives. In addition, keeping the feet dry by wearing absorbent socks and less occlusive footwear is important to decrease the recurrence of tinea pedis infections.

CASE PATIENT 9
Patient 9 is a healthy 44-year-old man who presents to his family physician with a 6-week history of rash on his left wrist. During the visit, his physician notes an oval-shaped, red scaly patch approximately 3 × 4 cm that looks like a fungal infection and prescribes nystatin cream to be used twice daily. After 4 weeks, patient 9 reports no improvement in his condition. The physician then prescribes Lotrisone cream (a combination of
clotrimazole and betamethasone), which rapidly improves the patient’s itching but does not resolve the rash. Because the surface area involved is small, patient 9 is able to use the original prescription of Lotrisone cream for 6 months, after which he calls his family physician for a refill. The physician schedules a re-examination, and several firm, red deep nodules are found in the area of the previous rash (Figure 9). Patient 9 has a job taking care of livestock on a farm.

- Why did patient 9’s rash not respond to treatment?

DISCUSSION

History and Physical Examination

The history of patient 9’s rash strongly suggests a dermatophyte infection. His work with livestock, a source of zoophilic dermatophytes, would have exposed him to these organisms. Moreover, the fact that nystatin was ineffective against his rash and that Lotrisone cream, with its combination of the potent corticosteroid betamethasone and the antifungal clotrimazole, rapidly improved the itching but did not clear the rash suggests that dermatophytes are the source of the patient’s problem. Dermatophytes only infect epidermis; in this case, the epidermis that surrounds the hair follicle was deep enough to prevent full penetration of the antifungal, thus resulting in the red deep nodules noted on physical examination.

The characteristic rash can help differentiate tinea from localized eczema or dermatitis. Tinea often produces an annular patch with a well-defined border and central clearing, whereas nonspecific or irritant dermatitis and allergic contact dermatitis often cause less well-defined patches or plaques. A diagnosis of tinea perifolliculitis, or Majocchi’s granuloma, is confirmed by a small-punch biopsy in patient 9.
• What is the best treatment for patient 9’s condition?

**DISCUSSION**

**Treatment**

As previously mentioned, tinea perifolliculitis may not respond to topical antifungals because these agents do not penetrate deeply into hair follicles. In such cases, oral terbinafine (250 mg daily for 2 to 4 weeks) is effective; however, it has not been approved by the FDA for this indication. Although effective against Candida, nystatin is ineffective against dermatophytes. The use of combination steroid and antifungal preparations (eg, Lotrisone) should be avoided because this “shotgun” approach may prevent diagnosis and prolong a patient’s infection. Additionally, steroids may temporarily improve tinea symptoms and may allow infections to persist; thus, steroid therapy should be reserved for steroid-responsive nondermatophyte dermatitis. Topical antifungals should be used for superficial KOH-positive dermatophyte infections, and oral antifungals should be used for deeper KOH-positive, culture-confirmed or biopsy-confirmed tinea perifolliculitis.

**III. INDICATIONS FOR REFERRAL TO A DERMATOLOGIST**

Internists can often treat patients with psoriasis or dermatophyte infections. However, a referral to a dermatologist is suggested when the following conditions are present.

**PSORIASIS**

• Localized psoriasis unresponsive to medium-potency topical corticosteroids
• Generalized psoriasis requiring ultraviolet light therapy or systemic therapy
• Erythrodermic psoriasis or pustular psoriasis

**DERMATOPHYTE INFECTIONS**

• Rash unresponsive to topical treatments
• Suspected tinea that is unresponsive to topical antifungal agents

**IV. SUMMARY POINTS**

**PSORIASIS**

• Psoriasis is a chronic skin disease most often presenting as red plaques with silvery-white scales.

• Plaque psoriasis is the most common form of the disorder. Other types include pustular, guttate, palmar-plantar, inverse, and erythrodermic psoriasis. Occasionally, psoriasis can be exacerbated by drugs, infections, and other stressors.

• In most cases, a diagnosis can be made clinically. Differential diagnosis includes nummular dermatitis, pityriasis rosea, lichen planus, pityriasis rubra pilaris, and secondary syphilis.

• Treatment may consist of topical emollients, topical corticosteroids, ultraviolet light therapy, and systemic immunosuppressive agents. Erythrodermic psoriasis can require hospital admission for “wet wraps” and systemic treatment. The goal of therapy is to control skin symptoms and lesions.

**DERMATOPHYTE INFECTIONS**

• Dermatophytes (including Trichophyton, Epidermophyton, and Microsporum species) cause skin infections referred to as tinea or “ringworm.” Keratin in the skin, hair, or nails can be affected; however, the sites most favored by these organisms are the feet, groin, and nails.

• Most tinea infections are self-limited, but itching and burning can be bothersome. Sometimes, tinea infections can provide a portal of entry for other infections, especially in diabetic patients or those whose immune systems are compromised.

• The rash caused by tinea often has a well-defined serpiginous or annular border with central clearing. Diagnosis can usually be made in the office by potassium hydroxide preparation.

• Topical terbinafine or azoles are effective for superficial dermatophyte infections. Oral terbinafine or itraconazole is needed for nail involvement or deeper skin involvement (eg, tinea perifolliculitis). Nystatin is effective for Candida but ineffective for dermatophytes. Preparations that combine antifungals and steroids should not be used because they may predispose the patient to chronic tinea perifolliculitis.

**BOARD REVIEW QUESTIONS**

Choose the single best answer for each question.

1. Which of the following is NOT associated with psoriasis exacerbations?
   A) Medications
   B) Infections (eg, streptococcal and HIV)
   C) Sunlight exposure
   D) Physical illness or other stressors
2. **What method is commonly used to diagnose psoriasis?**
   A) Skin biopsy  
   B) History, including family history  
   C) Physical appearance and location of typical psoriasis plaques  
   D) Response to usual treatment

3. **What is NOT included in the differential diagnosis of psoriasis?**
   A) Nummular dermatitis  
   B) Pityriasis rosea  
   C) Secondary syphilis  
   D) Stevens-Johnson syndrome

4. **Treatment of psoriasis may include all of the following EXCEPT:**
   A) Tetracycline  
   B) Cyclosporine  
   C) Topical corticosteroids  
   D) Coal tar preparations

5. **Dermatophyte infections usually do NOT involve:**
   A) The scrotum  
   B) The torso  
   C) The nails  
   D) The feet

6. **What is the most practical office method to accurately diagnose a dermatophyte infection?**
   A) History of family member with similar rash  
   B) Appearance of the rash  
   C) Potassium hydroxide preparation  
   D) Culture

7. **Which of the following is NOT included in the differential diagnosis of dermatophyte infection?**
   A) Contact dermatitis  
   B) Atopic dermatitis  
   C) *Candida*  
   D) Polymorphous light eruption

8. **Topical treatment for dermatophytes includes all of the following EXCEPT:**
   A) Nystatin  
   B) Terbinafine  
   C) Miconazole  
   D) Ketoconazole

**REFERENCES**


**ANSWERS**


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