A 41-year-old woman with a 4-year history of polymyositis with lupus features has had constant rectal pain for 4 months. She has not noticed any factors that either aggravate or relieve the pain. The patient complains of intermittent constipation (but no discharge or rectal bleeding), generalized weakness and malaise for the past 2 months, a low-grade fever for the past month, and a 4.1-kg (9-lb) weight loss over the past 6 weeks. She denies night sweats or chills, anorexia, vision problems, drug allergies, and tobacco or alcohol use.

A 41-year-old woman with a 4-year history of polymyositis with lupus features has had constant rectal pain for 4 months. She has not noticed any factors that either aggravate or relieve the pain. The patient complains of intermittent constipation (but no discharge or rectal bleeding), generalized weakness and malaise for the past 2 months, a low-grade fever for the past month, and a 4.1-kg (9-lb) weight loss over the past 6 weeks. She denies night sweats or chills, anorexia, vision problems, drug allergies, and tobacco or alcohol use. She has a family history of hypertension and diabetes mellitus. For the past 4 years, she has been receiving immunosuppressive therapy: currently, 20 mg of prednisone and 150 mg of azathioprine per day. She also takes cimetidine and a multivitamin supplement. The patient is well oriented to time, place, and person. Heart rate is 102 beats per minute; temperature, 37.7°C (99.8°F); blood pressure, 110/66 mm Hg; and respiration rate, 18 breaths per minute. Rectal examination reveals moderate tenderness with heme-negative stool. The remainder of the examination is normal. White blood cell count is 2600/?L (normal, 4000 to 11,000/?L), with 87% segmented forms, 7.9% lymphocytes, 4.9% monocytes, and 0.1% basophils. Hemoglobin level is 9 g/dL; hematocrit, 33.9%; and platelet count, 184,000/?L (normal, 150,000 to 450,000/?L). Serum electrolyte levels and renal and liver function test results are normal. Results of a tuberculin test and a chest radiograph also are normal. Flexible sigmoidoscopy shows erythema with ulcers in the sigmoid colon and rectal area and internal hemorrhoids (Figure 1). A biopsy specimen of the sigmoid colon reveals multifocal viral inclusions in epithelial as well as endothelial cells consistent with cytomegalovirus (CMV) colitis (Figure 2). A 5-week course of intravenous ganciclovir is started. Two weeks after discharge, all symptoms have resolved.

CMV INFECTION: AN OVERVIEW

Although more than 40% of the general population is infected with CMV, symptoms are typically seen only in children. The infection is generally asymptomatic in immunocompetent adults. Clinically significant disease occurs in patients with defective cell-mediated immunity, particularly those who have undergone transplantation, have AIDS, or are receiving chemotherapy. Most published cases of CMV infection in immunocompetent patients are associated with malnutrition, long-term corticosteroid use, or alcoholism. The prevalence of CMV infection is high in the homosexual community. In the United States, CMV can be isolated in 81% of homosexual men older than 35 years. These persons have elevated CMV titers, but clinical evidence of disease is relatively rare. CLINICAL FEATURES Primary CMV infection in immunocompetent persons is usually asymptomatic; however, it may result in a
immunocompromised persons, such as solid organ or bone marrow transplant recipients and patients with AIDS. These infections may involve the pulmonary, hepatic, ophthalmic, or GI system. GI involvement is serious and often associated with diarrhea, abdominal pain, hemorrhage, and even perforation. Pneumatosis intestinalis, myenteric plexus involvement with intestinal pseudo-obstruction, and hemorrhoids are infrequently noted. As in this case, rectal pain may be the only symptom of CMV infection. According to one study, 67% of GI infections were found in the colon, which was the most common site of infection. Other GI sites were the esophagus (20%), rectum (7%), and small bowel (3%).

**DIAGNOSIS** CMV disease is caused by a herpesvirus that remains latent in white blood cells for the life of the host. Because disease usually results from reactivation of the latent virus rather than reinfection, measurement of CMV antibody levels is usually not helpful in establishing a diagnosis. The best test for diagnosing invasive CMV disease is a biopsy. Specimens characteristically show cytomegalic cells with enlarged nuclei that contain a large, central, basophilic, intranuclear inclusion. The histologic sensitivity of GI mucosal biopsies can be increased by obtaining deep biopsy specimens that include endothelial cells and fibroblasts in the lamina propria, because these cells are more likely to be infected with CMV than are epithelial cells. Confirmation of CMV disease of the GI tract requires the demonstration of infection in addition to gross pathologic abnormalities. Infection can be demonstrated by culture; detection of CMV antigen in tissue, CMV genome in tissue, or typical CMV cytopathology; or visualization of CMV virions with electron microscopy. However, tests for the presence of CMV in the GI tract, such as stool cultures or swabs of the mucosa, indicate viral shedding and do not necessarily prove that tissue-invasive CMV disease is present. CMV inclusion bodies are not seen in all cases of CMV disease. Therefore, viral culture—a more sensitive diagnostic technique—has been recommended. When cultures are performed, some authors recommend that the processed tissue specimens be added to human fibroblasts and examined for cytopathic effects for 10 to 14 days. The shell vial culture technique allows for specific diagnosis within 18 to 36 hours. Polymerase chain reaction assay cannot differentiate between CMV infection and CMV disease but can be used for early detection. The radiologic findings in CMV disease are nonspecific; however, in the appropriate clinical setting, they can be highly suggestive. Findings such as segmental or focal involvement, mucosal granularity, erosion ulceration, and ulcers projecting intraluminally may indicate CMV colitis. Complications of the GI tract have been fatal in as many as 83% of affected patients.

**TREATMENT** The use of ganciclovir or foscarnet has dramatically improved the prognosis for patients with CMV disease. The dosage of ganciclovir must be adjusted based on the patient’s renal function. The most common side effect noted with ganciclovir therapy is reversible bone marrow suppression. Relapses after ganciclovir therapy for CMV disease of the GI tract occur in up to 40% of immunocompromised patients who do not have AIDS. There is little evidence to recommend routine maintenance therapy with ganciclovir. Consider a maintenance regimen for the rare patient who has frequently recurring episodes of CMV disease. Other agents, such as acyclovir, vidarabine, and interferon, have not reduced mortality or provided consistent antiviral activity. Foscarnet, a newer antiviral drug, demonstrates good activity against CMV and has been used successfully to treat CMV infections in patients with AIDS. No studies have investigated the efficacy of foscarnet in patients with GI CMV disease who do not have AIDS. The **TAKE-HOME** MESSAGE Consider CMV colitis when a patient who is receiving immunosuppressive therapy presents with GI symptoms. Early diagnosis and treatment can prevent complications and reduce mortality.

**References:**


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