Inflammatory bowel disease (IBD) is thought to result from inappropriate activation of the body’s immune system, and current medical therapy is aimed at modulating this immune response. Human immunodeficiency virus (HIV) infection slowly destroys the patient’s T helper cells and leads to immunosuppression, infection with opportunistic pathogens and eventually death. One may therefore expect IBD to improve in the presence of HIV infection. This may be the case with Crohn’s disease (CD) (1,2), but there are reports of active ulcerative colitis (UC) in the face of HIV infection with undetermined (3), normal (4,5) and diminished CD4 cell counts (6,7). We report a case of chronically active UC in an HIV-infected patient with a reduced CD4 lymphocyte count.
CASE PRESENTATION

A 40-year-old man was simultaneously diagnosed with HIV infection and UC in March 1986. UC diagnosis was based on a history of frequent bloody bowel motions and abdominal cramps with negative stool cultures and confluent proctosigmoiditis on sigmoidoscopic examination. Colon biopsies showed acute inflammation with cryptitis, crypt abscesses and goblet cell depletion, but no viral inclusions (Figure 1). He had a negative family history for IBD but had recently quit a 15-pack/year smoking habit. His only risk for HIV exposure was homosexuality. He received zidovudine from December 1989 to January 1993, switching to dideoxyinosine until March 1994. He has not had an acquired immune deficiency syndrome-defining illness and his most recent CD4 cell count was 300 cells/mL (normal range 353 to 1316).

The patient’s UC was mild and controlled with intermittent oral sulphasalazine until December 1989 when he began having more frequent episodes of bloody stools and intestinal cramps, which responded to 2 to 3 g/day oral sulphasalazine. In April 1993 he presented with a four-month exacerbation of his symptoms which had not adequately responded to sulphasalazine. He initially responded to the addition of a tapering course of prednisone 25 mg/day, but over the ensuing months he required larger doses to control the symptoms. Attempts to reduce the prednisone dose to less than 20 mg/day resulted in increased cramps and bloody diarrhea. He did not respond to the addition of cortifoam enemas on retiring or the temporary discontinuation of sulphasalazine.

Because dideoxyinosine has been associated with diarrhea (2), it was stopped in March 1994 without effect. A colonoscopy performed in April 1994 revealed contiguously inflamed mucosa to 55 cm without evidence of Kaposi’s sarcoma. Repeated colonic biopsies over several years have all shown a mixed inflammatory infiltrate of the lamina propria with polymorphonuclear cell infiltration and focal destruction of the epithelium, as well as crypt shortening, fission and drop-out, features of chronic active idiopathic UC (Figure 1).

The patient has anti-cytomegalovirus (anti-CMV) immunoglobulin G, but viral culture and electron microscopic examination of the biopsies for CMV have been negative, and viral inclusions have not been identified histologically. Repeated stool cultures for bacterial pathogens were negative, while microscopic examination showed Endolimax nana and Entamoeba coli on one occasion each. His course to June 1994 is summarized in Figure 2. He has not had fevers, chills, significant weight loss or extra-intestinal manifestations of UC. Colectomy has been discussed, but he prefers to continue medical therapy.

DISCUSSION

Making the diagnosis of IBD in the face of HIV infection may be difficult because diarrhea is a common complaint among HIV-infected patients (8). Symptoms mimicking IBD may be caused by a variety of intestinal infections, and in HIV-infected patients such symptoms have been reported due to CMV (9,10) and Kaposi’s sarcoma (3,4). The pathogenic role of Kaposi’s sarcoma in the latter cases (3,4) is unclear because both presented with a severe UC-like illness and were subsequently found to have extensive colonic Kaposi’s sarcoma. The lack of evidence of any alternative diagnoses in our patient, the chronic nature and relapsing course of his disease, its typical colonoscopic and histological appearance, and its response to sulphasalazine and prednisone all support the diagnosis of idiopathic UC.

The combination of IBD and HIV is rare. There are three other well-documented cases in the literature of active UC in HIV infection (5-7). In these reports the disease was ac-
Active ulcerative colitis and HIV infection

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REFERENCES
7. Sturgess I, Greenfield SM, Teare J, O'Doherty MJ. Ulcerative colitis disease in HIV-positive patients with CD4 counts less than 50 cells/mL. (12). Azathioprine and 6-mercaptopurine are effective in active UC (13,14) but may cause lymphopenia and pancreatitis, adverse effects shared with antiretroviral agents. Cyclosporine is effective in acute severe UC unresponsive to corticosteroid therapy (15) but has significant toxicity and increases the risk for opportunistic infection. Colectomy does not carry an increased risk for infection, although the risk for pouchitis is unknown.

CONCLUSIONS
The presence of active UC in an HIV-infected individual with a subnormal CD4 cell count is a complex problem because the most potent medical therapies for UC increase the risk of developing infection, and HIV is associated with intestinal infections that can exacerbate UC symptoms, truly a 'pas de deux'. It also raises interesting questions about IBD pathogenesis and challenges the clinician to manage an active inflammatory process in an immunocompromised patient.
