An Unexpected Cause of Gastrointestinal Symptoms after Initiation of HAART for HIV

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A 43-year-old female with HIV/AIDS (CD4 count 35 cells/mm$^3$) presented with one week of crampy abdominal pain and four days of melena. She was found to be anemic with hemoglobin 8.3 g/dL, down from 9.5 g/dL two months prior. She had restarted HAART medications one month before onset of symptoms after going without treatment for the previous five years. One month prior, her CD4 count was 35 cells/mm$^3$ and her HIV viral load was 296,878 copies/mL. In addition to HAART, she was started on azithromycin, TMP/SMX and acyclovir for opportunistic infection prophylaxis.

The patient underwent upper GI endoscopy, which showed normal esophagus and stomach but proximal duodenum with patchy whitish mucosa with diffuse flattening of villi. Biopsies were obtained. She then underwent colonoscopy, which found sessile polyps and granular mucosa in the distal transverse colon, all of which were biopsied. Pathology results from duodenum, transverse colon mucosa and polyps showed mucosal expansion by foamy macrophages laden with numerous acid-fast mycobacteria on AFB stain, consistent with *Mycobacterium avium* complex (MAC). The patient was started on clarithromycin, ethambutol and rifabutin, with plans for a prolonged course of at least one year. Her HAART medications were continued.

Discussion

This case illustrates the importance of considering MAC infection of the GI tract, along with other opportunistic infections in HIV-infected patients with low CD4 cell counts and GI symptoms or GI bleeding. Diagnosis of MAC infection may require upper endoscopy and/or colonoscopy with multiple biopsies. Microscopically, the tissue is filled with distended histiocytes packed with acid-fast organisms. The macrophages are unable to lyse or digest the bacilli because of the CD4 T-cell immunodeficiency of HIV/AIDS.

Disseminated MAC appears to result from primary acquisition of the pathogen, in contrast to tuberculosis in AIDS, which results from reactivation of previously contained infection. The patients at highest risk of disseminated MAC are those with CD4 counts <50 cells/mm$^3$, with infections rare with CD4 >100 cells/mm$^3$. In patients receiving HAART, the risk of developing MAC (and other opportunistic infections) is highest during the initial months of therapy, with low CD4 counts being the best predictor. Since the introduction of HAART, MAC infections of the GI tract are rare except in patients who progress to advanced HIV/AIDS.

First-line treatment for MAC consists of clarithromycin and ethambutol, with many clinicians adding rifabutin for associated decreased resistance and improved survival. Amikacin or streptomycin may be added for patients at high risk of death from MAC. Although the optimal duration of therapy remains unclear, IDSA guidelines suggest at least 12 months plus six months of immune reconstitution with HAART.

REFERENCES