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Gastrointestinal Complications of HIV-1 Infection

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Gastrointestinal Complications of HIV-1 Infection

John E. Pandolfino, MD

INTRODUCTION

Patients with HIV-1 infection commonly develop gastrointestinal (GI) complications—usually opportunistic infections. Traditionally, gastroenterologists have been focused on the prophylaxis and treatment of GI infections in patients infected with HIV-1 by using antibiotics. However, highly active antiretroviral therapy (HAART) may also play a substantial role in the prophylaxis and treatment of GI complications of HIV-1 infection by leading to improvements in immune function. This manual reviews the role of the GI tract in the transmission of HIV-1, describes key features in the pathogenesis and pathophysiology of HIV-1 infection, and uses case-based discussions to highlight the diagnosis and treatment of several GI complications of HIV-1 infection in the era of HAART.

GASTROINTESTINAL ASPECTS OF HIV-1 INFECTION

The mucosal tissue of the genital and GI tracts serves as the portal of HIV-1 entry in vertical transmission and sexual transmission (ie, oral-genital, anal-genital, and genital-genital). Excluding transmission of the virus directly into the bloodstream during transfusion therapy and the use of illicit intravenous drugs, HIV-1 is typically transmitted across this mucosal tissue—from mother to child and from one sexual partner to another.

The development of infection is related to the amount of virus inoculated onto the recipient's mucosal tissue, as well as the susceptibility of the individual. The amount of virus inoculated correlates with the viral load of the individual spreading the infection and is highest during primary infection and end-stage HIV-1 disease. The susceptibility of an individual to infection is increased with factors that disrupt the mucosal barrier and with those factors that enhance the susceptibility of lymphoid cells to HIV-1 infection. Infection-related inflammation and ulceration of mucosal tissue (eg, from herpes simplex or syphilis) or disruption of the rectal mucosa from any cause (eg, by trauma) enables pathogens to directly access the mucosal microcirculation. Additionally, chemokine coreceptors (eg, CCR5) used by macrophage-tropic and T-cell-tropic HIV-1 for entry into CD4 cells may be up-regulated by preexisting infections.¹

HIV-1 comes into contact with T cells once it enters the lamina propria. The virus enters T cells expressing CCR5 and begins to replicate. Eventually, HIV-1 will induce T-cell death by cell lysis, apoptosis, and cytotoxic lymphocyte-induced killing. The depletion of lamina propria T cells leads to local immunosuppression, and the GI tract consequently becomes predisposed to opportunistic infections. Virus infecting activated T cells in organized lymphoid structures may lead to the dissemination of viral particles to distant mucosal sites and subsequently the destruction of T cells in those areas. However, the depletion of T cells at the primary site of infection in the lamina propria of the GI tract generally precedes the depletion of T cells in other sites.¹

EFFECTS OF HAART

HAART consists of several anti-HIV-1 drugs—usually 2 nucleoside reverse transcriptase inhibitors and a protease inhibitor (PI). The favorable effects of HAART on morbidity and mortality among patients with HIV-1 infection are likely the result of the regimen's action in inhibiting HIV-1 replication and the subsequent recovery of immune function.¹ Specifically, the decreased replication of HIV-1 results in an increase in the number of CD4 cells, a decrease in the level of B-cell and cytotoxic T-cell activation, and a restoration of the normal architecture of lymphoid structures.

HAART has led to a reduction in the incidence of opportunistic infections, including those of the GI tract, and a decreased rate of reactivation of latent infections. The incidence of the 3 most common opportunistic infections (*Pneumocystis carinii* pneumonia [PCP], *Mycobacterium avium* complex [MAC] disease, and cytomegalovirus [CMV] retinitis) has declined dramatically as a result of HAART.² Moreover, some adverse effects of infection with untreatable pathogens such as *Cryptosporidium* and microsporidia have been reported to improve with HAART.^{3,4}

In many cases, HAART may also allow the patient to discontinue prophylactic antibiotic therapy. This may be beneficial in decreasing antibiotic resistance and the rate of antibiotic-associated enteric infections. However, recovery of immune function is not immediate with HAART. Moreover, it is recommended that prophylactic